Eating Disorders, Addictions and Substance Use Disorders

Research, Clinical and Treatment Perspectives

Timothy D. Brewerton
Amy Baker Dennis
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To all the great teachers in my life, especially my patients, who taught me how to listen and to follow their lead, and to Therese, for whom I am infinitely grateful in so many ways

T.D.B.

To Daniel and our children, Jessica, Nicole, Michelle, and Christopher

A.B.D.

Foreword

We spend so much time studying in our specific area, evaluating and treating patients in our specialty, that we sometime lose sight of the forest for the trees. While we see smoking patients, who stop only to gain 20 pounds over the next year, or alcoholics and addicts who become ravenous upon successful abstinence, we somehow rarely think about the overlaps and relationship between substance use disorders and eating disorders. How often do we hear about a patient who has sought treatment multiple times at a substance use residential treatment program, never addressing his or her eating disorder, only to relapse over and over again until finally finding treatment that addresses both the eating and substance use disorders (and vice versa)? It shouldn't be surprising that cross-sectional studies of women with eating disorders have a higher prevalence of alcohol and substance abuse compared to the general female population as well; women with substance abuse disorders report a higher prevalence of eating-disordered behavior more often than in the general female population. *Eating Disorders, Addictions and Substance Use Disorders: Research, Clinical, and Treatment Perspectives* is a manual that is long overdue and one that will help guide future research and treatment of patients in both of these fields.

The intended audience of this book is researchers and clinicians who work in either field (substance use disorders or eating disorders). It is likely that if a clinician works in either of these fields, he or she, in a sense, works in both fields, whether the clinician is aware or not. We can only begin to imagine how often a patient with a dual diagnosis of substance use and eating disorder presents seeking treatment in either setting, yet only one problem is addressed or even recognized upon presentation. This book serves as a guide for new clinicians and a reminder for clinicians already aware of the overlap between these two fields. It is also a tool for the eating disorder specialist who is perhaps less familiar with working with individuals with substance use disorders as well as a tool for the substance abuse specialist who is less familiar with eating disorders.

In addition to the clinical perspectives offered in this book, the book also offers new research that is emerging that suggests that the overlap between these two areas is even stronger than initially expected. In particular, the chapter, "Neuroimaging of Eating Disorders, Substance Use Disorders and Addictions: Overlapping and Unique Systems," summarizes the most recent neuroimaging literature and compares the similarities between eating disorders and substance use disorders.

While the neuroimaging literature on eating disorders is relatively limited compared with substance use disorders, the chapter highlights the similarities between bulimia nervosa, binge eating disorder, and addiction while pointing out the dissimilarities between anorexia nervosa and these disorders. It is fascinating how similar bulimia nervosa and binge eating disorder are to substance use disorders in multiple studies that looked at executive control, motivation and reward, learning and memory, emotion regulation, and interoceptive awareness. This parallels real life as the overlap between substance use disorders and eating disorders tends to occur primarily in individuals who have binge eating and purging behavior as opposed to individuals with anorexia nervosa with restrictive subtype (who do not engage in binge eating and purging behaviors).

The implications of the overlap between eating disorders and substance use disorders are profound as they can potentially guide future diagnosis and treatment. Based on the similarities between the two, one may ponder whether eating disorders are addictions and whether this has diagnostic and treatment implications. While certainly eating disorders are unique from substance use disorders, the similarities are powerful enough to suggest that more research is needed in this area. While we have focused on the brain and brain systems common to drug abuse, food preferences, eating disorders, and food addictions, there is much more to this field than our observation of it. If it looks like a duck and quacks, it is a duck. This wonderful book conceived and brought to this point by Tim Brewerton, M.D., and Amy Baker Dennis, Ph.D., and takes the field and interrelationships to the next, very logical and compelling level. Thanks, for this wonderful addition to our field.

Gainesville, FL, USA

Amelia Davis
Mark S. Gold

Preface

In the past several decades, there have been numerous books published on the science and treatment of eating disorders (ED) and hundreds of volumes written on the diagnosis, assessment, and treatment of substance use disorders (SUD)/addictions. At least to our collective knowledge, this is the first textbook to completely focus on the co-occurrence of these disorders. This project was born out of our surprise and deep frustration with both the lack of evidence-based research and treatment protocols for this comorbid population and the lack of available treatment programs, at all levels of care, that effectively serve consumers with both ED and SUD. With up to 50 % of ED patients meeting criteria for a SUD or addiction and 1/3 of SUD patients reporting eating pathology, it is remarkable how few people in either field are fully trained to address both disorders. Additionally, we were hoping to strengthen connections between two fields that rarely communicate. With these issues in mind, we felt the time was right to assemble leading experts from each field and ask them to work together to produce a "state-of-the-art" textbook that could not only educate practitioners in both fields but also promote cross-training and further collaborative research.

The 30 chapters of this volume are organized into three major parts. The first, "Research Perspectives," explores the science of ED, SUD, and addictions. Our researchers were tasked with the responsibility of reviewing and reporting on what is known about each disorder independently and to discuss the similarities and differences between these disorders according to their topic. In order to make this part relevant to treatment providers, each author was asked to identify how this research can guide and inform clinical practice with dually diagnosed patients. The first two chapters of this part focus on how animal research has informed our understanding of biological and behavioral factors that contribute to the development of ED and SUD as well as the relationship between eating and drug use behaviors. Chapter 3 explores the neurobiological mechanisms and neurotransmitter/brain circuit alterations that may be common to both disorders. The authors of Chapter 4 discuss the finding from neuroimaging studies that examine shared and distinct domains of functioning (i.e., executive control, reward and motivation, emotional reactivity, memory/learning, and interoceptive awareness) and outline opportunities for future research to further clarify the relationship between addictions and eating behaviors. Chapter 5 reviews the current empirical literature

indicating that anorexia nervosa, bulimia nervosa, and SUD are influenced by genetic factors and may indeed share a similar genetic architecture. In Chap. 6, the authors examine dimensions of personality pathology and personality disorders and their relationship to both ED and SUD. Chapter 7 tackles the issue of problematic exercise. Theoretically, is pathologically excessive exercise an addiction or a compulsion? The authors discuss the emerging literature and its relationship to eating pathology, SUD, and other behavioral addictions. Nutritional aspects, particularly the nutritional deficits and their clinical and neurological manifestations in both ED and SUD, are discussed in detail in Chap. 8. Chapter 9 explores the emergence of significant problems such as alcohol use disorders and ED following bariatric surgery. Finally, Chap. 10 begins with a scholarly review of effective prevention models that have been utilized in either the SUD or ED fields and concludes with proposed guidelines for the development of a program designed to address both disorders simultaneously.

Part II, "Clinical Perspectives," begins with two chapters that are designed to initiate cross-training between fields. Chapter 11 was specifically developed to educate substance abuse specialists that have limited knowledge or expertise in the diagnosis, assessment, and treatment of ED. Likewise, Chap. 12 provides an overview of the psychoactive properties of drugs of abuse, clinical characteristics of individuals with SUD, and evidence-based treatments for SUD. This chapter was specifically written for the ED treatment provider that has not had adequate training or supervision in the treatment of SUD. Chapter 13 confronts the highly controversial issue "*Are eating disorders addictions*?" The author identifies both the pros and cons of this debate and challenges professionals from both fields to review the assembled data from a theoretical, clinical, and research perspective. The authors of Chap. 14 provide a comprehensive overview of both self-report and semi-structured interview instruments currently used in both fields for children, adolescents, and adults. They conclude with a discussion of the need to broaden the accessibility of technology-based measures to simultaneously assess both disorders. Chapter 15 is one of the most comprehensive chapters in this book. The authors provide a detailed description of the medical complications commonly encountered in ED and SUD and explain the importance of medical management and monitoring throughout the treatment and recovery process. The role of negative affect in both the development and maintenance of ED and SUD is explored in depth in Chap. 16. Similarly, Chap. 17 investigates the role of stress, adversity, and trauma in the etiology and treatment of ED, SUD, and behavioral addictions, with a focus on the role of PTSD in mediating comorbidity between ED and SUD. In addition, the emerging science of epigenetics as applied to these populations is considered. Chapter 18 is devoted to a comprehensive discussion of behavioral addictions including gambling, kleptomania, Internet addiction, and hypersexual behavior. The authors examine what is known about the clinical presentation, epidemiology, etiology, and treatment of behavioral addictions commonly comorbid with ED and SUD. Chapter 19 takes a closer look at another behavioral addiction, compulsive buying disorder, and its relationship to both ED and SUD. Chapter 20 concludes this part with an

informative discussion of muscle dysmorphia, body image dissatisfaction, ED, compulsive exercise, and anabolic steroid abuse in males.

Part III, "Treatment Perspectives," was designed to provide the reader with a comprehensive overview of evidence-based practices in both the treatment of ED and SUD. Based on what is known from each field independently, we asked each author in this part to make recommendations on how they could integrate these findings for the treatment of comorbid patients. We are extremely pleased, not only with their collaboration with experts from a different field but also the creative and thought-provoking recommendations that are detailed in each chapter of this part. Chapter 21 begins with a discussion of how programs that are currently dedicated to the treatment of either ED or SUD can modify their protocols to more effectively integrate treatments for the comorbid patient. In Chap. 22, the authors explore the evidence-based approach of motivational interviewing to help patients with both ED and SUD resolve their ambivalence about change and recovery. Nutritional therapy for dually diagnosed patients is discussed in detail in Chap. 23, with a specific emphasis on the creation of an individualized treatment plan. Cognitive behavioral therapy (CBT) is an evidence-based treatment for ED, SUD, and several other related comorbid conditions. Chapter 24 proposes a model for combining the CBT models that currently exist in each field into a single treatment that allows for the use of modular-based integration, targeting aspects of several comorbidities, and personality. Treatments that include mindfulness as a core therapeutic strategy are outlined in Chap. 25, with a special emphasis on Acceptance and Commitment Therapy (ACT), Dialectical Behavior Therapy (DBT), and Mindfulness-Based Relapse Prevention (MBRP). The authors of Chap. 26 summarize the empirical evidence for the use of family and couples therapy in the treatment of ED and SUD and then outline how to adapt family-based treatments for adolescents with both ED and SUD. The authors of Chap. 27 provide the reader with a comprehensive review of all the self-help approaches that are currently utilized to augment treatment for individuals with ED and SUD. Chapter 28 tackles the complex issue of exercise in the treatment of patients with these comorbid disorders. Alternative and complementary therapies including yoga, acupuncture, therapeutic massage, hypnosis, herbal medicine, light therapy, spiritual healing, and art therapy are often provided in the course of treatment for both ED and SUD. The authors of Chap. 29 evaluate the effectiveness of each of these approaches for patients with this comorbid disorder. The book concludes with a discussion of ethics and civil commitment. In Chap. 30, the author reviews the controversial use of civil commitment in each field and discusses its role as a legitimate tool in emergent situations when ED and SUD become life threatening.

Finally, we want to personally thank each author that contributed to this book. In many cases, we asked them to collaborate with an expert or immerse themselves in data from a different field. Each author utilized their extensive knowledge and expertise to make this book clinically relevant to both fields. We are proud to have contributing authors from many different countries and parts of the world, including Australia, Canada, Germany, Italy, and the USA (with contributions from institutions in 21 states). Several people contributed to more than one chapter,

and to them we are especially grateful, including Tamara Pryor, Therese Killeen, James Mitchell, Astrid Müller, David Wiss, Kristin von Ranson, and Brian Cook.

Bloomfield Hills, MI, USA — Amy Baker Dennis
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This book has been in the works for many years, but it would never have become a reality without the encouragement and involvement of my coeditor, Tim Brewerton. We have known each other for decades, but you really get to know and appreciate someone intimately when you collaborate for 18 months, shepherding a book from inception to publication. We quickly fell into a supportive, working relationship, negotiating details and dividing up responsibilities with ease and efficiency. It has been a real pleasure working with him and I am proud to be both his coeditor and friend.

My professional career began in the field of substance abuse. After a semester of working at Eastern Pennsylvania Psychiatric Institute in Philadelphia and attending a comprehensive professional development program for therapist provided by the National Institute of Substance Abuse, I returned to graduate school at Ohio State and went to work at a Community Mental Health Center. In 1976, I was unexpectedly introduced to anorexia nervosa and from that point forward, I have immersed myself in research, writing, teaching, and running conferences on the treatment of eating disorders.

My journey as a clinical psychologist has been filled with many mentors, collaborators, and colleagues. I would like to acknowledge my earliest mentors, S.R. Thorward MD, James Lantz Ph.D., Earl Greer Ph.D., and Aaron "Tim" Beck MD. They gave me their time and wisdom, provided skills training and supervision, and believed in me. I also need to acknowledge a few special people who supported me throughout this process. To Craig Johnson Ph.D., Michael Levine Ph.D., and Ann Kearney-Cooke Ph.D., thank you for always being available for advice, consultation, and encouragement. I will be forever grateful for your collective wisdom, humor, and long-standing friendship. And to my long-term business partner, Ann Moye Ph.D., thank you for "holding down the fort" and affording me the time and opportunity to complete this project.

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Bloomfield Hills, MI, USA Amy Baker Dennis

This book represents the melding of many minds from many fields. I want to especially acknowledge my coeditor Amy Baker Dennis, who has provided not only her exceptional collaboration and teamwork but also her keen intelligence, balanced perspectives, attention to detail, and incredible organizational skills. From the very beginning I was impressed with how well we communicated and complemented each other's strengths and weaknesses. Her involvement has made this project considerably easier, more enjoyable, and a great learning experience. Although Amy and I have known each other for many years and have worked together on occasion, we became much closer collaborators, colleagues, and friends during the development and creation of this book. The process of us coming together to coedit this book evolved somewhat serendipitously and was the culmination of many interacting forces, internal and external to ourselves, to our fields, and to our respective disciplines. Given the incredible growth in the eating disorder and addiction fields, and the simultaneously growing links and chasms between them, this project was an inevitability. It was just a matter of who would take on the challenge—if not us then whom?

There are many people in my life who in part have ultimately inspired this textbook, from the many great teachers and mentors I have been fortunate enough to have, to all my fellow clinical practitioners and research scientists that I have had the pleasure to know, work with, and learn from, to the many patients and families who rightly have challenged our paradigms and treatment approaches, and to the patients I have lost along the way to death by suicide, eating disorder, and/or addictions. Similar to Amy, I began my psychiatric career in medical school working with adult patients with alcohol use disorders under the supervision of Don Gallant, MD, who was a strong early influence. And thank you to John Kuehn, MD, for inspiring me to go into psychiatry in the first place. I also want to thank Steve Karp, DO, an addiction-certified psychiatrist who treats eating disorders, for his encouragement to pursue investigation into this borderland. I am deeply indebted to the Medical University of South Carolina Department of Psychiatry

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Part III Treatment Perspectives

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Part I

Research Perspectives

Animal Models of Eating Disorders, Substance Use Disorders, and Addictions

1

Susan Murray, Monica Gordillo, and Nicole M. Avena

Abstract

Laboratory animal models are valuable in that they allow researchers to better understand the various biological and behavioral factors that may contribute to eating disorders and substance use disorders, as well as an opportunity to discover effective treatments for each. This chapter describes how animal models have been used to study features of anorexia nervosa, bulimia nervosa, and binge eating disorder, with a particular focus on the variables associated with the development of such behavior in animals. The second half of this chapter focuses on the various animal models that have been used to explore key characteristics of addiction. This chapter concludes with a brief discussion of the overlaps that exist between the two types of disorders and suggestions for future research directions.

Keywords

Addiction • Animal models • Anorexia nervosa • Bulimia nervosa • Binge eating • Binge eating disorder • Hyperactivity • Purging • Rat • Rodent

1.1 Introduction

Laboratory animal models allow researchers the unique opportunity to study both physical and psychological disorders in ways that would otherwise often be unfeasible among clinical samples. Further, such techniques allow researchers to isolate

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the biological mechanisms associated with a given disorder without the influence of many of the potentially confounding variables seen in humans, such as various social and cultural influences. As a result, animal models can serve as valuable tools in discovering the physiological bases of psychiatric disorders as well as viable treatment options. In some cases it is difficult, if not impossible, to model each characteristic of a specific disorder with the use of animals. Rather, animal models provide a method of replicating and investigating specific *symptoms* of a disorder. This chapter will discuss the behavioral symptoms of eating disorders and substance use disorders that have been explored through the use of animal models. Further, this chapter will describe how these symptoms have been isolated and studied by researchers, as well as some noteworthy findings that have been revealed as a result.

1.2 Animal Models of Eating Disorder Symptoms

1.2.1 Anorexia Nervosa

Anorexia nervosa (AN) is a psychiatric disorder characterized primarily by the refusal to maintain a healthy body weight and an unrelenting fear of gaining weight. In the United States, the prevalence of AN among adults is reported to be approximately 0.9 % and 0.3 % in women and men, respectively (Hudson, Hiripi, Pope, & Kessler, 2007). This disorder has been associated with a range of both medical and psychological comorbidities, including osteoporosis, bradycardia, anxiety, and depression (Attia, 2010). Although AN has been recognized by the *Diagnostic and Statistical Manual of Mental Disorders* for several decades, many questions remain regarding the biological alterations that may underlie and result from behaviors associated with AN and which treatment approaches are most effective for this disorder. The use of animal models may provide the field with new and unique insights into this complex disorder.

The primary animal model that is presently available for studying AN is activity-based anorexia (ABA). This model is based on the finding that rats given limited access to food (i.e., 1–2 h/day) and unlimited or nearly unlimited access to a running wheel will increase their running activity, particularly preceding scheduled food access (known as food anticipatory activity); lose a significant portion of their initial body weight; and, if the experimental protocol is not stopped, eventually die of self-starvation (Epling & Pierce, 1984; Routtenberg & Kuznesof, 1967). This model is thus able to capture three critical symptoms associated with AN: decreased calorie intake, hyperactivity, and weight loss (see Fig. 1.1). Female ABA rats also show changes in their hormonal cycles (Dixon, Ackert, & Eckel, 2003; Watanabe, Hara, & Ogawa, 1992), providing further congruence between this model and the feature of amenorrhea seen in AN. It should be noted that while hyperactivity is not considered a diagnostic criteria for AN, this feature has been reported in 31–80 % of individuals with AN (Hebebrand et al., 2003). Additionally, recent research shows that AN patients have significantly higher total energy expenditure and physical

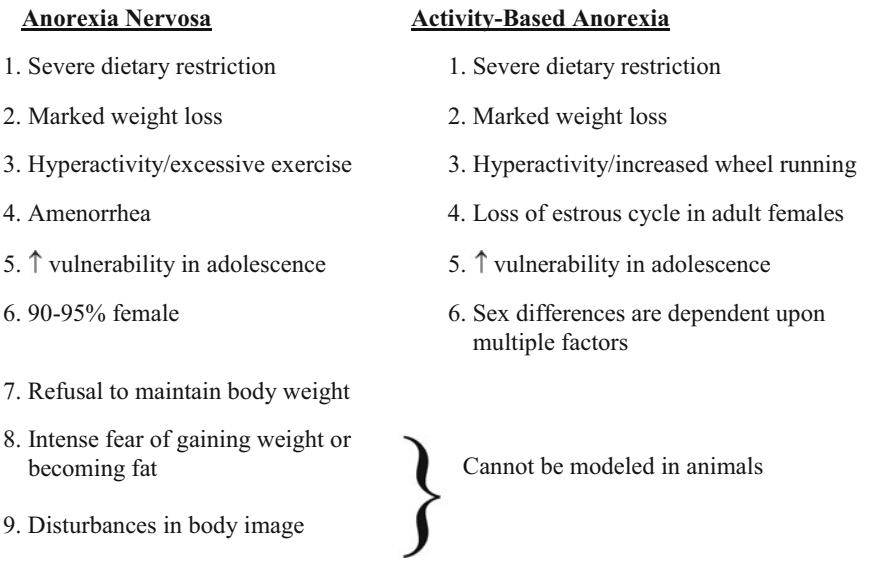

Fig. 1.1 Behavioral similarities between individuals with anorexia nervosa and animals vulnerable to activity-based anorexia. Reprinted and adapted from *Anorexia nervosa: Symptoms, treatment, and neurobiology*, A translational approach to understanding anorexia nervosa, p. 167, 2012, N. Barbarich-Marsteller, with permission from Nova Science Publishers, Inc

activity patterns compared to either normal or overweight controls (Elbelt et al., 2013). Research using the ABA model has allowed investigators the opportunity to explore three main questions: (1) what factors might increase an animal's susceptibility to develop ABA, (2) how does ABA alter physiological mechanisms, and (3) what factors might protect against or attenuate ABA behavior and, ultimately, contribute to the recovery of body weight? (See Fig. 1.1.)

1.2.1.1 Theories on Energy Expenditure

Two main theories have been proposed to explain why animals increase their energy expenditure when food is restricted. First, it has been proposed that animals may respond to calorie limitations with increased exercise as an adaptive strategy (Epling & Pierce, 1988). Indeed, one can imagine that when resources are limited, it would prove advantageous for survival to move in search of food. Recent research has supported this theory by demonstrating that ABA rats with access to sucrose, which is energy dense, run less than ABA rats with access to saccharin, which does not provide energy (Duclos, Ouerdani, Mormede, & Konsman, 2012). Evidence that food-restricted animals do not increase their running activity when given several small chances to feed throughout the day, as opposed to one meal or two shorter meals, provides further support for the idea that this behavior may be motivated by a search for food (Kanarek & Collier, 1983).

In addition to the possible evolutionary value of this behavior, it has been argued that running has reinforcing qualities (Belke & Wagner, 1996; Scheurink, Boersma,

Nergardh, & Sodersten, 2010), and it has been shown that running serves as a more potent reinforcer when animals are food restricted (Pierce, Epling, & Boer, 1986). Research from our laboratory has revealed increased extracellular dopamine (DA) release in the nucleus accumbens (NAc) of ABA rats when running compared to controls (Avena, Murray, Barbarich-Marsteller, & Rada (September, 2013). These findings contribute to the theory that animals may engage in high levels of exercise to experience the rewarding effects of running which may, in turn, be further enhanced by a state of hunger. While the two theories presented here are sometimes described as distinct or even competing, they may not be incompatible. As has been previously noted (Gutierrez, 2013; Garland et al., 2011), it is possible that adaptive coping strategies are promoted by natural sources of reinforcement.

1.2.1.2 Variables Associated with the Development of ABA

Research investigating the factors that increase subjects' susceptibility to or serve to protect against developing ABA may provide unique insights into the complex set of variables that influence the development of AN in humans. To date, a number of characteristics have been identified as important when studying ABA in animals, including weight, age, and sex. Rats with a low baseline body weight, for example, tend to lose more weight when subjected to ABA-like conditions (1.5 h of food access and 2 h access to a running wheel) than rats of the same sex and age with a higher baseline body weight (Boakes & Dwyer, 1997). Likewise, differences in age have been shown to influence the progression of ABA, with younger animals tending to lose weight more rapidly (Boakes, Mills, & Single, 1999). Because this effect may be a function of low body weight, investigators have compared older female and younger male rats of similar weights and found that older female rats showed less body weight loss during ABA (Boakes et al., 1999), supporting the idea that older animals may be less susceptible to the detrimental effects of this model. This corresponds with findings that AN onset typically occurs earlier in life (Hudson et al., 2007).

AN is also more prevalent among females (Hudson et al., 2007), leading some researchers to explore possible sex differences when studying the effects of ABA. However, the literature remains mixed regarding which sex is more or less vulnerable. While early research reported that female rats exhibit increased susceptibility to ABA compared to males due to characteristically high rates of running, this study did not report the ages of the rats or whether or not there were significant differences between the groups' baseline body weights (Pare, Vincent, Isom, & Reeves, 1978), which are now known to be key factors in the development of ABA. Additionally, while females do tend to run more (Boakes et al., 1999), males tend to lose weight more quickly, as females have also been shown to eat significantly more (Doerries, Stanley, & Aravich, 1991). Research investigating sex differences has also found this to be the case even when males had a higher baseline body weight (Doerries et al., 1991). Further, research comparing same age male and female rats did not show a significant difference between body weight loss, despite lower initial body weights and increased running observed among females, which the authors propose to be an indication that females may be less vulnerable than males to ABA.

Interestingly, it has been shown that while females remain consistent in their level of running activity despite declines in body weight, males tend to increase their wheel running as their body weight decreases (Boakes et al., 1999). Other research, however, has found that same-age females, weighing significantly less than their male counterparts, reach ABA removal criterion more quickly (Hancock & Grant, 2009), indicating that further research is needed to elucidate the sex differences that exist in this context and, more specifically, the factors that may contribute to them.

Baseline wheel running activity has also been shown to be a strong predictor of body weight loss for both mice and rats exposed to ABA conditions, with higher baseline rates predicting worse outcomes (Pjetri et al., 2012). This suggests that it may be relevant for clinicians to consider a patient's pre-morbid physical activity levels and, perhaps, that individuals involved in strenuous exercise routines may be more likely to develop AN-like symptoms when a diet is introduced. This theory has been tenuously supported by clinical research showing a higher prevalence of eating disorders among adolescent athletes compared to controls (Martinsen & Sundgot-Borgen, 2012), despite findings to the contrary (Martinsen, Bratland-Sanda, Eriksson, & Sundgot-Borgen, 2010; Reinking & Alexander, 2005).

The effects of stressful early life experiences, often modeled in animals with variations of maternal separation methods, have also been studied as a potential factor that may contribute to the development of ABA. However, this line of research has resulted in somewhat mixed findings. For example, rat pups separated from their dams for 180 min each day during the pre-weaning period show accelerated body weight loss, less food intake, and more pronounced increases in running activity compared to controls (15 min separation) (Hancock & Grant, 2009), suggesting that extended maternal separation during this developmental time period may increase an animal's vulnerability to ABA. Conversely, long maternal separation (3 h daily separation from dams) during pre-weaning has been shown to increase survival time in ABA in adult female rats compared to non-handling (Carrera, Cerrato, Sanchez, & Gutierrez, 2009). Finally, postnatal handling, which is known to increase maternal contact (licking and grooming behaviors) when the pup has been returned, has been shown to delay the amount of time before reaching removal criterion during ABA in adult females compared to non-handling (Carrera, Gutierrez, & Boakes, 2006). It is important to note that there are a number of variables to consider when interpreting these results which are beyond the scope of this chapter, including significant differences in baseline body weights (as seen in Carrera et al., 2006), as well as the implications of the control groups used.

Studies have also considered the protective role that certain factors may play in preventing or attenuating ABA behavior. For instance, social housing has been shown to decrease the deleterious effects of ABA and similar conditions on body weight loss compared to individual housing (Boakes & Dwyer, 1997; Ness, Marshall, & Aravich, 1995). Further, the palatability of the diet offered can affect weight loss, with a high-fat diet diminishing ABA (Brown, Avena, & Hoebel, 2008). These findings suggest the potential importance of social interaction as

well as diet composition in mediating the progression of ABA. Future research in this area may prove beneficial in informing prevention and/or attenuation strategies.

1.2.2 Bulimia Nervosa and Binge Eating Disorder

Bulimia nervosa (BN) is marked by (1) excess consumption of food during a distinct period of time (*binge eating*) which is accompanied by a sense of loss of control and (2) behaviors such as vomiting, laxative use, and intense exercise (*purging*), which are employed to compensate for the excess calories consumed while bingeing. The following section will discuss the various animal models that have been used to study these two criteria. It should be noted that models of binge eating are relevant to both BN and binge eating disorder (BED) as this symptom is a feature of both disorders.

1.2.2.1 Binge Eating

Over the last several decades, a number of paradigms have been used to model binge eating in animals. The results of such research have revealed a number of key factors related to this behavior, including a history of food restriction, availability of palatable food, and stress.

Several research efforts have sought to characterize the role of dieting in the development of binge-related eating disorders. While it appears that dieting is not necessary for the onset of binge eating, a considerable number of individuals who report binge eating also report prior dieting (Manwaring et al., 2006; Spurrell, Wilfley, Tanofsky, & Brownell, 1997). In animals, cycles of food restriction and subsequent refeeding have been associated with increased consumption of both chow and palatable food during test meals following weight restoration and the normalization of food access (Hagan & Moss, 1991; Hagan & Moss, 1997). It is interesting to note that when spontaneously tested (without an acute period of food deprivation), only rats with both a history of food restriction and access to palatable food during refeeding showed increased food intake during the test meal, mostly in the form of palatable food (Hagan & Moss, 1997). The presence of palatable food appears to play a major role in binge eating behavior among animals. In our laboratory, binge eating has been elicited by limiting rats' food access to 12 h per day (Avena, Rada, & Hoebel, 2006; Bocarsly & Avena, 2012). During this time, rats are provided unlimited access to palatable food and standard rodent chow. Food is presented 4 h after the start of the dark cycle (4 h after they typically begin to feed), and thus, animals are hungry and exhibit binge intake of the palatable food. This pattern of overeating does not develop in rats that are only provided chow during the 12 h access period, nor does it develop in rats with ad libitum access to the palatable food. Though not universally supported (Raymond, Bartholome, Lee, Peterson, & Raatz, 2007), there is clinical research to suggest that individuals with BED and BN also tend to consume high amounts of palatable foods during binge meals (Bartholome, Raymond, Lee, Peterson, & Warren, 2006; Kales, 1990; Rosen, Leitenberg, Fisher, & Khazam, 1986; Yanovski et al., 1992).

Binge eating has also been modeled in rats by coupling food restriction and refeeding with an environmental stressor. For example, it has been shown that while an increase in food intake occurs among female rats following 1 week of restricted food access (2 h/day) this increase was greatest among rats that were assigned to the 1 week 2 h/day food restriction schedule and were subsequently placed in a small, movement-limiting cage (Inoue et al., 1998). Similarly, while a 48 h period of food and water deprivation caused food intake to increase in rats, a shorter, 12 h period of food and water deprivation coupled with a 10 min swim test in cold water produced both a greater increase in food intake and a marked increase in high-fat diet consumption in particular (Vaswani, Tejwani, & Mousa, 1983). The role of palatable food in mediating the binge eating response was further shown by Hagan et al., (2002) who found that while the combination of food restriction, refeeding, and a foot shock did not significantly increase chow intake in female rats, hyperphagia was reported when rats were given access to a palatable food (Oreo cookies). In a fascinating study by Cifani, Polidori, Melotto, Ciccocioppo, and Massi (2009), researchers found that both a history of several food restriction/refeeding cycles and a stressor, which involved allowing animals to see and smell but not eat palatable food, resulted in significant increases in palatable food intake. The authors propose that this form of stress may resemble that experienced by humans when encountering what are considered to be "forbidden food" items. Collectively, this line of research has led to the notion that a history of the combination of food restriction, stress, and access to palatable food may promote binge eating. Interestingly, a recent laboratory study assessing the effects of stress on eating in humans found that patients with BED exhibited increased initial eating rate and an decreased eating deceleration when exposed to stress unlike participants without BED (Schulz & Laessle, 2012). Likewise, clinical research has shown stress to increase intake of sweet high-fat foods among emotional eaters (Oliver, Wardle, & Gibson, 2000).

Similar to studies investigating the effects of maternal separation on the development of ABA, research has explored the effects of maternal contact in early life on the development of binge eating. For example, 6- and 9-week-old female rats that had been separated from their mothers for 6 h/day for 3 weeks after birth exhibited significantly increased food intake following a period of restricted feeding compared to control rats who had been handled early in life (Iwasaki, Inoue, Kiriike, & Hikiji, 2000). This effect was not observed in female rats tested at either 3 or 12 weeks, nor was it shown in male rats at any time, indicating both age- and sex-specific effects. Additional evidence demonstrates that while hyperphagia was initially observed in both non-handled and maternally separated offspring during a period of refeeding following food restriction, only maternally separated rats continued to exhibit this response over multiple cycles (Ryu et al., 2008). Female offspring with lower levels of maternal care (measured by licking and grooming behaviors) during early life have also been shown to be more likely to binge eat palatable food after experiencing a shock, whether or not they had undergone food restriction. However, rats that had been food restricted exhibited this behavior sooner (Hancock, Menard, & Olmstead, 2005). Notably, these results were only

observed when this paradigm was introduced during adolescence but not in adulthood, which corresponds with evidence that the average onset for BN is during late adolescence (Hudson et al., 2007). Collectively, these findings suggest the relevance of early life stressors and maternal contact for binge eating behavior.

Corwin et al. (1998) have shown that food deprivation and stress are not necessary establishing operations for bingeing; by simply offering palatable food to rats on a limited access schedule (e.g., 2 h daily or 2 h 3 days a week), rats demonstrate excessive consumption. Likewise, binge eating has been modeled by providing intermittent access to a cafeteria-style diet including a variety of different foods, many of which are rich in fat and sugar (Leigh, Stock, Lacey, & Wilson, 1998). Wilson and Cantor (1987) have also elicited hyperphagia in food-satiated rats. When placed on an intermittent schedule of reinforcement, the reinforcer being electrical brain stimulation, most rats markedly increased their food intake. The authors suggest that, in this case, overeating may be seen as a type of adjunctive behavior engaged in between the receipt of reinforcers.

The number of animal models available to study binge eating behavior offers researchers unique opportunities to isolate and explore certain variables of interest. For instance, while binge eating may be related to increased body weight in certain models, our laboratory has noticed that sugar-bingeing animals tend to reduce their chow intake, effectively compensating for the excess calories taken in while bingeing (Avena, Rada, & Hoebel, 2008). Recent research by Hargrave and Kinzig (2012) has investigated the effects of enlarging the stomach by implanting and inflating chronic gastric balloons in the stomachs of rats, which may serve as an alternative model of binge eating that does not affect body weight. Such models can provide clinically relevant insights as body weight is not always increased in individuals who binge eat (Masheb & White, 2012). This also allows researchers to discriminate between the effects of binge eating versus those that may result from being overweight or obese. The different models that have been developed also provide the means to study subjects who engage in both binge eating and periods of self-imposed food restriction, as well as those that only report binge eating. Researchers may also wish to manipulate when palatable food is made available (i.e., every day or a few times a week) to reflect the clinical reality or the order of life events being studied (food restriction preceding stress or vice versa). Finally, the diversity of animal models allows investigators opportunities to study the effects of different stressors (i.e., early life stressors, acute, chronic) on binge eating behavior.

1.2.2.2 Purging

While the compensatory behaviors associated with BN represent a greater challenge to replicate among animals, it has been possible to explore the effects of this behavior through the use of the sham feeding technique. This method involves the implantation of an intragastric fistula which allows the contents of the stomach to be drained during meals. Studies employing this technique have shown sham feeding to increase food intake in both rats (Davis & Campbell, 1973) and monkeys (Gibbs & Falasco, 1978). Such findings suggest that purging may inhibit normal

post-ingestive feedback processes that signal satiety and, thus, increase food consumption. Interestingly, cues associated with prior sham feeding have also lead to increased consumption during real feeding (Van Vort & Smith, 1987).

Insights into purging behavior may also be gleaned from research investigating captive gorillas that have been shown to regurgitate and then reingest their food (Gould & Bres, 1986). This behavior was exhibited least by gorillas that were both born in captivity and reared by their mothers compared to those born in captivity but hand-reared and those captured in the wild and thus may have also experienced maternal separation. While this may not be an ideal model of BN, this finding does provide further support for theories that stressful early life experiences, and maternal separation in particular, may alter feeding behavior later in life.

1.3 Animal Models of Substance Use Disorders and Addictions

Numerous animal models have been developed over the years to study the various behaviors that characterize addiction. Similar to those used within the field of eating disorders, animal models employed within the context of understanding drug abuse and addiction often focus on specific symptoms that can be replicated in the laboratory. These symptoms may be informed by the diagnostic criteria used to classify substance use disorders (SUDs) and/or clinical observations. For example, while relapse is not listed as a diagnostic criterion for addiction, it remains a subject of interest within the field. The following section describes how animal models have been used to provide insight into some of the key aspects of addiction.

1.3.1 Tolerance

According to the DSM-5, tolerance is defined as "requiring a markedly increased dose of the substance to achieve the desired effect or a markedly reduced effect when the usual dose is consumed" (American Psychiatric Association [APA], 2013). While tolerance may at first seem to represent a challenge to researchers as it may be thought to have its basis in subjective experience and appraisal, behavioral models have been developed to assess this characteristic of addiction in animals. One method for measuring tolerance is a tilting plane test. When studying tolerance to alcohol consumption, for example, Nikander and Pekkanen (1977) placed rats on a plane thats angle would regularly increase until the rat could no longer hold its ground. The angle at which the rat slid (the "sliding angle") was used to indicate the point at which the animal was intoxicated. As alcohol intake increases and tolerance for lower doses develops, rats are better able to coordinate motor functions and stay on the plane for longer periods of time. Similarly, Tiffany and Maude-Griffin (1988) administered varying levels of morphine to rats to see which dosages would elicit stronger analgesic effects (a common effect of morphine). Following morphine administration, a tail flick test is administered to determine how long it takes for the rat to move its tail from under a hot beam of

light. A rat that is considered tolerant to the lower doses of morphine will flick its tail quickly from under the hot beam of light but will be slower when administered a higher dose. Thus, at least two distinct animals models have been used to assess the presence of tolerance in response to exposure to drugs of abuse.

1.3.2 Withdrawal

Unlike tolerance, assessing the presence of withdrawal syndrome does not necessarily require much experimental manipulation. Withdrawal may be elicited, for example, by taking a drug away from an animal after a certain period of use or by administering the appropriate antagonist. One symptom that has been observed in response to withdrawal from several different drugs of abuse is anxiety (Emmett-Oglesby, Mathis, Moon, & Lal, 1990). Therefore, withdrawal has been measured through the use of experimental tests that are thought to detect anxiety in animals. One such method involves training rats to lever press for food in response to feelings of anxiety (Gauvin et al., 1996). For example, rats have been given pentylenetetrazole (an anxiogenic drug) and saline and taught to lever press for food only in response to the effects of pentylenetrazole (i.e., the anxiety). Similarly, the animal will lever press in response to withdrawal-induced anxiety.

Behavioral coding has also been used to identify characteristics of withdrawal in animals. For instance, withdrawal in rats has been shown to produce wet dog shakes, writhing (or abdominal stretching), jumping, stereotyped head bobbing, sweeping tail movements, yawning, and increases in irritability (defined as episodes of conflict-induced vocalizations). Teeth chatter episodes (separated by at least 3 s), discrete episodes of chewing (without anything in mouth), lacrimation, piloerection, ptosis, salivation, and diarrhea have all been observed among animals during withdrawal (Rasmussen, Beitner-Johnson, Krystal, Aghajanian, & Nestler, 1990). In chimpanzees, symptoms of withdrawal are similar to those observed among humans. After several weeks of chronic ethanol intake, hyperreflexia and irritability were noted when blood alcohol levels neared zero. Concomitant symptoms include photophobia, rapid respiration, sweaty palms and feet, decreased responsiveness to auditory stimuli, and, in severe cases, convulsions (Pieper, Skeen, McClure, & Bourne, 1972). Again, symptoms such as these can be coded to provide a measure of the severity of withdrawal produced by a particular substance.

1.3.3 Craving

The DSM-5 now includes craving, defined as "an intense desire or urge for the drug that may occur at any time but is more likely when in an environment where the drug previously was obtained or used," as a diagnostic criterion for SUDs (American Psychiatric Association, 2013). Measuring this construct in animals may serve as a proxy for the excessive time or effort humans may spend in attempts to get and take a particular substance when addicted. Markou et al. (1993) propose that

craving is "reflected in enhanced effectiveness of the drug as a reinforcer." Based on this conceptualization, craving can be determined by assessing, in various ways, how much effort animals will exert to receive a drug of abuse. For example, animals may first be provided access to a drug on a fixed-ratio schedule. Once the animals' level of responding is consistent, investigators may increase the level of responding necessary for the receipt of the drug by switching to a progressive ratio reinforcement schedule. Likewise, craving may be indicated by how quickly an animal traverses an runway to gain access to a drug (Vanderschuren & Ahmed, 2013). Craving has also been measured in animals using an experiment based on the extinction process. In this procedure, animals are first trained to lever-press for access to a drug, then the experimenter removes the drug to assess how much the animal will continue to respond without reinforcement.

An additional approach to determining craving in animals is through the use of choice paradigms. During this type of test, subjects are allowed to choose between two stimuli, one of which is the drug, and depending on the test, selection of one stimulus may prohibit animals from accessing the alternative stimuli until the following trial. The principles of classical conditioning have also been employed to study craving in animals; the conditioned reinforcement paradigm includes the pairing of a drug and a conditioned stimulus (CS) during training. Depending on an animal's level or rate of responding for the CS when the drug no longer accompanies it, an animal is considered to demonstrate more or less craving.

Investigators can also learn about addictive behavior by observing an animal's behavior following a period of abstinence from a drug. For example, animals may demonstrate a change in intake of or responding for the drug when it is reintroduced after abstinence (Koob, 2000). This is called the "deprivation effect" and may be used to indicate the presence of strong cravings or as a model of relapse. Other methods used to measure craving include second-order schedule paradigms and conditioned place preference tests (see review by Markou et al., 1993).

1.3.4 Use Despite Consequences

The DSM-5 includes several criteria that may fit under the broader category of "use despite consequences." For example, criterion 6 reads, "the individual may continue substance use despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of the substance." Additionally, criterion 9 reads, "The individual may continue substance use despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by the substance (American Psychiatric Association, 2013)." To determine if animals would show persistent drug-seeking behavior despite adverse consequences, an experiment was designed in which animals were trained to lever press for an additional lever which, when pressed, would provide access to cocaine. After training with these contingencies, rats were presented with both a CS (a tone) and footshock. Later, drug seeking was tested in the presence of the CS. The results of this experiment showed that a subset of subjects continued to

exhibit drug-seeking behavior despite the presence of this cue. Interestingly, this effect was only observed after animals were given extended access to cocaine but not in animals given access to the drug for a more limited period (Vanderschuren & Everitt, 2004). Similarly, certain animals with extended access to ethanol continue to ingest the drug after a period of abstinence even when it is given a bitter taste by adding quinine (Wolffgramm, Galli, Thimm, & Heyne, 2000).

1.3.5 Relapse

Animal models have been helpful in identifying the various circumstances that may contribute to a relapse. For example, relapse has been elicited by stress, cues associated with the drug, as well as small doses of the drug (Deroche-Gamonet, Belin, & Piazza, 2004; Lynch, Nicholson, Dance, Morgan, & Foley, 2010). To determine the factors or conditions that may promote relapse, researchers have employed a reinstatement procedure which consists of allowing animals the chance to respond for access to a drug, subsequently removing the drug and assessing whether certain stimuli elicit responding again (Deroche-Gamonet et al., 2004; Lynch et al., 2010).

1.4 Overlaps Between Eating Disorders and Addictions

Eating and drug use are both motivating, appetitive behaviors with underlying similarities in their neural circuitry (Volkow & Wise, 2005), leading some researchers to investigate whether eating disorders may represent some form of an addictive behavior (Davis & Claridge, 1998; Kaye et al., 2013; Marrazzi & Luby, 1986). Although some do not consider this is a valid conceptual approach to the study of eating disorders (Wilson, 2010), there is support for this idea. From a reward perspective, food restriction, a feature of certain eating disorders, can increase the reinforcing effects of drugs of abuse (Carr, 2002). Eating disorders commonly begin during adolescence, a period of vulnerability for the development of addictive behaviors. Much like individuals with drug addiction, who forgo many activities and responsibilities in order to seek and consume drugs of abuse, individuals with eating disorders can adopt a similar pattern of behavior, with weight loss efforts, bingeing or rituals regarding food, and exercise occupying the majority of their time and energy. When food intake in AN does occur, it is often associated with anxiety, a symptom that is also often reported during periods of drug abstinence or withdrawal (Barbarich-Marsteller, Foltin, & Walsh, 2011). Likewise, eating disorder behavior is often engaged in despite serious consequences to one's health and is vulnerable to relapse. Thus, eating disorders share many commonalities with addiction.

Studies using animal models of eating disorders support the idea that aspects of addiction may be involved. In our laboratory, we have used a model of binge eating to show signs of "addiction" to sugar and other palatable foods (Avena et al., 2008),

Table 1.1 Summary of findings in support of sugar addiction in rats using an animal model of sucrose or glucose bingeing

Substance dependence	Animal model of sugar dependence
A. DSM-5	
Tolerance	Escalation of daily sugar intake Colantuoni et al. (2001)
Withdrawal	Somatic signs (teeth chattering, tremor), anxiety measured by plus maze, ultrasonic distress vocalizations Colantuoni et al. (2002), Avena et al. (2008)
Consuming more than intended	Deprivation effect Avena, Long, and Hoebel (2005)
B. Behavioral signs	
Locomotor cross-sensitization	Amphetamine Avena and Hoebel (2003)
Proclivity to consume other drugs of abuse	Alcohol Avena, Carrillo, Needham, Leibowitz, and Hoebel (2004)
C. Neurochemical changes in the NAc	
Repeated release of DA	Rada, Avena, and Hoebel (2005), Avena, Rada, and Hoebel (2006)
↑D_1 receptor binding	Colantuoni et al. (2001)
↓D_2 receptor binding	Colantuoni et al. (2001)
↑D_3 receptor mRNA	Spangler et al. (2004)
↓preproenkephalin mRNA	Spangler et al. (2004)
DA/ACh imbalance during withdrawal	Colantuoni et al. (2002), Avena et al. (2008)

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including signs of withdrawal, craving, and neurochemical changes in reward-related brain regions that are consistent with those seen when animals are given a drug of abuse (See Table 1.1). Likewise, rats prone to binge eating have been found more likely to traverse a shock grid for the receipt of palatable food than binge-resistant rats (Oswald, Murdaugh, King, & Boggiano, 2011), indicating use despite deleterious consequences. Clinical support for such findings comes in part from studies assessing food addiction using a self-report scale. For example, in a sample of obese individuals with BED, 41.5 % were considered to meet the criteria for "food addiction" (Gearhardt, White, Masheb, & Grilo, 2013). In terms of AN, it has been shown that ABA rats display withdrawal signs in response to an opioid antagonist (Kanarek, D'Anci, Jurdak, & Mathes, 2009). Future research incorporating models typically used to assess characteristics of addiction to study eating disorder pathology may shed additional light on similarities that may exist between these two disorders.

Several studies have also found increased substance use/abuse among eating disorder samples, particularly among individuals with bulimic symptoms (Krug et al., 2009; Root et al., 2010). Further, patients with substance use disorder and post-traumatic stress disorder who report bingeing have been found to demonstrate less success during drug abstinence (Cohen et al., 2010). Such findings suggest the

relevance of assessing and appropriately addressing the symptoms of both substance use disorders and eating disorders during treatment.

Conclusion

Animal models have been used to further our understanding of the symptoms associated with AN, BN, BED, as well as addiction. The clinical utility of these models lies in their ability to identify characteristics which may increase one's vulnerability to develop such symptoms, and to help researchers identify and explore the potential of promising treatment approaches. Further, the overlaps that have been identified between these two types of disorders present a possible guide for further study.

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The Relationship Between Feeding and Drug-Seeking Behaviors

2

Marilyn E. Carroll and Nathan A. Holtz

Abstract

This chapter focuses on the relationship between avidity for sweet substances and drug abuse using rats that were selectively bred for high (HiS) vs. low (LoS) saccharin intake. These rats serve as genetic models for several aspects of drug abuse such as initiation, maintenance, escalation, and relapse to drug seeking. Neurobiological differences in brain areas associated with drug and food reward underlie the behavioral differences. In addition to dietary compulsions, animal models of high vs. low novelty reactivity (HR vs. LR), novelty preference (HNP vs. LNP), impulsive choice (HiI vs. LoI), impulsive action (HI vs. LI), avidity for exercise (HiR vs. LoR), and attention to reward-related stimuli, such as sign- (reward-associated stimuli) vs. goal-tracking (reward) (ST vs. GT), also predict high vs. low drug seeking, respectively. The high-performing traits have some overlap in predicting addictive behavior, but in many respects they appear to be independent predictors of addictive behavior. In contrast, rats selected for low reward seeking are more reactive to stressful or aversive events associated with drugs and less likely to engage in drug seeking. These traits provide a model of resilience to drug abuse. Segregating individual differences into reward sensitive and aversion reactive may allow for customized addiction treatment. It is hypothesized that reward-sensitive individuals would be responsive to reward-replacement therapy, such as exercise, while aversion-reactive individuals may react more to negative outcomes for drug use. Initial data indicate better treatment success in the LoS (vs. HiS) and LoI (vs. HiI) rats, yet higher drug-seeking females respond better to treatment than males. Knowledge of specific

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vulnerability factors is important to designing maximally effective prevention and treatment strategies.

Keywords

Aversive effects • Drug seeking • Feeding • Impulsivity • Novelty preference/seeking • Reward substitution • Punishment • Sweet intake • Treatment • Vulnerability

2.1 Introduction

Drug abuse and feeding are related to biological, genetic, and environmental factors that play a role in determining when drug- or food-rewarded behavior becomes out of control and manifests itself as an addiction. Drug- and food-related addictions interact such that reduction in availability of one substance leads to overconsumption of another (Carr & Cabeza de Vaca, 2013; Carroll, Holtz, & Zlebnik, 2013), and drug- and food-rewarded behaviors are mediated by overlapping brain reward circuitry. Drug addiction and excessive eating leading to obesity, and the corresponding metabolic syndrome, are among the top causes of death in the USA and an enormous cost to society. This close relationship between feeding and drug seeking (addiction) has been studied for many years and has been reviewed previously at behavioral (e.g., Ahmed, 2005, 2012; Avena, 2010; Belin, Berson, Balado, Piazza, & Deroche-Gamonet, 2011; Belin & Deroche-Gamonet, 2012; Bocarsly & Avena, 2012; Carroll, 1999; Carroll, Holtz, & Zlebnik, 2013, Carroll, Morgan, Anker, Perry, & Dess, 2008) and neurobiological levels (e.g., Carr & Cabeza de Vaca, 2013; Johnson & Kenny, 2010; Levine, Kotz, & Gosnell, 2003a, b; Olsen, 2011; Pelchat, Johnson, Chan, Valdez, & Ragland, 2004; Volkow & Wise, 2005). The present review focuses on behavioral-genetic aspects of this interaction, specifically drug-seeking behaviors in rats that are *selectively bred* to consume excessive amounts of sweet substances (see reviews by Carroll et al., 2008, Carroll, Holtz, & Zlebnik, 2013), and rats that have been *selected* for other addiction-related behaviors such as novelty reactivity, novelty preference, impulsivity, exercise, and incentive salience of drug-related stimuli (see reviews by Carroll, Anker, Mach, Newman, & Perry, 2010, Carroll, Johnson et al., 2013; Carroll, Mach, LaNasa, & Newman, 2009).

A major difference between excessive food intake and drug addiction is that food is necessary for survival, while recreational drugs are not. However, the tenacity of food and drug addiction is similar in strength, factors that initiate and maintain these self-destructive behaviors are similar, both disorders are highly treatment resistant, they readily substitute for each other, and both food and drug addiction result in similar rates of morbidity and mortality. When considering addiction using animal models, several laboratories have attempted to use addiction criteria described for diagnosing human drug abusers according to DSM-IV and DSM-5 criteria (Deroche-Gamonet, Belin, & Piazza, 2004; Vanderschuren &

Ahmed, 2013). This approach has been helpful in identifying mechanisms for disordered behavior, developing treatments, and translating animal research findings to clinical practice. In an increasing number of studies, these criteria are being applied in the design of animal models that allow us to learn more about the development of addictive behavior and interventions and treatments that will translate to applications for changing pathological human behaviors.

The goal of this chapter is (1) to briefly review seminal research on the relationship between feeding and drug seeking; (2) to examine a genetic relationship between drug-seeking behavior and feeding-related traits using rats that were selectively bred for high (HiS) and low (LoS) saccharin intake. i.e., HiS rats also eat more food, weigh more, and show more drug seeking than their low sweet-preferring (LoS) counterparts; (3) to determine how these genetic influences relate to other individual differences that predict vulnerability to drug abuse, and to recognize their commonalities and underlying neurobiology; (4) to examine differences in HiS and LoS rats' reactivity to stress and aversive events; and (5) to discuss behavioral and pharmacological treatments for drug abuse as they relate to the underlying vulnerability factors and how these factors influence treatment effectiveness.

2.2 Models of Addiction

An important guideline for studying addictive behavior in the animal laboratory is to closely model the behavior in humans. In considering hedonic overindulgence in food or binge eating as an addiction, there are similar diagnostic criteria for drug dependence and problematic food intake (Gearhardt, Corbin, & Brownell, 2009; Gearhardt, Davis, Kuschner, & Brownell, 2011), and DSM-IV criteria for substance dependence, such as binge eating or overindulgence in preferred (sweet) high-caloric foods, have been applied in the animal models. In animal studies, behavioral models have been designed to emulate DSM-IV drug addiction criteria (Deroche-Gamonet et al., 2004, Vanderschuren & Ahmed, 2013), and there are parallels to measures of the DSM-IV binge eating disorder (BED) criteria described in humans. In fact, instruments such as the Yale Food Addiction Scale (YFAS) (Gearhardt et al., 2009) that has been used to link binge eating and food addiction in humans show that nearly half of BED patients meet criteria for "food addiction" (Bocarsly & Avena, 2012; Cassin & von Ranson, 2007; Gearhardt, White, & Potenza, 2011). Drug abuse has been modeled in rats (Ahmed, 2012; Belin et al., 2011; Belin & Deroche-Gamonet, 2012; Carroll & Meisch, 2011; Jupp, Caprioli, & Dalley, 2013; Vanderschuren & Ahmed, 2013) and nonhuman primates (Foltin, 2013) using DSM-IV criteria for substance use disorders (SUD) such as (1) tolerance (Perry, Dess, Morgan, Anker, & Carroll, 2006), (2) difficulty limiting use (Perry et al., 2006), (3) excessive time spent seeking (Perry et al., 2006), (4) impaired control over use (Lynch, Arizzi, & Carroll, 2000), (5) activities given up or drug use preferred over other activities (Carroll et al., 2008), (6) continued use despite negative consequences (Holtz, Anker, & Carroll, 2013), and (7) withdrawal signs

Table 2.1 HiS vs. LoS rats and criteria for drug addiction

Criteria for addiction	HiS vs. LoS	References
Tolerance	HiS > LoS	Perry et al. (2006)
Difficulty limiting use	HiS > LoS	Perry et al. (2006)
Excessive time seeking	HiS > LoS	Perry et al. (2006)
Impaired control over use	HiS > LoS	Lynch et al., (2000)
Activities given up	HiS > LoS	Carroll et al. (2008)
Use despite punishment	HiS > LoS	Holtz, Anker, Regier, Claxton, & Carroll (2013)
Withdrawal	LoS > HiS	Dess et al. (2000, 2005); Radke, Zlebnik, & Carroll (2014)

after termination of use (Dess et al., 2000; Dess, O'Neill, & Chapman, 2005). As summarized in Table 2.1, rats that were selectively bred for sweet preference (HiS) scored higher on criteria for addictive behavior than their low saccharin-preferring counterparts (LoS), except LoS rats exceeded HiS rats on withdrawal measures. These criteria that are shown in Table 2.1, when adapted to animal models (Vanderschuren & Ahmed, 2013), include tolerance, impaired control over use, difficulty limiting use, excessive time spent seeking drug, activities given up (or replaced) by drugs, and continued use despite punishment. The HiS animals exceeded LoS in behaviors that were developed to emulate the DSM criteria that are used to describe human addiction.

To evaluate drug-seeking behavior using these criteria for drug addiction, we have used several animal models to mimic the establishment of drug use in humans and its progression through regular use to escalation and compulsive use. For example, studies have modeled *impulsive action*, inability to withhold responding for long periods of time each day, and *impulsive choice*, choosing a smaller amount of drug immediately over a larger amount after a delay, or a smaller probability of a sooner reward vs. a larger probability of a delayed reward (see reviews by Carroll & Meisch, 2011; Carroll et al., 2010).

2.3 Drug Seeking in Rats Selectively Bred for High and Low Saccharin Intake

With these models of addiction, we have pursued a line of research that allows us to address the relationship between feeding and drug-seeking behavior as it is related to genetic background by studying rats that have been selectively bred to prefer sweet substances. These rats were originally derived from the Sprague-Dawley strain, and the HiS rats eat more and weigh more than their low sweet-preferring counterparts (LoS) or outbred control rats from the Sprague-Dawley background strain (Carroll et al., 2008). Thus, the HiS rats could also serve as an animal model for overconsumption of rewarding substances, particularly sweet substances, and drugs of abuse. The rats we are reporting data on were initially selectively bred for

high (HiS) and low (LoS) saccharin intake by Dr. Nancy Dess at Occidental College in Los Angeles, CA. Progenitor rats were tested for their intake of saccharin during a 24 h 2-bottle test with water concurrently available, and their 24-h saccharin intake was compared to 24 h water intake from a previous day when only water was available in the two bottles. Rats exhibiting extremely high saccharin preferences were mated together, and rats with low saccharin preferences or saccharin aversion (less consumption than water) were mated together. Breeding continued with HiS pairs and LoS pairs, and initially Dess and coworkers studied differing levels of emotionality and taste preferences in these rats (Dess, 2001, Dess et al., 2000; Dess & Minor, 1996). Subsequently, they studied alcohol intake and found that the HiS rats exceeded the LoS rats in their consumption, but LoS rats were more affected by alcohol withdrawal effects (Dess et al., 2005).

Our laboratory obtained some of the Occidental HiS and LoS rats and demonstrated that the HiS rats also showed elevated cocaine intake compared to LoS rats during initiation of drug self-administration in drug-naïve rats and in cocaine-experienced rats under a progressive ratio (PR) schedule (Carroll, Morgan, Lynch, Campbell, & Dess, 2002). Subsequent studies extended these findings with cocaine to several phases of the addiction process (see reviews by Carroll et al., 2008, Carroll et al., 2010; Carroll, Holtz, & Zlebnik, 2013). These reviews described studies indicating that HiS rats exceeded LoS rats during initiation or acquisition of cocaine self-administration (Carroll et al., 2002), escalation of bingeing on cocaine during long (LgA) vs. short (ShA) access (Perry et al., 2006). The HiS rats also showed more resistance to extinction when cocaine was replaced with saline and reinstatement of responding (relapse) that occurred after extinction when drug access had been terminated and the rats were later given experimenter-administered priming injections of cocaine, or a stressor—yohimbine (Holtz, Anker, & Carroll, 2013; Perry et al., 2006). It has recently been shown that HiS rats similarly exhibited binge-like behaviors when given access to fat- or sugar-based substances (Yakovenko, Speidel, Chapman, & Dess, 2011).

It is important to note that when the HiS and LoS rats' drug-seeking behavior was examined in these models of drug abuse, they were saccharin naïve. Their HiS or LoS status was based entirely on their breeding history, and they were not given access to saccharin prior to drug exposure. This allowed us to verify the breeding status after the drug-seeking experiments were completed, because testing their saccharin intake to verify the HiS or LoS phenotype might interfere with their drug self-administration behavior (Carroll, Lac, & Nygaard, 1989). At least 2 weeks after the end of the drug self-administration experiments, when rats were returned to the home cage with lab chow and water freely available, they were tested for their saccharin preference score to verify their selection status, and the phenotypes were confirmed. Across many studies, on average, The HiS rats achieved a score ranging from 25.2 to 39.6 for HiS males and females, respectively, and 5.2 to 7.6 for LoS males and females, respectively (Carroll et al., 2008). An interesting result of comparing saccharin preference scores over several studies, however, was that the rats that had more access to cocaine before their saccharin testing showed lower saccharin scores (less saccharin preference) than those with less cocaine

experience (Carroll et al., 2008). Thus, the rewarding effect of prior exposure to cocaine may have produced a contrast effect and reduced the hedonic value of saccharin (see Grigson & Twining, 2002). This provided further evidence for the interaction of hedonic effects of food and drugs and addresses the DSM criteria of *other activities given up* (Table 2.1).

The selectively bred HiS and LoS rats' corresponding high and low drug seeking has also been demonstrated in outbred rats that were screened for high or low sweet intake, and they subsequently showed high vs. low drug seeking, respectively. For example, Bell, Gosnell, Krahn and Meisch (1994) separated Wistar rats into "high," "intermediate," or "low" groups based on their saccharin intake, and they found that the groups maintained their rank order on measures of alcohol consumption. Also, rats screened as saccharin/sweet likers (SL) consumed more alcohol (Gahtan, Labounty, Wyvell, & Carroll, 1996) and morphine (Gosnell, Krahn, Yracheta, & Harasha, 1998; Gosnell, Lane, Bell, & Krahn, 1995), than saccharin/sweet dislikers (SDL). Similarly, rats selected for high sucrose feeding (HSF) consumed more amphetamine and acquired cocaine self-administration faster than their low-sucrose feeding (SLF) counterparts (DeSousa, Bush, & Vaccarino, 2000; Gosnell, 2005).

The connection between avidity for dietary sweets and substance use disorders (SUD) has also been reported in human populations, such as those who abuse alcohol (Chester, Blose, & Froehlich, 2003; Kampov-Polevoy, Garbutt, & Janowsky, 1999; Kampov-Polevoy, Tsoi, Zvartau, Neznonov, & Khalitov, 2001; Wronski et al., 2007), cocaine (Janowsky, Pucilowski, & Buyinza, 2003), nicotine (Pepino & Mennella, 2007; Pomerleau, Garcia, Drewnowski, & Pomerleau, 1991), and opioids (Weiss, 1982). In these studies drug users/abusers experienced greater hedonic effects from sweets and consumed more sweets than those who do not abuse these drugs. These parallels between the selectively bred rats, outbred rats, and human self-report studies suggest that the feeding and drug-seeking behaviors are moderated by genetically mediated traits (Uhl, Drgon, Johnson, & Liu, 2009) and common neural mechanisms (Carroll et al., 2008; Holtz & Carroll, 2013).

It is also important to note that the findings of differential vulnerability to drug abuse in the HiS and LoS rats due to their selective breeding history are not unique to these sweet-preferring/non-preferring phenotypes (e.g., HiS, LoS). In recent years, there have been numerous examples of individual differences on other dimensions that correspond with high vs. low drug seeking. As Table 2.2 indicates, drug abuse liability is also predicted by novelty reactivity (Davis et al., 2008; Piazza et al., 1989), impulsive choice (Perry, Nelson, & Carroll, 2008), impulsive action (Dalley et al., 2007), shock avoidance (Fattore et al., 2009), incentive stimulus reactivity (Saunders, & Robinson, 2013), sex and hormonal conditions (Carroll & Anker, 2010), age (O'Dell et al., 2006), and exercise avidity (Ferreira et al., 2006). Similar differences have also been found across different rat strains such as Lewis and Fischer 344, which are compared for other characteristics, like differences in hypothalamic-pituitary-adrenal axis responses (Kosten & Ambrosio, 2002; O'Dell et al., 2006).

Table 2.2 indicates that high performers are also more drug abuse prone relative to corresponding low-performing individuals that are resistant to drug-seeking

Table 2.2 Risk factors for drug seeking, aversion to drugs, and response to treatment

Vulnerability	Prone	Resistant	References
Sweet intake	HiS	LoS	Carroll et al. (2008)
Impulsive choice	HiI	LoI	Carroll et al. (2010)
Impulsive action	HI	LI	Dalley et al., 2007
Novelty reactivity	HR	LR	Piazza, Deminiere, Le Moal, and Simon (1989)
Bred for novelty reactivity	bHR	bLR	Davis, Clinton, Akil, and Becker (2008)
Avoidance	RHA	RLA	Fattore, Piras, Corda, and Giorgi (2009)
Exercise	HiR	LoR	Larson and Carroll (2005), Ferreira et al., 2006
Sign/goal tracking	ST	GT	Saunders and Robinson (2013)
Sex	Female	Male	Carroll and Anker (2010)
Female hormones	Estrogen	Progesterone	Anker and Carroll (2010)
Age	Adolescent	Adult	O'Dell et al. (2006)
Lewis/Fischer	Lewis	Fischer	Kosten and Ambrosio (2002)

behavior. Some of the low performing individuals (e.g., LoS, LoI, males, and Fischer rats) have been shown to be more responsive than their high preferring counterparts to aversive effects of drugs. Differences in these lines of rats shown in Table 2.2 are not limited to drug seeking and self-administration. While these are the variables most studied, the selectively bred HiS vs. LoS rats exhibit a range of other behaviors that are associated with drug addiction, such as (1) impulsive behavior, (2) dysregulation of intake during self-selection of high vs. low drug doses, (3) and sensitization of drug-induced locomotor activity. For example:

1. The HiS and LoS rats were tested for their impulsivity of choice for a small immediate vs. a larger delayed reward using food and cocaine with a delay-discounting task for food or IV cocaine. In this task a response on one lever was rewarded by one food pellet or a small cocaine infusion after a short delay, while responding on another lever was rewarded by three food pellets or three times the dose of the cocaine infusion following a long delay. HiS rats were more impulsive than LoS rats for food on a delay-discounting task (Perry et al., 2008), and HiS rats were also more impulsive for cocaine than LoS rats on a go/no-go task (Anker, Gliddon, & Carroll, 2008), wherein they showed more no-go responding (i.e., impulsive action). HiS rats also exceeded LoS rats on another measure of impulsive action—responding that occurs during the drug infusion in a drug self-administration paradigm in which drug is available under a fixed-ratio 1 (FR 1) schedule. This ineffective responding is counted after the infusion pump begins to deliver drug, and during the infusion and the timeout period after the infusion. These ineffective responses that occur after the infusion begins are counted but have no consequences, and when they were compared, they were higher in HiS rats than LoS rats (Carroll, Holtz, & Zlebnik, 2013). Thus, food and cocaine were not only more rewarding for HiS than LoS rats, but HiS rats were more impulsive than LoS rats in their food- and cocaine-seeking behavior.

2. Another behavior that is related to drug seeking is regulation or dysregulation of drug dose, and that differs between HiS and LoS rats. For example, when rats have a choice between responding on one lever that increases the next dose of cocaine and responding on the other lever that decreases the next dose of cocaine, their ability to regulate their dose is determined by a negative correlation between the dose they received and the time before a response occurs for the next infusion. Thus, high or low doses of cocaine were not necessarily preferred and seemed to be nonsystematically chosen by the rats, but there was a very precise regulation of the amount consumed per unit of time. A comparison of HiS and LoS rats in this paradigm showed that HiS rats did not regulate their dose quite as precisely as LoS rats, and HiS rats spent more time than LoS rats perseverating on the lever that increased the dose, thereby self-administering a larger number of the highest doses than the LoS rats (Carroll, Anderson, & Morgan, 2007b; Lynch et al., 2000, Lynch & Carroll, 1999, Lynch, LaBounty, & Carroll, 1998).
3. HiS and LoS rats also showed differences (HiS > LoS) in sensitization to cocaine-induced locomotor activity, a neuronal adaptation that is thought to be related to the rewarding effects of cocaine (Robinson & Berridge, 1993). For example, HiS rats exceeded LoS rats on cocaine-induced locomotor activity and sensitization to repeated cocaine injections after five daily injections of cocaine, and when an additional injection was given 2 weeks later and compared to the first injection (Carroll, Anderson, & Morgan, 2007a).

2.4 Neurobiological Differences in HiS and LoS Rats

The consistent differences between food seeking and drug seeking between HiS and LoS rats using several measures of motivated behavior suggest that the HiS and LoS differences are related to underlying differences in neurobiology of reward circuitry. In recent research, initial attempts have been made to examine differences in neuronal activity in the HiS and LoS rats by examining c-Fos reactivity in brain areas associated with drug reward. In these studies drug-naïve HiS and LoS rats were given one injection of 15 mg/kg cocaine HCl or saline (controls) and sacrificed 20 min later. In one study several brain areas (orbital frontal cortex, cingulated gyrus 1, nucleus accumbens shell, and dorsomedial and dorsolateral caudate putamen), associated with cocaine's rewarding effects, were examined for c-Fos counts (fold change from the cocaine-treated to the saline-treated group). Results indicated that the LoS rats showed higher neuronal activity than the HiS rats in the nucleus accumbens shell and dorsomedial and dorsolateral caudate putamen. Thus, HiS rats that are more vulnerable to cocaine-seeking behavior at several phases of the addiction process than LoS rats also exhibit less neuronal reactivity compared to LoS rats, after one injection with 15 mg/kg cocaine, in brain areas associated with food and drug reward. However, since it was an experimenter-injected dose and not self-administered, and the dose was high, it is not clear whether it was having a rewarding or aversive consequence.

In another study of HiS and LoS rats, the neurobiological connection between food and drug seeking was examined by counting the number of orexin-A-positive cells in the lateral hypothalamus and perifornical areas in HiS and LoS rats that had been injected with 15 mg/kg of cocaine or saline. Orexin-A is a neuropeptide that stimulates the motivation to ingest preferred substances and mediates dopamine release that affects motivation for cocaine and other highly valued rewards, including food. Orexin-A antagonists reduce intake of highly palatable foods in rats (Bocarsly & Avena, 2012; Cason & Aston-Jones, 2013; Kotz, 2006) and humans (Cason et al., 2010), and cue-induced reinstatement of drug-seeking behavior in rats via actions in the mesolimbic dopamine system (Espana et al., 2010; Moorman & Aston-Jones, 2009; Shoblock et al., 2011; Smith, Tahsili-Fahadan, & Aston-Jones, 2010). After an injection of cocaine or saline, HiS rats had more orexin-positive cells than LoS rats, suggesting that the HiS rats had higher endogenous orexin, and this may be related to their higher motivational states for sweet substances, alcohol, cocaine, and other drugs than LoS rats (Holtz, Zlebnik, & Carroll, 2012). Initial evidence suggests that the orexin-A antagonist reduces cue-induced drug-seeking behavior (Smith, See, & Aston-Jones, 2009).

2.5 Other Individual Differences and Drug Seeking

The findings of a relationship between different feeding preferences in HiS and LoS rats and corresponding differences in drug-seeking behavior are strong evidence for an innate connection between feeding and drug-seeking behavior. However, recent reports have also revealed a wide array of other motivated behaviors or traits that are also associated with drug abuse and eating disorders (ED), such as (1) impulsivity in humans (e.g., Dawe & Loxton, 2004) and in animal studies (Carroll et al., 2010); (2) novelty reactivity (Piazza et al., 1989), novelty preference in animals (Belin et al., 2011), or novelty/sensation seeking in humans (Kreek, Nielsen, Butelman, & Laforge, 2005), traits that are highly predictive of drug abuse; (3) physical activity (Larson & Carroll, 2005); and (4) sign vs. goal tracking in regard to attention to stimuli (incentive salience) associated with food reward (Flagel et al., 2010; Flagel, Watson, Akil, & Robinson, 2008). Some of the following examples of studies involving these vulnerability factors have used both rats selected for particular traits and rats selectively bred for those traits (see review by Carroll, Holtz, & Zlebnik, 2013).

1. *Novelty reactivity and preference*. Early studies selected rats for high (HR) vs. low (LR) novelty reactivity in a novel environment and showed that the HR rats initiated drug seeking more than the LR rats (Piazza et al., 1989). In subsequent work, similar results were found in rats selectively bred for high or low (bHR, bLR) reactivity in a novel environment (Cummings et al., 2011; Davis et al., 2008; Kabbaj, 2006; Kabbaj, Devine, Savage, & Akil, 2000). Recently, rats have been selected for novelty preference in a free-choice paradigm in which there is a choice between a familiar and novel environment, and those that are selected for the high-novelty-preferring (HNP) phenotype show more impulsive and

compulsive drug seeking compared to low-novelty-preferring (LNP) rats (Belin et al., 2011).

2. *Impulsive choice and impulsive action*. Several studies have compared high vs. low impulsive behavior determined by delay discounting (HiI, LoI) (see reviews by Carroll et al., 2010; Perry & Carroll, 2008) or with a 5-choice serial reaction time task (5-CSRTT) (HI, LI) that detects prepotent responding for food reward (Dalley et al., 2007). There is extensive evidence that rats selected for high and low impulsivity have similar propensities and disinterest, respectively, in drugs of abuse as shown in HiS and LoS rats (Carroll et al., 2010, Carroll, Holtz, & Zlebnik, 2013). For example, when HiS and LoS rats were compared on an impulsive choice measure, such as a delay-discounting task for food and cocaine, HiS rats were more impulsive than LoS rats for food (Perry, Nelson, Anderson, Morgan, & Carroll, 2007). However, under a go/no-go task for impulsive action, HiS rats were more impulsive than LoS for cocaine (Anker et al., 2008). There is extensive evidence that rats selected for high and low impulsivity have similar propensities and disinterest, respectively, to drugs of abuse as shown in HiS and LoS rats (Carroll et al., 2010); however, the sweet-preferring and impulsive phenotypes are not an expression of the same underlying factor, as HiS and LoS rats are not consistently high and low impulsive, and HiI and LoI rats do not show consistent differences in saccharin preference scores. Thus, the sweet-preferring and impulsive phenotypes are not completely overlapping, and they may represent different genetically-determined traits.
3. *Physical activity*. A propensity for physical exercise is another factor that is related to addictive behavior such as avidity for physical activity or exercise (Larson & Carroll, 2005; Olsen, 2011), and others have shown that physical activity is a genetically mediated characteristic (Bauman et al., 2012) that predicts SUD. For example, Larson and Carroll (2005) reported that rats selected for high wheel running (HiR) subsequently self-administered more cocaine than rats selected for low wheel running (LoR), and in a reinstatement (relapse) paradigm with HiR and LoR rats, Larson and Carroll (2005) also showed that HiR rats exhibited greater reinstatement of lever pressing that was previously reinforced by cocaine than LoR rats. Similarly, Ferreira et al. (2006) reported that wheel running in a heterogeneous rat population was positively related to amphetamine-induced locomotor activity, a behavior that is correlated with the initiation of drug seeking. Thus, avidity for exercise, like seeking preferred foods, predicts drug abuse in animal models.

   However, as in the case of preferred food, exercise functions as an economic substitute for drug abuse and seems to have a therapeutic function in treating drug abuse. For example, exercise reduces cocaine self-administration, and cocaine availability reduces exercise (Cosgrove, Hunter, & Carroll, 2002). Initial studies indicate that the interaction of drug seeking and exercise is likely related to common neurobiological reward mechanisms. (Zlebnik, Hedges, Carroll, & Meisel, 2013). Earlier work by Kanarek and coworkers (Kanarek, D'Anci, Jurdak, & Mathes, 2009; Kanarek, Gerstein, Wildman, Mathes, & D'Anci, 1998) indicated that exercise and drug seeking may be mediated by

the endogenous opioid system. A difficulty in considering exercise as a treatment for drug abuse, in terms of a substitute for drug addiction, is that in a small subset of individuals, too much exercise can become addictive and result in activity-induced anorexia (Davis, Kennedy, Ravelski, & Dionne, 1994). This has also been modeled in rats (Spear & Hill, 1962).

4. *Sign* vs. *goal tracking*. Another dimension of individual differences that has recently been reported to be predictive of addictive behavior involves rats selected for attention to cues associated with food reward (sign tracking—ST), or the food receptacle (goal tracking—GT). For the ST rats, stimuli associated with food reward have more incentive salience, while goal tracking rats focus their attention on the goal or food reward and its delivery receptacle (Flagel et al., 2008). These studies found that ST rats, for which stimuli associated with food reward have more incentive salience, self-administer more cocaine and show more drug-primed reinstatement of cocaine seeking compared to GT rats that focus their attention on the food reward and its delivery receptacle (Saunders & Robinson, 2011a, b; Saunders, Yager, & Robinson, 2013). Distinctions between stimulus (sign) and reward (goal) seeking in rats have not yet been associated with parallels for these attentional differences in humans, but recent studies with animal models suggest there is some overlap between impulsive and high-novelty-seeking characteristics. Thus, as discussed regarding Table 2.2, these relatively recent findings on ST and GT individuals might indicate another behavioral dimension that predicts high vs. low probability for addictive behavior. While this dimension could have some overlap with impulsive behavior, further work is needed to determine whether it is a unique trait that is predictive of drug abuse in humans and other animals.

The high performing phenotypes on all of these measures, compared with the low performing phenotypes, exhibited increased drug-seeking behavior using several models and phases of the addiction process. Thus, sweet preference is one example of several forms of nondrug reward-seeking traits that predict higher levels of drug seeking. There are other biological factors that determine vulnerability to drug abuse and predict high levels of drug seeking, such as sex (female) and age (adolescent), and these factors add to the selected or selectively bred addiction-prone phenotypes to predict even higher levels of drug-seeking behavior, or in the case of low drug preferring attributes, more resilience. Thus, avidity for palatable foods, as shown in the HiS rats, is only one example of several forms of reward-seeking traits in rats that predict high rates of drug-seeking behavior. However, sweet preference seems to be one of the strongest and most reliable traits that predict drug abuse, and it may be related to the interchangeability of food and drug addiction. Corresponding evidence has also been reported in humans with impulsivity as a predictor of drug abuse (Yi, Mitchell, & Bickel, 2010).

As indicated in Table 2.2 there are also several other strain or line differences in rats that predict drug abuse, and some have corresponding differences in feeding behavior. For example, there are rats that have been selected or bred to have high vs. low fear, anxiety, and emotionality, and these rats parallel the LoS

vs. HiS and LoI vs. HiI rats in terms of opposite relationships between aversion sensitivity and amount of drug self-administered (Holtz & Carroll, 2013). Also, Lewis (LEW) rats exhibit more drug seeking than Fischer (F344) rats, high alcohol consuming (HAC) rats consume more alcohol than low alcohol consuming (LAC) rats, and Roman high avoidance (RHA) rats consumed more ethanol compared to Roman low avoidance (RLA) rats (Guitart-Masip et al., 2006; Manzo et al., 2012) and LAC rats. However, the RLA and LAC rats have higher measures of stress reactivity, suggesting that rats that are avid drug seekers seem to be resilient to stress and aversive consequences of drug taking, while less drug addiction-prone rats are more susceptible to stress and aversive consequences of drug self-administration.

2.6 Reactivity to Aversive Events Associated with Drug Seeking in HiS and LoS Rats

Early findings by Dess and colleagues showed that while HiS rats consumed more of a variety of palatable substances such as sugars, saccharin, polycose, and salt than LoS rats, LoS rats were responsive in their aversion to bitter components of ethanol and taste mixtures (Dess, 1993, 2000; Thiele, Badia-Elder, Keifer, & Dess, 1997). Subsequent work by Dess and colleagues (2005) on the HiS and LoS rats and recent studies from our laboratory (Carroll, Holtz, & Zlebnik, 2013; Holtz & Carroll, 2013) have shown that the interaction between feeding behavior and drug seeking is more complicated than substitution of one rewarding substance (e.g., drug) for another (sweet food), and in fact, the relative reactivity to the aversive effects of ingested substances has an important role in the HiS/LoS difference in drug taking in rodent strains. Recent evidence suggests that the balance of rewarding and aversive effects is important to take into account when assessing addiction liability (Riley, 2011; Verendeev & Riley, 2013). Table 2.2 summarizes examples of high vs. low drug-abuse-vulnerable rat lines and indicates that the low drug-seeking counterparts of each pair of lines have a greater reaction to aversive events than their high reward-seeking counterparts.

While the HiS rats inform us about general vulnerability characteristics that predict drug-seeking behavior, the LoS rats provide valuable information about resilience to and avoidance of addictive behaviors involving both food and drugs. Initial work by Dess and colleagues (Dess et al., 2000; Dess & Minor, 1996; McLaughlin, Dess, & Chapman, 2011) and in our laboratory (Carroll, Holtz, & Zlebnik, 2013) indicates that LoS rats are more reactive to aversive stimuli, such as food restriction, than HiS rats. LoS rats are also more severely affected by the adverse effects of withdrawal of rewarding substances such as ethanol, glucose (Dess, O'Neill, & Chapman, 2005; Radke, Zlebnik, & Carroll 2014; Dess, Badia-Elder, Thiele, Kiefer, & Blizard, 1998; Yakovenko et al., 2011), or food (McLaughlin et al., 2011); LoS rats also react more than HiS rats to brief intermittent bursts of white noise (acoustic startle) (Dess et al., 2000), and during ethanol withdrawal, Dess et al. (2005) also used ethanol withdrawal-induced conditioned

taste aversion (CTA) as an indicator of the aversive effects of withdrawal in HiS and LoS rats, and the CTA was significantly greater in LoS than HiS male rats. These studies were expanded by examining the effects of stress (inescapable foot shock) on startle amplitude in the HiS and LoS rats, and results indicated a greater effect on startle amplitude on LoS (vs. HiS) rats (Gonzales, Carrett, Chapman, & Dess, 2008).

In a recent study, spontaneous and naloxone-precipitated morphine withdrawal effects were measured in HiS and LoS rats using elevation in intracranial self-stimulation thresholds, and LoS rats' thresholds were more elevated than HiS rats' indicating greater aversion to morphine withdrawal in LoS than HiS rats. These effects of drug withdrawal in HiS and LoS rats have recently been found with forced glucose abstinence (withdrawal) (Yakovenko et al., 2011). Rats were given extended access to glucose, and escalation of glucose intake was positively correlated with an increase in acoustic startle responding in the LoS vs. HiS rats. Similar findings with drug and glucose withdrawal in HiS vs. LoS rats are consistent with previous reports of parallel findings in studies of drug dependence, dysregulation of food intake, and food addiction (Avena, Long, & Hoebel, 2005; Avena, Rada, & Hoebel, 2006; Blumenthal & Gold, 2010).

Another approach to examining differences in HiS vs. LoS rats with regard to aversive effects has been to train rats to self-administer IV cocaine infusions and, after behavior stabilizes, to punish the cocaine-taking behavior by adding an aversive stimulus, histamine, to the cocaine solution. Thus, cocaine self-administration was punished by allowing the rats to self-administer a dysphoric consequence, histamine, during cocaine self-administration (Holtz, Anker, Regier, Claxton, & Carroll, 2013). First, a cocaine-only baseline was obtained for ten stable daily 2-h. sessions, followed by 10 days of cocaine + histamine, and subsequently, there was a 20-day return to cocaine only. Figure 2.1a indicates that both HiS and LoS rats reduced their cocaine self-administration by more than half during histamine treatment, but LoS rats were very slow to recover during the first 15 days of return to cocaine alone, while HiS rats returned to pre-histamine baselines in 3 days or less. Figure 2.1b illustrates the delay in recovery of the cocaine baseline over 2 weeks in the LoS rats. Thus, the aversive effects of punishment had similar aversive effects as stress measures that were previously discussed, in that LoS rats were more affected by aversive events than HiS rats.

2.7 Treatment Models in HiS and LoS Rats

The ultimate goal in understanding vulnerability to drug abuse, and how it relates to aberrant feeding patterns, is preventing the escalation of food or drug use by delivering treatment to those with food or drug addiction. While much has been learned about vulnerability factors, and their interactions, there are almost no clinical data on treatment in high vs. low vulnerable groups. One area that has been studied in this regard shows that addictive behavior is related to sex differences (F > M), and ovarian hormonal cycles (follicular > luteal) (see reviews

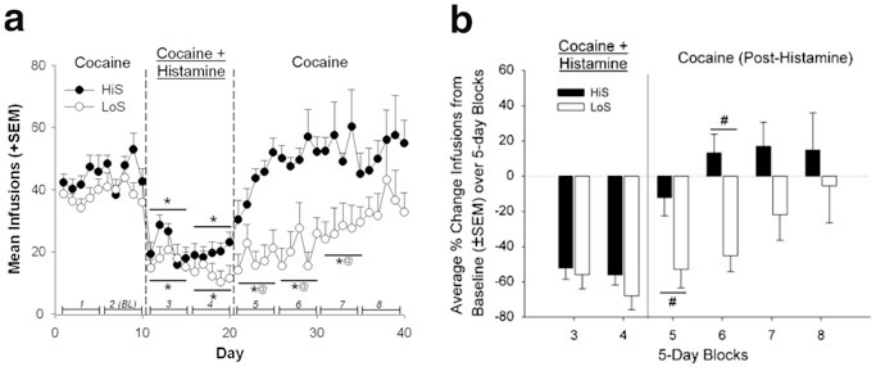

Fig. 2.1 Mean daily cocaine infusions (± SEM) are presented over 40 days in rats that were (**a**) selectively bred for high (HiS) or low (LoS) on measures of saccharin consumption or (**b**) high (HiI) or low (LoI) impulsive based on a delay-discounting task with food reward. The first 10-day period represents a stable baseline period when only cocaine (0.4 mg/kg) was available under a fixed-ratio 1 (FR 1) schedule. During days 11–20, histamine (4 mg/kg/infusion) was added directly to the cocaine syringe, and subsequently on days 21–40 only cocaine was available. Filled symbols indicate the HiS or HiI groups, respectively, and open symbols represent the LoS and LoI groups. An *asterisk* indicates a significant difference from baseline block 2 (Days 6-10), and @ indicates days when there were significant differences between the phenotypes at the $p < 0.05$ level (reprinted with permission from Holtz, Anker, et al., 2013)

by Anker & Carroll, 2010, 2011). Initial studies indicate that females exceed males during all phases of drug abuse that are modeled in the laboratory, except during withdrawal when males show more severe withdrawal effects than females (Carroll, Mach, et al., 2009), and in females withdrawal severity varies with phase of the menstrual cycle (Carroll, Johnson et al., 2013). In the few animal studies of sex differences in treatment effects, females were also more responsive to both behavioral and pharmacological treatments than males (Anker & Carroll, 2010, 2011; Carroll & Anker, 2010). In treatment of women for cigarette smoking, treatment also varies with phase of the menstrual cycle and when during the cycle the quit attempt is initiated (Allen, Bade, Center, Finstad, & Hatsukami, 2008; Franklin et al., 2007; Mazure, Toll, McKee, Wu, & O'Malley, 2011).

Much less is known about differential treatment effects with other individual differences such as HiS and LoS, HiI and LoI; however, in a few treatment studies that have recently been completed, it appears that the low drug-abuse-vulnerable phenotypes (LoS, LoI) were more responsive to treatment than their high vulnerable counterparts. For instance, as indicated in Fig. 2.2a, when HiS rats were allowed to self-administer cocaine and escalate their intake over 6-h sessions, progesterone (which functions as a $GABA_A$ modulator) treatment initially reduced cocaine infusions in LoS rats, but increased cocaine in HiS rats at the end of the 21-day escalation period (Anker, Holtz, & Carroll, 2012). Similarly as indicated in Fig. 2.2b, baclofen, a $GABA_B$ agonist, reduced cocaine infusions throughout the 21-day escalation phase in LoS rats and increased cocaine infusions during the last

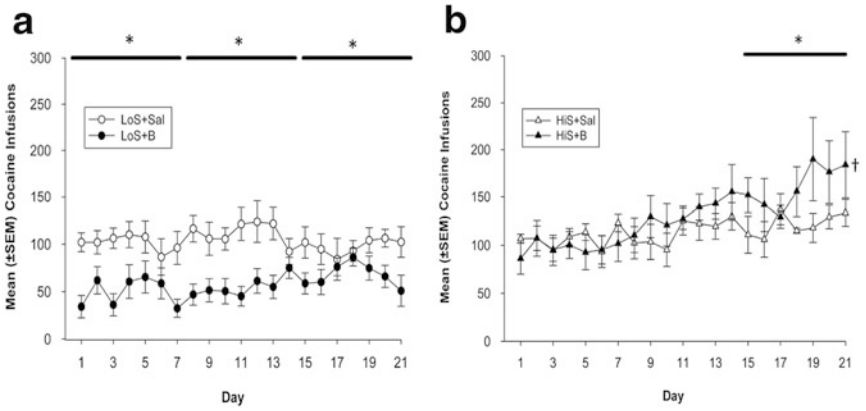

Fig. 2.2 Percent change of infusions self-administered by HiS and LoS rats are averaged into 5-day blocks and compared to the 5-day baseline average. The *hash* sign indicates phenotype differences in percent change of infusions self-administered between the phenotypes compared to baseline at the $p < 0.05$ level (reprinted with permission from Holtz, Anker, et al., 2013)

few days in the HiS rats (Holtz & Carroll, 2011). In a recent study of allopregnanolone (progesterone metabolite), treatment of HiI and LoI rats during reinstatement of cocaine seeking (relapse) primed by cocaine or caffeine, LoI rats showed a greater reduction in reinstatement than HiI rats (Regier, Zlebnik, Claxton, & Carroll, 2013). Similarly, combined exercise (wheel running) and atomoxetine had greater effects than each treatment alone and a better reduction in reinstatement responding in LoI than HiI rats. These results that indicated better treatment results in the LoS and LoI, less vulnerable groups, are in contrast with the treatment results in male and female rats and monkeys. In several studies, the more vulnerable females showed a better treatment response with both medications such as ketoconazole for heroin (Carroll, Campbell, & Heideman, 2001), or baclofen (Campbell, Morgan, & Carroll, 2002), or bremazocine (Cosgrove & Carroll, 2002) for cocaine self-administration, and behavioral interventions such as access to saccharin for phencyclidine (Cosgrove & Carroll, 2003) or exercise for cocaine (Cosgrove et al., 2002) self-administration.

The recent data reviewed here regarding the LoS rats' greater sensitivity to aversive effects of drugs and the HiS rats' greater sensitivity to the rewarding effects provides information regarding the design of customized treatment strategies for drug abuse. For example, the LoS rats responded more to histamine, which may have functioned as an aversive treatment or a negative factor that when added to the positive cocaine effect neutralized the rewarding effects of cocaine, while in the HiS rats for which cocaine's rewarding effects may have been stronger, histamine treatment was unable to reduce the rewarding effects of cocaine beyond the immediate treatment phase. In terms of translating the present animal studies to customized strategies to humans, a method used for drug addiction in humans that has been relatively successful is the community reinforcement approach (Higgins

et al., 2003), a form of contingency management whereby compliance with drug abstinence is rewarded over time by an accelerating point schedule that can be used for valuable commodities in the community (e.g., school tuition, rent, etc.), while noncompliance is punished by resetting the point contingencies. Based on the animal data, it would be hypothesized that those with lower addiction severity assessments at intake would respond better to more severe punishment (point reset) contingencies, while those with high addiction severity might have more success with a steeper point acceleration to enable nondrug alternatives to interfere with drug seeking. There is substantial evidence that nondrug alternatives interfere with drug seeking (Carroll, Bickel, & Higgins, 2001; Cason & Grigson, 2013); however, little laboratory work has been done to model treatment in differentially vulnerable groups using animals. The initial findings with HiS vs. LoS and HiI vs. LoI rats suggest that a better understanding of how vulnerability interacts with treatment receptivity might lead to optimal customized treatments for drug abusers.

Conclusion

This chapter has discussed accumulating evidence for a strong relationship between feeding behavior and drug seeking. Recent studies using DSM-IV criteria such as difficulty limiting use, increased motivation to use, and continued use despite negative consequences have shown in laboratory models with rats that there is a strong relationship between "food addiction," which may be related to DSM eating disorders, and drug addiction. Palatable food and addictive drugs, such as cocaine, are interchangeable as reward; thus, one form of maladaptive behavior may be substituted for another. In this chapter we focused on a genetic model—rats that are selectively bred to prefer and binge on sweet substances (HiS), and their low saccharin-preferring counterparts (LoS) that do not exhibit these excessive behaviors. The HiS line shows preferences for drugs of abuse and drug bingeing behavior according to the DSM criteria, while LoS rats do not; thus, these lines serve as models for a wide range of human drug users. There are related vulnerability factors such as high and low impulsivity (HiI, LoI) that show preference for and bingeing on cocaine and novelty reactive (HR, LR) selected rats that initiate drug seeking more than low reactive (LR) rats. There is also a high-novelty-preferring (HNP) phenotype that exhibits more impulsive and compulsive drug seeking compared to low-novelty-preferring (LNP) rats. The HiS and LoS rats and the other selected or selectively bred lines have unique characteristics but similar predictions for addictive behavior. There is some overlap in vulnerability factors mentioned here that can result in additive vulnerability to addiction and which allows for a wide range of vulnerability profiles that can determine severity of drug abuse, but the factors focused on in this chapter (e.g., sweet preference, impulsivity) are relatively independent. Yet, the vulnerability characteristics may be additive with each other and with age and sex as well, allowing for a wide range of vulnerability profiles that can determine drug abuse. Also, vulnerability status is a factor to consider in designing treatment strategies, as initial studies indicate that treatment outcome varies considerably in differentially vulnerable

phenotypes, suggesting that custom-designed or multiple treatments might be an important consideration for the high-vulnerability phenotypes. Initial studies with rats suggest that vulnerability status is an important determinant of treatment outcome. Since factors that determine food and drug addiction are interwoven, it is likely that treatments for drug abuse would benefit other disorders of behavioral dysregulation such as obesity.

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The Role of Neurotransmitter Systems in Eating and Substance Use Disorders

3

Guido K.W. Frank

Abstract

Eating disorders (ED) as well as substance use disorders (SUD) commonly start during adolescence and young adulthood, an important time of brain maturation that includes neurotransmitter receptor expression. Increasing attention should be paid to neurobiological mechanisms and brain circuit alterations that may be shared across those potentially related disorders. Studies in ED suggested lower cerebrospinal fluid (CSF), serotonin (5-HT), and dopamine (DA) metabolite levels, neurotransmitters involved in the regulation of eating, mood, and anxiety, among other functions. Higher 5-HT metabolite levels after recovery suggested that this could be a trait alteration. The body of CSF neurotransmitter research in SUD is small. However, alcoholism may be associated with reduced CSF 5-HT metabolites, and acute substance use may increase 5-HT release but also inhibit 5-HT neuronal activity through auto-inhibition, while withdrawal from most substances is associated with reduced extracellular 5-HT. More recent research in ED using brain imaging implicated neurotransmitter receptors such as the 5-HT_{1A} receptor, 5-HT_{2A} receptor, and 5-HT transporter or DA D2/3 receptors, which predicted high anxiety and harm avoidance. Studies in SUD suggested 5-HT and DA receptors may undergo adaptive changes during stages of the illness. Other addictive disorders include tobacco use and gambling behavior, and their neurobiology has been linked to reward and DA pathways. Overall, research suggests that 5-HT and DA are involved in the neurobiology ED and SUD as well as behavioral addictions, and comparative research across disorders should be undertaken to identify underlying mechanisms.

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3.1 Introduction

The eating disorders (ED) anorexia (AN), bulimia nervosa (BN), and binge-eating disorder (BED), as well as the substance use disorders (SUD), commonly start during adolescence or young adulthood (American Psychiatric Association, 2013), a time full of changes in brain biology and maturation (Rumsey & Ernst, 2009). Advances in technology have allowed us to examine aspects of central neurotransmitter systems that could be related to eating and addictive disorder pathophysiology. Older studies have focused on cerebral spinal fluid (CSF) to examine metabolite levels of various neurotransmitters such as serotonin (5-HT), dopamine (DA), norepinephrine (NE), and opioids, as well as other indirect measures of central neurochemical concentration and activity, such as platelet studies and pharmacologic challenge studies. Since then, various brain imaging techniques have been employed to examine brain neurotransmitter receptor availability, especially positron emission tomography (PET), which uses radio tracers that bind to specific neurotransmitters in the brain. In the more recent past, brain imaging techniques such as functional magnetic resonance imaging (fMRI) have used behavioral paradigms that stimulate specific neurotransmitter circuits, an approach that helps tie brain neurotransmitters to disorder-relevant behaviors. One such approach is the study of the brain reward system. Both food and substances of abuse provide powerful stimulation of brain reward circuits (Kelley & Berridge, 2002; Kelley, Schiltz, & Landry, 2005), and hence, there should be significant overlap in the neurobiology of neurotransmitters across those disorders (Kaye et al., 2013). This chapter will review past neurotransmitter research in ED and SUD as well as describe recent developments that may help identify altered brain circuits that may be shared between those disorders.

3.2 Serotonin

3.2.1 Anorexia Nervosa

Several authors have reviewed evidence for 5-HT dysregulation in individuals who were ill with AN and BN (Brewerton, 1995; Jimerson, Lesem, Kaye, Hegg, & Brewerton, 1990; Jimerson et al., 1997; Kaye, Strober, & Jimerson, 2004; Steiger, Gauvin, et al., 2001; Treasure & Campbell, 1994). Brain 5-HT is involved in mood, anxiety, feeding, and sleep regulation among other processes (Naughton, Mulrooney, & Leonard, 2000). Early studies used CSF 5-HT metabolite levels to approximate 5-HT *brain* levels (Stanley, Traskman-Bendz, & Dorovini-Zis, 1985).

Ill restricting-type AN subjects had a significant reduction in CSF of the serotonin metabolite 5-hydroxyindoleacetic acid (5-HIAA) compared to control women (Jimerson, Lesem, Hegg, & Brewerton, 1990; Kaye, Ebert, Gwirtsman, & Weiss, 1984; Kaye, Gwirtsman, George, Jimerson, & Ebert, 1988). Interestingly, a recent study has found increased 5-HT but also DA metabolites in obesity (Markianos, Evangelopoulos, Koutsis, & Sfagos, 2013), indicating that symptomatic AN and OB may be on opposite ends of a neurobiological spectrum. In comparison, recovered restricting-type AN subjects had elevated concentrations of CSF 5-HIAA, proposing the possibility that elevated 5-HT could be a premorbid trait. 5-HT neuronal pathways play a role in the expression of anxiety and fear, obsessional behaviors, and depression (Charney, Woods, Krystal, & Heninger, 1990; Kaye et al., 2008; Price, Charney, Delgado, & Heninger, 1990), and increased 5-HT activity may be related to harm-avoidance traits (Kaye et al., 2008). By depleting tryptophan, the dietary precursor of 5-HT (Fernstrom & Wurtman, 1971), food restriction may produce anxiolytic effects for those predisposed to ED development. In fact, studies have found that depletion of tryptophan (TRP) may reduce dysphoric mood in ill and recovered AN subjects (Kaye et al., 2003), and food reduction may thus produce "self-medicating" effects.

Neurotransmitter receptor imaging studies assess the "functional availability" of 5-HT receptors in the brain. In particular, the 5-HT_{1A} and 5-HT_{2A} receptors are believed to be involved in the modulation of mood, feeding, impulse control, sleep, and anxiety. Using PET and the radioligand [11C]WAY, 5-HT_{1A} receptor binding has been found to be elevated across most brain regions in a mixed group of symptomatic restricting- and binge-eating/purging-type AN subjects compared to healthy controls, as well as in binge-eating/purging-type AN after recovery (Bailer et al., 2005). In contrast, recovered restricting-type AN individuals showed normal brain 5-HT_{1A} binding (Bailer et al., 2005). In addition, there appears to be reduced 5-HT_{2A} binding in the frontal, parietal, and occipital cortices in both ill and recovered AN individuals (Audenaert et al., 2003; Frank et al., 2002). In summary, after recovery, 5-HT_{1A} receptor binding seems to differentiate AN subtypes, whereas 5-HT_{2A} receptor binding is reduced in both restricting and binge-eating/purging AN in various brain regions. Since these disturbances occur after recovery, they may reflect either trait disturbances or scars from the illness.

Some progress has been made to tie such neuroreceptor abnormalities to ED-related behaviors. Harm avoidance, a behavioral correlate of anxiety, has been found to be positively correlated with mesial temporal cortex (amygdala and hippocampus) 5-HT_{2A} binding in recovered binge-eating/purging AN, and with mesial temporal cortex 5-HT_{1A} binding in recovered restricting-type AN. Most recently, the interaction of 5-HT transporter binding with DA receptor availability was associated with harm avoidance in AN after recovery (Bailer et al., 2013), suggesting that interaction of those two neurotransmitter systems contributes to the modulation of complex behaviors such as anxiety.

The 5-HT_{1A} receptor has been implicated in food reward modulation (Carli & Samanin, 2000), while the 5-HT_{2C} receptor may in part transmit "reward signals" (Higgins & Fletcher, 2003). Serotonin brain levels influence weight in rodents,

there is cross talk with reward modulating sites (Konkle & Bielajew, 1999), and 5-HT is implicated in delay of reinforcement and reward processing (Cardinal, Winstanley, Robbins, & Everitt, 2004). Furthermore, 5-HT may be involved in the learning and "appraisal" of rewarding stimuli (Merali, Michaud, McIntosh, Kent, & Anisman, 2003). Thus, the 5-HT system appears to take part in cognitive, emotional, and salience aspects of eating modulation.

Some, but not all, genetic studies support altered 5-HT receptor function in AN (Collier et al., 1997; Enoch, Greenberg, Murphy, & Goldman, 2001; Hinney, Ziegler, Nothen, Remschmidt, & Hebebrand, 1997; Nacmias et al., 1999; Sorbi et al., 1998), and the possibility that genotype could contribute to 5-HT receptor function in AN will need further study.

3.2.2 Bulimia Nervosa

Ill individuals with BN have normal CSF 5-HIAA levels, but the more severely affected present with lower CSF 5-HIAA levels (Jimerson, Lesem, Kaye, & Brewerton, 1992). In contrast, recovered BN individuals showed elevated CSF 5-HIAA (Kaye & Weltzin, 1991). This suggests that abnormally high 5-HT brain levels could be a trait marker for AN as well as BN. Considerable evidence also exists for a dysregulation of 5-HT processes in BN. Examples include blunted prolactin response to the 5-HT receptor agonists m-chlorophenylpiperazine (m-CPP), 5-hydroxytrytophan, and DL-fenfluramine and enhanced migraine-like headache response to m-CPP challenge (Brewerton & George, 1993; Brewerton, Mueller, et al., 1992; Monteleone, Brambilla, Bortolotti, Ferraro, & Maj, 1998). Acute perturbation of 5-HT tone by dietary depletion of tryptophan has also been linked to increased food intake and mood irritability in individuals with BN compared to healthy controls (Bruce et al., 2009), which further suggested that this neurotransmitter system could be involved in the psychopathology of BN.

Brain imaging studies (Kaye et al., 2001) have found reduced orbitofrontal 5-HT_{2A} receptor binding in recovered BN using PET and the radioligand [18F] altanserin. A number of studies have implicated the orbitofrontal cortex in inhibitory processes (Robbins, 2005) and in the representation of food-related affective values (Kringelbach, O'Doherty, Rolls, & Andrews, 2003). Thus, orbitofrontal alterations may contribute to behavioral disturbances associated with BN, such as impulsivity and altered emotional processing (Steiger, Young, et al., 2001), although this has not been explored empirically yet using specific behavior tasks with 5-HT-related probes. BN women failed to show the negative correlations of age and 5-HT_{2A} binding found in normal controls (Kaye et al., 2001), and this lack of correlation may reflect a scarring effect from the illness. Symptomatic BN patients have also shown reduced 5-HT transporter binding in the thalamus and hypothalamus (Tauscher et al., 2001), but increased 5-HT_{1A} receptor binding (Tiihonen et al., 2004), most prominently in the medial prefrontal cortex, posterior cingulate, and angular gyrus of the parietal cortex. After recovery, patients with BN had increased 5-HT_{1A} binding compared to healthy controls as well (Bailer et al.,

2011), and 5-HT_{1A} binding in BN predicted measures for inhibition. The dynamics between 5-HT receptor expression and synaptic 5-HT are not well understood. Reduced 5-HT_{2A} binding in recovered BN subjects may be related to higher level of endogenous 5-HT in the synaptic cleft or a downregulation of the receptor. With the same schema, increased 5-HT_{1A} receptor binding during the symptomatic state may reflect reduced 5-HT synaptic level and an upregulation of the receptor (Enoch et al., 1998; Jimerson et al., 1992). In addition, reduced 5-HT transporter availability in ill individuals with BN may be an adaptation in response to lowered 5-HT concentrations in the premorbid state. Of interest, selective 5-HT reuptake inhibitors (SSRIs) are effective in the treatment of BN, but symptomatic BN requires higher doses of such medications compared to, for instance, patients being treated for depression. This relative resistance to SSRI treatment may be related to an upregulation of 5-HT_{1A} autoreceptors, which inhibit 5-HT release.

3.2.3 Binge-Eating Disorder

A new ED, binge-eating disorder (BED), was just introduced in the last edition of the DSM (American Psychiatric Association, 2013). That disorder is characterized by binge episodes as in bulimia nervosa but lacks compensatory mechanisms, and BED is therefore typically associated with obesity. BED individuals share high depression and anxiety in addition to preoccupation over shape and weight with AN and BN. The use of the 5HT reuptake inhibitors fluoxetine and sertraline showed some promise reducing binge-eating symptoms and body weight in BED (Leombruni et al., 2008), but it is yet unclear whether the use of such medication is in fact targeting binge eating or rather depression and anxiety, with eating behavior changes as secondary effects (Akkermann, Nordquist, Oreland, & Harro, 2010). A small study with ten BED subjects studying prolactin response to the 5HT stimulating challenge drug D-fenfluramine did not find a difference on this measure compared to controls (Monteleone, Brambilla, Bortolotti, & Maj, 2000). The supplement chromium effects mood and eating behavior and has shown some promising pilot results, but larger studies will be needed for confirmation (Brownley, Von Holle, Hamer, La Via, & Bulik, 2013).

3.2.4 Substance Use Disorders

Studying neurotransmitters in addictive disorders is similarly difficult as in ED, as the biological effects here of drug or alcohol interact with existing predisposing biological factors, and teasing apart short-lived state from illness determining trait factors is complex. This has complicated human research in this area, and most of the knowledge stems from animal studies, although they might not reflect "real-life" circumstances. However, it is known that acute administration of substances such as alcohol, stimulants, cocaine, and opioids increases extracellular 5-HT but in return decreases 5-HT neurotransmission via either 5-HT_{1A} receptor auto-inhibition

(in response to alcohol, cocaine, stimulants) or gamma-aminobutyric acid (GABA) action (in response to opioids) in the brain (Kirby, Zeeb, & Winstanley, 2011). Interestingly, 5-HT neuronal response tends to normalize again during chronic use, while withdrawal has been associated with decrease of extracellular 5-HT, and thus possibly contributing to dysphoria and craving during removal of the substance from the organism (Kirby et al., 2011).

The body of literature on neurotransmitter research in humans with SUD problems has been small and is not always easy to interpret with respect to disorder pathology. CSF studies have found a variety of results across alcohol and substance addictions. 5-HIAA levels of alcoholics 1–2 months after their last drink were significantly lower compared to healthy controls or alcoholics within 1–2 days after their last drink (Ballenger, Goodwin, Major, & Brown, 1979). Others found reduced platelet 5-HT content, uptake, and CSF 5-HIAA secondary to chronic alcohol use, which was hypothesized be related to anxiety and depression associated with alcohol use (Tollefson, 1989). However, another study did not find 5-HIAA levels in CSF in alcohol dependence different from controls, and there was also no group difference in DA or NE metabolites (Agartz, Shoaf, Rawlings, Momenan, & Hommer, 2003). More than ten brain imaging studies have assessed the 5-HT transporter in alcohol dependence, but no uniform picture has emerged, that is, studies found increased, normal, or decreased transporter availability (Cosgrove, 2010).

A study in cocaine addiction found no significant relationships between cocaine craving scores and CSF 5-HIAA concentrations, but that study did not report on a comparison group (Roy, Berman, Gonzalez, & Roy, 2002). Ecstasy users however showed lower CSF 5-HIAA compared to controls (Stuerenburg et al., 2002). The number of studies that has investigated CSF neurotransmitters including 5-HT is small, but chronic alcohol or substance use may be associated with lower 5-HT metabolites in CSF compared to controls. How this contributes to illness behavior is uncertain. However, ED and SUD are associated with alterations in 5-HT system activity, and this could contribute to mood and anxiety problems that could prolong the illness or promote relapse. Brain imaging of the 5-HT transporter found increased availability in the brain stem of cocaine-dependent individuals during acute abstinence, which could indicate a compensatory upregulation (Jacobsen et al., 2000). In contrast, in alcoholism, 5-HT transporter availability was lower in some studies (Heinz et al., 1998; Szabo et al., 2004) but normal in another (Brown et al., 2007) compared to controls. Importantly, tobacco smoking may have an important confounding role in those studies by apparently suppressing 5-HT transporter availability in alcoholism (Cosgrove et al., 2009).

3.2.5 Other Addictive Disorders

Gambling disorder is new in DSM-5, and tobacco use has now the same criteria as the other SUD. A multitude of neurotransmitter systems are involved in gambling, including 5-HT (Leeman & Potenza, 2013), but whether for instance increase

(Cuomo et al., 2013) or depletion (Koot et al., 2012) of 5-HT promotes the behavior is uncertain. The highly addictive substance nicotine acts on nicotinic acetylcholine receptors and 5-HT among transmitters such as adenosine, cannabinoids, DA, and glutamate, but the exact mechanisms need further study (Wooters, Bevins, & Bardo, 2009).

3.2.6 Summary

While our understanding of 5-HT in the pathophysiology of those disorders is limited, there may be important overlap between the importance of 5-HT in ED and SUD. As mentioned above, 5-HT has been associated with high anxiety and inhibition in ED. In SUD and addictions, high impulsivity has been associated with disease risk, and 5-HT might have an important role in this trait behavior (Winstanley, Dalley, Theobald, & Robbins, 2004; Winstanley, Olausson, Taylor, & Jentsch, 2010). For instance, individuals vulnerable to addiction may show higher "impulsive choice," a construct that includes the inability to await larger rewards in the future, instead selecting smaller but immediate rewards (Ainslie, 1975; Reynolds, 2006). Clinically, this could be translated into the inability to work toward the benefits of long-term recovery and rather chose the immediate, short-term perceived benefits from substance or alcohol use. Therefore, neurobiologically, AN and addictive disorder individuals could be on opposite ends of a spectrum of low to high impulsivity, possibly mediated at least in part by 5-HT function. The BN population is somewhere in the middle in this framework with aspects of both inhibition and disinhibition, which is reflected phenotypically in binge/purge episodes alternating with food restriction (Tozzi et al., 2005). AN, BN, and SUD populations have heightened sensitivity to salient stimuli (Brunelle et al., 2004; Jappe et al., 2011; Lyvers, Duff, Basch, & Edwards, 2012; Wagner et al., 2006). How those potential traits interact with high anxiety and inhibition in ED (Fig. 3.1) but frequently low inhibition and high impulsivity in SUD will require further careful and comparative study.

3.3 Dopamine

The DA pathways are a neuromodulatory system that arises from cells in the midbrain (Kapur & Remington, 1996). These midbrain neurons release DA, which acts on DA receptors. DA function contributes to the modulation of motor activity (Alexander, Crutcher, & DeLong, 1990), weight and feeding behaviors (Halford, Cooper, & Dovey, 2004), and reinforcement and reward (Volkow, Fowler, & Wang, 2002). There is some indication that AN responds to typical and atypical neuroleptics (Brewerton, 2004; Brewerton, 2012; Cassano et al., 2003), which may indicate alterations in the DA system in that disorder.

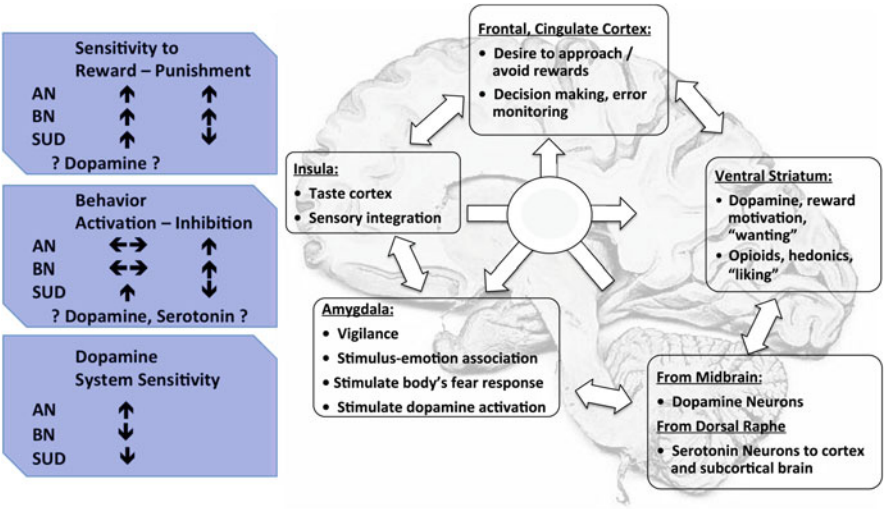

Fig. 3.1 To the left are behavioral and biological aspects that could drive eating and substance use disorder behaviors. To the right is a schematic of brain structures involved in reward processing. *AN* anorexia nervosa, *BN* bulimia nervosa, *SUD* substance use disorder

3.3.1 Anorexia Nervosa

In ill AN, CSF homovanillic acid (HVA), the major DA metabolite, was reduced by about 30 % compared to controls (Kaye, Ebert, Raleigh, & Lake, 1984). In addition, recovered restricting-type AN subjects had significantly reduced concentrations of CSF HVA compared to controls and other ED subjects (Kaye, Frank, & McConaha, 1999). Whether individuals with restricting-type AN have an intrinsic disturbance of DA (James & Starr, 1980; Kapur & Remington, 1996; Ugedo, Grenhoff, & Svensson, 1989) remains uncertain. A mixed group of recovered restricting-type and recovered binge-eating/purging-type AN women had increased DA D2/D3 receptor binding potential (BP) in the anteroventral striatum (Frank et al., 2005), while decreased D2/D3 receptor binding has been found in obese subjects (Wang, Volkow, Thanos, & Fowler, 2004). That finding supports the possibility that D2/D3 receptor binding may be inversely related to weight and eating, with restricting-type AN on one end and obesity on the other end of the spectrum. Thus, increased D2/D3 receptor availability in AN may contribute to a drive to become emaciated. Increased DA D2/D3 receptor binding in AN might help explain the underlying mechanism for why individuals with AN are able to lose weight, resist eating, and overexercise, are protected from substance abuse, and are insensitive to natural rewards. It is worth noting that food restriction sensitizes D2/D3 receptors in rats, and if the same mechanism happens in AN then this could be a factor complicating recovery (Carr, Tsimberg, Berman, & Yamamoto, 2003). The mentioned PET imaging studies tested available DA receptor profiles, but they could not test the

functionality of those receptors in relation to actual behavior. Others found increased eye blink compared to controls (Barbato, Fichele, Senatore, Casiello, & Muscettola, 2006) that suggested heightened DA sensitivity (Karson, 1983) in AN, which could provide important clues that a downregulation of receptor sensitivity might be important, despite the notion of low extracellular DA in AN.

3.3.2 Bulimia Nervosa

The early research indicated that CSF HVA is normal in individuals with BN when they are ill (Kaye et al., 1990). A very recent study found that BN individuals had a trend to lower DA D2/3 receptor binding in the striatum compared to controls, and BN individuals showed less DA release compared to controls in response to methylphenidate application (Broft et al., 2012). While this body of research is small, it suggests that food restriction in AN may increase, and episodic binge eating in BN could reduce DA pathway activity.

3.3.3 Binge-Eating Disorder

Having the addiction model in mind as in BN, research in BED has also focused on brain DA function. A fairly solid model exists that describes DA in the mechanism of binge eating (Avena, Bocarsly, & Hoebel, 2012) suggesting an addictive quality **(see Chaps. 1 and 13)**. Further, human studies have found higher DA release in relation to binge eating (Wang et al., 2011), and the DA D2 receptor may be involved in this disorder (Davis et al., 2012). A recent study in rodents supports that notion (Halpern et al., 2013). Another recent study using the DA reuptake inhibitor bupropion in BED led to short-term lower body weight, but did not improve behaviors such as binge eating or food craving (White & Grilo, 2013). All in all, the BED population is neurobiologically very heterogeneous, and no clear recommendations can be given for pharmacologic intervention (Marazziti, Corsi, Baroni, Consoli, & Catena-Dell'Osso, 2012).

3.3.4 Substance Use Disorders

DA has been extensively studied in animal models of addictive disorders. This stems from the understanding that this neurotransmitter is intimately involved in the processing of and response to salient, "rewarding or punishing" stimuli (Kelley & Berridge, 2002; Ross & Peselow, 2009). Studying brain circuits that are related to DA models in the context of brain reward function is particularly interesting for several reasons. First, within brain reward circuits, DA is critically associated with providing signals regarding the presence and amplitude of rewards (Kelley, Baldo, Pratt, & Will, 2005; Schultz, 2002). Such signals facilitate reinforcement learning (Daw & Doya, 2006) and have been found to code the value of a stimulus (Daw,

Gershman, Seymour, Dayan, & Dolan, 2011; Jocham, Klein, & Ullsperger, 2011), which may even include the metabolic value of food (de Araujo, Ren, & Ferreira, 2010). Second, computer models for DA neuron reward activation exist that can be related to human in vivo brain function (Sutton & Barto, 1998), helping in the study of in vivo dynamics of human DA function.

Alcoholism was not associated with alterations in HVA levels compared to controls in an early study (Ballenger et al., 1979), in a small follow-up study (Sjoquist & Borg, 1984), or in a larger more recent investigation (Agartz et al., 2003). One study though found higher HVA levels in early onset alcoholics compared to controls or late onset alcoholic subjects, but HVA levels were unrelated to craving measures (Petrakis et al., 1999). In contrast, cocaine-dependent individuals showed significantly higher CSF HVA than did the healthy controls, as did short-term abstinent cocaine-dependent individuals (Roy, Berman, Gonzalez, et al., 2002; Roy, Berman, Williams, Kuhn, & Gonzalez, 2002). Thus, in alcoholism DA metabolites do not seem to reflect the addiction pathophysiology, although the time of onset of the alcohol disorder could be related to HVA levels. There may be more to be learned from CSF HVA in cocaine addiction, but the research body is small and cannot be interpreted without using great caution.

DA transporter availability was in most studies increased in cocaine addiction compared to controls (Crits-Christoph et al., 2008; Jacobsen et al., 2000; Malison et al., 1998). It was hypothesized that this would be a compensatory upregulation, and this was supported by the gradual decline in receptor levels during abstinence. The DA D2/3 receptor was found to be lower compared to controls in most cocaine dependence studies, possibly a sign of downregulation in response to overstimulation (Martinez et al., 2004; Volkow et al., 1993; Volkow et al., 1990). In contrast, lower DA D2/3 receptor availability seems to be a more consistent marker for alcohol dependence (Cosgrove, 2010). Importantly, lower DA D2/3 receptor availability may be related to greater alcohol craving (Heinz et al., 2004) and higher consumption (Martinez et al., 2005), as well as predict treatment outcome or relapse (Guardia et al., 2000).

3.3.5 Other Addictive Disorders

As stated above, multiple neurotransmitter systems are involved in addictions including tobacco and gambling. This is complicating treatment development, and effective treatments were frequently found by chance, while systematically studied compounds that promised success in animal models have failed (Pierce, O'Brien, Kenny, & Vanderschuren, 2012). Gambling disorder is new to the field of addictions and as there are only moderately well-formed concepts of neurotransmitter involvement. Similarly to tobacco use, careful study will be required to identify key neurotransmitters that can become targets for pharmacologic intervention. DA appears to be the obvious first neurotransmitter, but as above in SUD, its involvement is intertwined with many other systems in the brain.

3.3.6 Summary

DA function is a particularly interesting neurotransmitter system to study across ED, SUD, and other addictive disorders. There is a clear overlap between natural rewards such as food and substances of abuse and addiction with respect to activation of the reward pathways (Kelley & Berridge, 2002). Prospective studies in rodents that were exposed to over- or underconsumption of food (Avena, Rada, & Hoebel, 2008; Carr, 2007; Johnson & Kenny, 2010) suggest adaptive DA-related changes to food intake with sensitization to food restriction but desensitization to excessive food ingestion quality (see Chaps. 1 and 13). Similar models have been proposed for addictive disorders, where the organism eventually desensitizes so much that exposure to alcohol or drugs only improves the mood state toward "normal" without providing the "high" when first using (Koob & Le Moal, 2005). For the addiction field a so-called reward deficiency model has been established, proposing that such a reduced reward response could drive excessive use (Blum, Gardner, Oscar-Berman, & Gold, 2012; Comings & Blum, 2000). This model could fit the proposed psychopathology in BN and BED with a downregulation of response to food stimuli and excessive sensitivity in AN after deprivation. Importantly, such adaptations could be targeted when in search for neurobiological treatments for those disorders.

3.4 Norepinephrine

NE is part of the body's stress system, which is also closely linked to the corticoid system and corticotropin-releasing factor (CRF). Foods, as well as substances of abuse, are regularly used by individuals in response to stress, while in AN food *restriction* may be used in an attempt to handle stress better. Animal research has shown that during withdrawal of substances of abuse anxiety increases, the responsiveness to rewards is lowered, DA system function is reduced, and this is accompanied by increases of CRF in the amygdala (Koob, 2010, 2013). Thus this biologic system around stress may be highly important as a trigger for behaviors that involve eating or alcohol and drug use. Other behaviors and central functions that norepinephrine is involved are learning and memory, sleep–wake cycle, reinforcement, and general body metabolism, functions that have been associated with eating behavior (Cooper, Bloom, & Roth, 2003).

3.4.1 Eating Disorders

The CSF levels of NE in underweight AN individuals and in these patients a few weeks after weight restoration were similar to those in normal subjects, while long-term (20 ± 7 months) weight-recovered AN individuals had a 50 % decrease in CSF NE levels compared with those of controls (Kaye, Ebert, Raleigh, et al., 1984), which could point toward trait alterations. A study in obese individuals did not find

alterations in NE metabolites (Markianos et al., 2013), while another study found that obese individuals compared to controls had reduced NE metabolite methoxyhydroxyphenylglycol (MHPG) levels in addition to reduced corticotropin-releasing hormone, beta-endorphins, and neuropeptide Y (Strombom et al., 1996).

Bulimic patients had a significantly lower mean CSF NE concentration, while CSF 5-HIAA and HVA were normal compared to controls (Kaye et al., 1990).All in all, there may be alterations in ED with respect to the body's stress system, but the data are too few to draw meaningful conclusions at this point.

3.4.2 Substance Use Disorders

The available basic research in SUD and NE is larger, and theoretical models exist on how this system could be involved. Most prominently, stress may be involved in a reduced ability to experience rewards, causing a "reward deficit" which may promote use (Koob, 2013). Clinically, alcohol-dependent individuals without overt autonomic nervous system signs showed no change in MHPG in an earlier (Fujimoto, Nagao, Ebara, Sato, & Otsuki, 1983) and a more recent study (Agartz et al., 2003), but there was a positive correlation between CSF MHPG and intensity of withdrawal symptoms in one study (Fujimoto et al., 1983). Yet another study that explored family background in alcohol-dependent individuals showed that CSF MHPG correlated negatively with subjective reported ethanol consumption as well as presence of first-degree relatives with alcohol problems and presence of memory lapses (Valverius, Hogstrom-Brandt, & Borg, 1993).

3.4.3 Summary

Most research on the relationship between stress-related neurotransmitters and hormones in the control of ED and SUD or addiction has been done in animals. However, this neurotransmitter system may provide a very promising avenue of research in targeting cognitive-emotional factors that contribute to those disorders.

3.5 Opioids

While DA has been mostly associated with the drive to approach rewards or so-called wanting, the opioids may be particularly involved in the hedonic ("liking") aspects of food and other rewarding stimuli (Berridge, Robinson, & Aldridge, 2009; Kelley & Berridge, 2002). One could then speculate that individuals with trait-related attenuation in this system might be prone to excessive need for hedonic stimulation, or if that system were overly expressed, it could be highly able to resist both food and alcohol or drug stimuli.

3.5.1 Eating Disorders

A small sample of subjects ill with AN showed significantly lower CSF concentrations of the opioid beta-endorphin, as well as beta-lipotropin and adrenocorticotropic hormone (ACTH), all derived from the same precursor molecule, proopiomelanocortin (POMC) (Kaye et al., 1987). However, those alterations remitted with recovery (Kaye, 1987). Another investigation found higher levels of CSF opioids in severely underweight patients with AN compared to controls and the same patients after weight restoration, while chronic AN with mild underweight had normal levels of CSF opioids (Kaye, Pickar, Naber, & Ebert, 1982). In light of those studies, it could be that food restriction sensitizes opioid activity in AN. This could be consistent with research in BN that found less mu-opioid receptor binding in the left insular cortex compared to controls while binding correlated inversely with recent fasting behavior in that study, suggesting that the episodic excessive food intake downregulates this receptor (Bencherif et al., 2005; Bencherif et al., 2004; Brewerton, Lydiard, Laraia, Shook, & Ballenger, 1992; Stoeckel et al., 2008).

3.5.2 Substance Use Disorders

Mu-opioid receptor binding has been found to be increased in active cocaine users, as well as during immediate abstinence, but then decline, with the steeper the decline indicating a longer time to relapse (Gorelick et al., 2005; Zubieta et al., 1996). This receptor may have a critical role in craving and relapse in cocaine users. In alcohol dependence, mu-opioid receptor availability may be increased or decreased (Bencherif et al., 2004; Heinz et al., 2005), and more research is needed to study the relevance of this neurotransmitter system in alcoholism.

3.5.3 Summary

There is a strong theoretical framework describing how the opioid system could be involved in ED and SUD. There is a large body of research in animals but a small amount of human work with respect to brain imaging. In contrast, a variety of studies have used opioid receptor active agents as potential treatments, although models of action for this receptor are still under development (Spetea, Asim, Wolber, & Schmidhammer, 2013). Interestingly, more commonly μ-opioid receptor agonists are used in the treatment of drug addiction, while antagonists have provided some but only modest success (Tetrault & Fiellin, 2012). Most recently, an opioid receptor antagonist showed some promise in altering brain response to images of food in binge-eating individuals (Cambridge et al., 2013), suggesting that this receptor could become an important treatment target, at least in eating-related disorders.

3.6 Brain DA and Reward Function to Advance Neurotransmitter Research in Eating and Substance Use Disorders

The use of CSF samples and radioligand brain imaging to study neurotransmitter systems has given us directions for further research; however, how those systems are related to actual behavior continues to be largely obscure. The combination of functional imaging that applies disorder-relevant tasks that have been associated with specific neurotransmitter function has been a current approach for human clinical in vivo research to answer such questions. Studying brain circuits that are related to DA models in the context of brain reward function could be particularly important here, as DA is critically associated with providing signals regarding the presence and amplitude of rewards (Kelley, Baldo, et al., 2005; Schultz, 2002), facilitates reinforcement learning (Daw & Doya, 2006), and has been found to code the value of natural and drug reward stimuli (Daw et al., 2011; de Araujo et al., 2010; Jocham et al., 2011; Kelley, Schiltz, et al., 2005). Second, computer models for DA neuron reward activation exist that can be related to human in vivo brain function. Predictions of brain neurotransmitter response can be compared with actual brain activation and compared across pathologic or control groups. Such a model is the temporal difference model (Sutton & Barto, 1981). This model is a theoretical framework for computational reward learning that predicts brain DA neuron response. This model has been previously tested for unexpected reward receipt and omission in animal studies (Schultz, Dayan, & Montague, 1997) and later validated for human brain imaging (D'Ardenne, McClure, Nystrom, & Cohen, 2008; O'Doherty, Dayan, Friston, Critchley, & Dolan, 2003). In brief, DA neurons exhibit a phasic burst of activation in response to the presentation of an unexpected rewarding stimulus (the primary, unconditioned reward stimulus US). After repeated presentation of an additional arbitrary stimulus (the conditioned stimulus CS) preceding the US, the phasic activation of DA neurons transfers in time to the presentation of the CS. Thus, the CS elicits a conditioned DA response. This conditioned response is thought to reflect a *prediction* regarding *upcoming* rewards, so that after presentation of the CS, there is a high likelihood of a reward appearing. As it is thought to be a prediction, such a prediction can be violated. If the CS (and therefore the conditioned DA response) is not followed by the expected reward (US), then there is a violation of the prediction, and as a consequence at the time of expected but omitted reward, there is a decrease in DA tone. This relationship between CS and US is termed a "prediction error," the difference between the value of the reward stimulus received and that predicted.

Most recently 21 underweight, restricting-type AN (age M 22.5, SD 5.8 years); 19 obese (age M 27.1, SD 6.7 years); and 23 healthy control women (age M 24.8, SD 5.6 years) were studied using functional magnetic resonance brain imaging (fMRI) together with a reward-conditioning task that elicits the prediction error response (Frank et al., 2012). The DA model reward-learning signal distinguished groups in the anteroventral striatum, insula, and prefrontal cortex, with brain responses greater in the AN group, but lower activation in the obese group,

compared to controls. These results suggested that brain reward circuits are more responsive to unexpected food stimuli in AN but less responsive in obese women. A study using the same task in BN found reduced presumably DA-related response in insula, ventral putamen, amygdala, and orbitofrontal cortex, and binge/purge frequency in BN inversely predicted reduced TD model response (Frank, Reynolds, Shott, & O'Reilly, 2011). Those results suggest that there may be adaptive changes in response to under- or overeating in ED as seen in the animal studies described above (Avena, 2013). The stronger response in AN but lesser activation in BN and obese also indicates that those groups have differences in strength of learning signals derived from DA-related activation provided to the prefrontal cortex. A few studies have investigated prediction error response in addictive disorders. For instance, methamphetamine has been shown to enhance the brain response during prediction error stimulation (Menon et al., 2007). Most recently, research in alcohol dependence has now shown that those individuals have a functioning prediction error response but that the prefrontal cortex does not adequately learn from those signals (Park et al., 2010). Thus, this model provides the opportunity to study a variety of potential brain circuitry alterations including subcortical stimulus-reward associations and higher-order learning-related pathways. As the prediction error response has been associated with specific DA receptors, namely, the D1 and D2 type, there could be important avenues for future drug interventions (Maia & Frank, 2011).

Conclusion

Research over the past 30 or years has provided a variety of important findings with respect to 5-HT, DA, NE, and opioid neurotransmitter system alterations across ED and SUD populations. There are no studies that investigated how those disorders overlap from a neurobiological level. The neurotransmitter system that may have the biggest promise to reveal similarities across disorders may be the DA system, as there is strong neuroscience-based knowledge, especially regarding its involvement in reward processing. However, 5-HT function may also be a strong candidate as it is involved in impulsivity and inhibition, behaviors relevant to both ED and SUD, and research into the NE system could provide important markers that relate to cognitive-emotional triggers for pathologic eating or substance use in the context of stress. More comparative research will be needed to disentangle interactions between these disorders and neurotransmitter systems.

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Neuroimaging of Eating Disorders, Substance Use Disorders, and Addictions: Overlapping and Unique Systems

4

Ashley N. Gearhardt, Rebecca G. Boswell, and Marc N. Potenza

Abstract

This book chapter examines neuroimaging studies of eating and addictive disorders in order to explore shared and distinct domains of functioning. We adopt a dimensional approach, focusing specifically on domains of impairment across eating and addictive disorders: executive control, reward and motivation, emotional reactivity, memory/learning, and interoceptive awareness. Although the literature is limited in some domains, binge-eating disorder and bulimia nervosa appear to share more neural commonalities with addictive disorders (e.g., diminished executive control, heightened reward sensitivity) relative to anorexia nervosa. The similarities and differences in eating and addictive disorders may have implications for the conceptualization of etiology and the development of treatments for eating disorders. Finally, we outline areas of future research needed to further clarify the relationship between addictive and eating concerns.

Keywords

Eating disorder • Addiction • Binge-eating disorder • Anorexia nervosa • Bulimia nervosa • Dimensional • RDoC • Neuroimaging • fMRI

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Table 4.1 Similarities and dissimilarities between addiction and eating disorder neuroimaging literature

Similar	Dissimilar
Executive control	
BED, BN, and addiction	AN
Motivation/reward	
Less clear, although BED/BN appears to be somewhat more related to addiction than AN	
Learning and memory	
BED, BN, AN, and addiction	
Emotion regulation[a]	
BN and addiction	AN
Interoceptive awareness[a]	
BN and addiction	AN

BED binge eating disorder, *BN* bulimia nervosa, *AN* anorexia nervosa
[a]No studies on emotion regulation and interoceptive awareness in BED could be identified

Addictions and some eating disorders (ED) share multiple characteristics (e.g., diminished control, elevated negative affect), but there are also substantial differences in proposed etiologies between the two classes of disorders. Further, there is significant variability within both ED (e.g., anorexia nervosa [AN] vs. binge-eating disorder [BED]) and addictive disorders (e.g., pathological gambling vs. opiate dependence), which increases the complexity of comparison. In the following book chapter, we will focus on the neuroimaging literature as a tool to potentially identify areas of overlap and distinction between eating and addictive disorders by examining neuroimaging studies that focus on functioning theoretically related to both groups of disorders: executive control, reward and motivation, emotional reactivity, memory/learning, and interoceptive awareness (see Table 4.1). Next, we will outline components that may be more unique to either disorder. Finally, we will outline areas of future research needed to further clarify the relationship between addictive and eating concerns in the hope that a greater understanding of the relationships between these disorders may lead to increased knowledge of etiologies and the development of novel, efficacious, and well-tolerated treatments.

4.1 Defining Eating and Addictive Disorders

The fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) has undergone significant revisions based on scientific inquiry into the etiology and treatment of psychological disorders, including new definitions of addictive and eating disorders (American Psychiatric Association, 2013). Importantly, DSM-5 includes a reorganization of addiction criteria, the classification of gambling and substance use disorders (SUD) together, and the inclusion of BED as a formal diagnostic entity. The reclassification of pathological gambling as a

"behavioral addiction" reflects extensive work that uncovered similarities between pathological gambling and SUD (Leeman & Potenza, 2012; Potenza, 2006). Studies also suggest commonalities between addictions (both substance and "behavioral") and certain ED, particularly BED (Gearhardt, White, & Potenza, 2011). Despite growing evidence of the potential clinical and social implications of a "food addiction" diagnostic category, there have been no food-related additions to the addiction classification of DSM-5 (Gearhardt et al., 2012), and the topic of food addiction remains debated (Avena, Gearhardt, Gold, Wang, & Potenza, 2012; Ziauddeen, Farooqi, & Fletcher, 2012).

Importantly, the inclusion of BED as a formal ED diagnosis allows for an extended investigation of its psychopathology and comparison to other ED. A transdiagnostic model of ED suggests that a core psychopathology is overevaluation of weight and shape concerns, induced by dieting and maintained by mood dysregulation (Fairburn, Cooper, Shafran, & Wilson, 2008). Both bulimia nervosa (BN) and BED are characterized by recurrent binge eating (eating a larger amount of food than intended during a discrete period of time) and a concurrent experience of a lack of control over eating. BN is differentiated from BED through recurrent inappropriate compensatory behavior in order to prevent weight gain and overevaluation of shape and weight concerns. In contrast, anorexia nervosa (AN) is defined by the restriction of food intake leading to a body weight less than normal, intense fear of (or behavior to avoid) gaining weight or becoming fat, and disturbance in the perception or evaluation of body weight or shape.

Elevated rates of comorbidity may suggest a core etiology involved in the development of eating and addictive disorders. BN appears to have a higher comorbidity with SUD than AN, which may be related to heightened impulsivity (Holderness, Brooks-Gunn, & Warren, 1994). Binging groups (BN and BED) are more likely to be high in impulsivity (demonstrating both high reward sensitivity and low inhibition) than AN groups, which are more likely to be high in obsessionality, perfectionism, and rigidity (Vitousek & Manke, 1994). Reward sensitivity has also been implicated in comorbid binge eating and substance abuse in women (Dawe & Loxton, 2004). These findings suggest that considering binge eating as an addictive process may be a fruitful way to understand failed regulation of reward processing across disorders.

Recently, the National Institute of Mental Health (NIMH) launched the Research Domain Criteria project (RDoC) to consider new ways of classifying psychopathology based on dimensions of observable behavior and neurobiological measures (Phillips, 2011). The intent is to translate rapid progress in research into an improved integrative understanding of psychopathology and the development of better treatments for mental disorders. The RDoC approach includes dimensional axes based on potential domains of impairment, including positive valence, negative valence, cognitive systems, social processes, and regulatory processes. In the case of addictive disorders and eating pathology, there are striking similarities between BN/BED/SUD in failed regulation of reward-related processes that may not appear in other ED (e.g., AN). This dimensional approach suggests that there

may be similarities between binge eating and substance use that may extend traditional categorical models of psychopathology.

4.2 Executive Control

4.2.1 Eating Disorders

Neuroimaging studies of executive control (which is conceptualized as top-down processes implicated in self-control, goal-direction, and inhibition) in disordered eating have identified differences among AN and BN/BED participants. AN participants, relative to healthy control subjects, exhibit increased activation in regions implicated in executive control (e. g., the prefrontal cortex (PFC) and anterior cingulate cortex (ACC)) when exposed to food stimuli (Uher et al., 2003). During tasks of inhibitory control, participants with AN (restricting type) showed increased activation in the medial PFC (Oberndorfer, Kaye, Simmons, Strigo, & Matthews, 2011) and the posterior visual and inferior parietal cortical regions (Lock, Garrett, Beenhakker, & Reiss, 2011), which was interpreted as reflecting elevated self-control. Zastrow et al. (2009) found that AN patients (compared to healthy controls) were less able to switch behavioral response styles when changes were needed, and AN participants exhibited hypoactivation in the ventral anterior cingulate-striato-thalamic loop associated with motivation. In contrast, AN participants had hyperactivation in neural regions associated with executive control (e.g., frontal cortex) during task performance. Thus, individuals with AN might not only have greater cognitive control, but may use this executive control rigidly.

Unlike the elevated activation of control-related regions observed in individuals with AN, different activation patterns have been observed in BN and BED participants. BN patients displayed less activation in the PFC (Joos et al., 2011; Uher et al., 2004), ACC, temporal lobe (Joos et al., 2011), and lateral orbitofrontal cortex (OFC) relative to healthy control subjects when viewing food pictures (Uher et al., 2004). Patients with BN also exhibited hypoactivation in the PFC, inferior frontal gyrus (IFG), lenticular and caudate nuclei, and ACC during an executive-control task (Marsh et al., 2009, 2011). Lock et al. (2011) found that participants with binge-purge behavior (including both AN and BN diagnoses) had greater activation in the dorsolateral PFC compared to healthy controls despite performing similarly on an executive-control task. These findings were interpreted as possibly reflecting less efficient recruitment of executive-control regions in the binge-purge group. Similar to BN participants, obese women with BED exhibited hypoactivation in the IFG, PFC, and insula relative to obese women without BED and healthy control subjects during a cognitive-control task (Balodis, Molina, et al., 2013). Further, higher levels of attempted dietary restraint in BED were related to reduced activation in the IFG and insula (Balodis, Molina, et al., 2013). Participants with BED also exhibited hypoactivation in the PFC and insula during the processing of reward and loss (Balodis, Kober, et al., 2013). Finally, BED

participants have demonstrated less activation in the lateral OFC relative to normal-weight control subjects when passively viewing food pictures (Weygandt, Schaefer, Schienle, & Haynes, 2012). These findings raise the possibility that the diminished control over food consumption associated with BN/BED may relate to neural differences in regions implicated in executive control and reward processing.

4.2.2 Addictive Disorders

Deficits in executive control associated with BN and BED are also implicated in addictive disorders. Structural abnormalities in drug users are related to self-control deficits. For example, decreased activation in prefrontal regions is related to reduced availability of striatal dopamine D2/D3 receptors in participants with addiction and decreases in baseline glucose metabolism in the OFC, ACC, and DLPFC (Volkow, Fowler, Wang, Swanson, & Telang, 2007; Volkow et al., 1997). Obesity is also related to reduced dopamine D2/D3 receptor availability (Wang et al., 2001) and genetic alleles that may be associated with reduced dopamine signaling (Stice, Spoor, Bohon, & Small, 2008). Further, animal models suggest that overconsumption of highly palatable foods may also reduce striatal D2 receptor levels (Johnson & Kenny, 2010), which may contribute to executive-control deficits associated with BN/BED. Yet, it is still unclear whether reduced D2/D3 receptor availability and hypofunctioning in control-related neural regions precede the development of addictive disorders/obesity or result from excessive substance/food consumption (or both).

Frontal cortical impairments in the OFC, ACC, and DLPFC have been described as a core feature of impulsivity and compulsivity in addiction (Fineberg et al., 2009; Goldstein & Volkow, 2011; Potenza, Sofuoglu, Carroll, & Rounsaville, 2011). Drug/alcohol users also have poorer performance on behavioral tasks reflecting impulse control, such as delay discounting and Go/No-Go (Bickel et al., 2007; Fu et al., 2008), which may relate to activation differences in the ACC and PFC observed in addicted individuals (Brewer, Worhunsky, Carroll, Rounsaville, & Potenza, 2008; Goldstein & Volkow, 2011; Leland, Arce, Miller, & Paulus, 2008). Thus, addiction-related executive-control findings may more closely relate to the undercontrol associated with BED/BN than the overcontrol associated with AN (see Table 4.1).

4.3 Motivation and Reward

4.3.1 Eating Disorders

Disordered eating is associated with motivation to seek out or avoid food and differences in hedonic reward in response to food consumption. As a central hallmark of AN is the avoidance of food consumption, one might predict that AN would be related to reduced activation in neural regions associated with motivation

in response to food cues, yet the neuroimaging literature is not consistent. AN participants exhibit less activation in the occipital cortex to food pictures (Santel, Baving, Krauel, Münte, & Rotte, 2006; Uher et al., 2003, 2004), which may be related to a diminished salience of food in this disorder. Further, Holsen et al. (2012) found that participants with AN (active and recovered) relative to control subjects had hypoactivation in the in hypothalamus, amygdala, and anterior insula in response to high-calorie food images prior to eating, which may reflect diminished motivation for food. However, others have found that AN and BN patients exhibit elevated activation in the medial OFC and ACC in response to food pictures, which could be interpreted as either elevated craving or a general increase in emotional response to food (Uher et al., 2004). Functional connectivity analyses may shed some light on how activation in the same region may relate to different responses. Kim, Ku, Lee, Lee, and Jung (2012) found AN and BN participants had greater activation in the left anterior insula (part of the primary gustatory cortex) in response to food relative to nonfood pictures, but the connectivity between these disorders differed. Specifically, insula activation in AN participants was functionally connected to regions implicated in control (e.g., IFG), whereas in BN it was associated with a reward region (e.g., medial OFC) (Kim et al., 2012). Thus, gustatory cues may trigger reward responses in participants with BN, but signal control responses in patients with AN. Finally, Cowdrey, Park, Harmer, and McCabe (2011) found that individuals recovered from AN relative to control subjects had not only increased activation in the ventral striatum and occipital cortex to pleasant food stimuli but also greater activation in the insula, putamen, ACC, and caudate to aversive food stimuli. This pattern of results may reflect greater incentive salience of food (whether positive or negative in valence) for individuals with AN (Cowdrey et al., 2011).

Additional neuroimaging studies of BN have suggested elevated motivation and reward response to foods. Brooks et al. (2011) found that BN relative to AN participants had increased activation in reward and somatosensory regions (e.g., caudate, insula) in response to food images. Relative to control subjects, participants with BN had greater activation in the ACC (Schienle, Schäfer, Hermann, & Vaitl, 2009; Weygandt et al., 2012), insula, and ventral striatum (Weygandt et al., 2012) to food cues. Further, greater negative affect for BN participants is associated with elevated activation in the putamen, caudate, and pallidum while anticipating palatable food consumption (Bohon & Stice, 2011). Thus, negative emotional states may increase the motivational properties of foods for participants with BN. Both BN and BED patients have been found to have increased gray matter in the medial OFC (Schäfer, Vaitl, & Schienle, 2010) and increased activation in this region in response to food cues (Schienle et al., 2009), but there have also been differences identified between these two disorders. BN participants appear to have greater activation in the ACC, insula, and ventral striatum in response to food cues than do BED patients (Weygandt et al., 2012). Compared to control subjects, BED participants had elevated responses in the medial OFC (Weygandt et al., 2012) and increased dopamine release in the caudate and putamen when exposed to food stimuli (Wang et al., 2011), which might reflect

increased motivation. Yet, BED relative to control participants has shown less activation in certain areas in response to food cues (e.g., ACC, ventral striatum) (Weygandt et al., 2012). Although the meaning of these results is not entirely clear, animal models of food consumption suggest that excess palatable food consumption may diminish functioning in reward-related neurocircuitry (Johnson & Kenny, 2010). Similarly, BED participants have been found to have less gray matter volume in the lateral OFC, medial OFC, and striatum (Schäfer et al., 2010). Thus, BED may be related to hypoactivation in certain reward regions due to overconsumption of food.

There are also differences among the ED during food receipt. AN is associated with a tendency to exhibit less activation in the ventral/dorsal striatum, insula, and medial OFC to taste (Vocks, Herpertz, Rosenberger, Senf, & Gizewski, 2011; Wagner et al., 2007), and (unlike in healthy control subjects) insula activation does not seem to differ based on the pleasantness rating. Thus, AN participants may be less sensitive to the hedonic nature of food. Although BN has been related to less activation in the ACC and cuneus in response to glucose (Frank et al., 2006), BN (relative to control and AN participants) has been associated with greater anterior ventral striatum activation to cream mixture. Thus, the association of BN with food-related reward activation is less clear. To our knowledge, no studies to date have examined the neural response to food receipt in BED participants.

4.3.2 Addictive Disorders

Motivation and reward are also key constructs in addictive behaviors. Motivation in addiction is related to dopaminergic response, especially in the NAc, ACC, OFC, DLPFC, amygdala, striatum, and ventral pallidum (Salamone, Correa, Farrar, & Mingote, 2007). Dopaminergic dysregulation is associated with drug-seeking behavior (Volkow & Li, 2005), and cues become powerful triggers of dopaminergic release and motivation in addicted individuals (Robinson & Berridge, 2001). Addicted participants exhibit increased craving and activity in many brain regions (e.g., ventral striatum, ACC, amygdala) during exposure to drug cues in fMRI studies (Chase, Eickhoff, Laird, & Hogarth, 2011; Shiffman et al., 2013). In contrast to the hyperactivation associated with drug cues, substance-dependent individuals versus healthy control subjects typically exhibit blunted dopaminergic release during actual drug consumption and report weaker hedonic responses (Martinez et al., 2007; Volkow, Wang et al., 2007; Volkow et al., 1997). It is still unclear whether the reduced neural response to consumption in participants with addiction is a preexisting risk factor related to a reward deficiency (Comings & Blum, 2000), a result of excessive substance use (Kalivas & O'Brien, 2007), or related to other factors. Yet, increased motivation in response to cues and a reduced response in reward-related regions during consumption appear to be important components of addictive behaviors. In comparison to disordered eating, the increased response in motivation regions to food cues in BED and BN are similar to the addiction literature, but the literature is not always consistent. The relative

dearth of studies examining food receipt in ED may contribute to this inconsistency. Further examination of food reward is an important area of future study.

4.4 Memory and Learning

4.4.1 Eating Disorders

Differences in learning and memory may also contribute to ED. Participants with active AN (who are markedly underweight) appear to have deficits in cognitive processing, which may be related to malnourishment (Delvenne et al., 1997). Neuroimaging studies of memory in ill AN patients found that despite normal performance levels on a working memory task, AN participants compared to healthy control subjects exhibited greater activation in the temporal and parietal lobes (Castro-Fornieles et al., 2010). Further, underweight status is related to hypometabolism of glucose in these same regions (Delvenne et al., 1995). Following treatment and weight gain, these differences in neural response were no longer present for AN participants, which suggests the worse nutritional status may be related to less efficient memory-related neural processing (Castro-Fornieles et al., 2010).

Differences in reward learning exist in both AN and BN individuals. Wagner et al. (2007, 2010) found that both AN and BN participants relative to healthy controls had abnormal anterior ventral striatum response to wins and losses. More specifically activation in this reward-related region did not differ whether the participants had won or lost money. This pattern of findings may be related to difficulty discriminating between the valence (positive/negative) of salient stimuli (Wagner et al., 2007, 2010). Further, BN participants appear to have hypoactivation in the insula, ventral putamen, amygdala, and OFC relative to healthy controls during a food-learning task (Frank, Reynolds, Shott, & O'Reilly, 2011). Finally, obese BED participants relative to obese participants without BED exhibited during anticipatory reward/loss processing diminished activation bilaterally in the ventral striatum, as well as in the midbrain, thalamus, and amygdala (Balodis, Kober, et al., 2013). Thus, patterns of reduced neural activation during reward learning may relate importantly to disordered eating.

4.4.2 Addictive Disorders

Addiction is also related to differences in memory and learning. Substances of abuse appear to decrease activity in neural areas associated with short-term memory and attention (Lundqvist, 2005). Substance-related problems are associated with differential neural response to working memory tasks. For example, cannabis users compared to healthy control subjects perform the same on a task of working memory, but parietal cortical activation differs (Jager, Kahn, Van Den Brink, Van Ree, & Ramsey, 2006), and this pattern of results persists following 1 month of

abstinence (Schweinsburg et al., 2008).Though opioid-dependent individuals have performed similarly to healthy controls on working memory tasks, they have concurrently shown heightened frontal, parietal, and cerebellar activation, which may be related to compensatory recruitment of frontal control regions and impaired working memory (Bach et al., 2012; Marvel, Faulkner, Strain, Mintzer, & Desmond, 2012; Yucel et al., 2007). Pretreatment deactivation in the thalamus during a working memory task predicts poorer addiction treatment response (Moeller et al., 2010). Further, chronic cocaine dependence is related to difficulties with adaptive learning that is related to increased connectivity between the ACC networks associated with mental processing (Camchong et al., 2011). Thus, like disordered eating, addictions appear associated with impairments in working memory and learning.

4.5 Emotional Reactivity

4.5.1 Eating Disorders

Increased negative affect has been related to the presence of disordered eating. AN is related to elevated anxiety and stress responsivity (Kaye et al., 2013). Kaye et al. (2013) suggest that AN is related to excess serotonin (related to harm prediction) and reduced dopamine (related to reward prediction), which may lead to increased tendencies towards aversive stimuli and over control. Elevated cortical serotonin 5-HT1A receptor binding is associated with AN (Galusca et al., 2008), and restriction of eating may rebalance serotonin and modulate negative affect or control anxiety for these patients (Kaye et al., 2013). Further, AN participants may find dopaminergic release anxiety provoking rather than hedonically pleasing (Kaye et al., 2013). Increased dorsal caudate/putamen dopamine D2/D3 receptor binding is associated with harm avoidance in AN (Bailer et al., 2007, 2012; Kaye et al., 2013), and dorsal caudate activation in response to negative and positive feedback is associated with trait anxiety in recovered AN (Wagner et al., 2007). Further research suggests that AN is associated with increased activation of neural regions implicated in emotional reactivity when exposed to food. AN patients relative to healthy control subjects had greater activation in the right amygdala when viewing food images (Joos et al., 2011) and greater activation in the amygdala when drinking a milk shake in a hungry state (Vocks et al., 2011). Thus, AN patients may have more intense negative emotional responses when exposed to food.

Binge-eating behavior in the context of ED is also associated with differences in emotion-related neural regions. Binge and purging behavior (in the context of both AN and BN) is related to elevated hypothalamus activation during a Go/No-Go task, which may reflect aberrant emotional responding to the need to inhibit responses (Lock et al., 2011). While some research has not found any differences between BN and healthy control subjects in response to affectively valenced stimuli (Schienle et al., 2004), other studies have found patterns of activation in BN that

suggest an avoidant response to emotions. For example, BN patients relative to healthy controls had decreased neural response in the precuneus to facial expressions of anger and disgust and decreased amygdala response to angry faces, which may be related to emotional avoidance (Ashworth et al., 2011). Pringle, Ashworth, Harmer, Norbury, and Cooper (2011) identified that patients with BN versus healthy control subjects displayed hypoactivation in parietal, occipital, and limbic areas (including the amygdala) when responding to negative self-referential personality words (e.g., evil). This pattern of hypoactivation might reflect emotional blunting or habituation to negative self-thoughts in BN patients (Pringle et al., 2011). Little research has examined emotional reactivity in the context of BED, although this is an important area of future research.

4.5.2 Addictive Disorders

Increased difficulty with emotion regulation is also associated with addictive disorders. Li and Sinha (2008) suggest that deficiencies in prefrontal regions associated with SUD contribute to difficulties with emotion regulation (in addition to executive control). Substance dependence is related to decreased reward responsiveness to pleasant stimuli that are not drug related, which could be related to anhedonia (Volkow, Fowler, & Wang, 2002; Zijlstra, Veltman, Booij, van den Brink, & Franken, 2009). Emotional circuitry may also be altered in addiction. For example, chronic cannabis use is related to reduced activation in the ACC and the amygdala to masked affective faces (Gruber, Rogowska, & Yurgelun-Todd, 2009), and addicted individuals exhibit reduced activation in the amygdala to affective pictures (negative and positive) (Wang et al., 2010). Thus, addiction may be related to reduced emotional response to nondrug stimuli. This pattern of results appears similar to the general pattern of hyporesponsivity to emotional stimuli in BN. BN and SUD may be similarly related to the use of substances (drugs, food) as a way to regulate emotions, whereas AN appears to be linked with caloric restriction to manage mood states.

4.6 Interoceptive Awareness

4.6.1 Eating Disorders

Interoceptive awareness is defined as sensitivity to physiological stimuli originating from the body (Craig, 2002). The anterior insula (a key neural region involved in interoceptive awareness) has been found to be less active for AN participants when thinking about eating food, which suggests less interoceptive awareness (Brooks et al., 2012). In contrast, Gizewski et al. (2010) found that AN compared to control subjects exhibited greater activation in the anterior insula during exposure to high-calorie food pictures, which was interpreted as recall of previously negative eating experiences or elevated emotional arousal. Unlike healthy control subjects, AN

participants did not exhibit activation in the insula when tasting sucrose, and subjective ratings of pleasantness were not associated with changes in insula activation (Wagner et al., 2007). In response to nonfood stimuli, AN relative to control participants had aberrant functioning in the anterior insula and dorsolateral PFC to pain (Strigo et al., 2013). Thus, AN individuals may exhibit difficulty in appropriately perceiving bodily signals, which may allow for greater ability to ignore signs of hunger (Strigo et al., 2013). Less research on interoceptive awareness has been conducted with binge-type ED, especially BED. Schienle et al. (2009) did find that participants with BN had greater activation in the insula to food cues, which suggests the disorder may be related to greater interoceptive awareness, although alternate explanations exist (e.g., greater emotional reactivity to food cues).

4.6.2 Addictive Disorders

Insula function has also been implicated in substance addictions. Insula activation has been related to craving for substances of abuse (Bonson et al., 2002; Wang et al., 2007). Elevated insula activation in response to cigarette cues predicts smoking relapse (Janes et al., 2010), and damage to the insula is associated with decreased craving and markedly increased success in abstaining from smoking (Naqvi & Bechara, 2009; Naqvi, Rudrauf, Damasio, & Bechara, 2007). Thus, differences in insula activation in AN may be related to reduced craving and increased ability to abstain from eating. In contrast, the limited literature in BN suggests that elevated insula response to food cues may be similar to hyperactivity of the insula during substance-related craving in addiction. Future research that examines neural responses to food craving in the context of ED is important.

4.7 Neuroimaging Studies of Comorbid Eating and Addictive Disorders

In addition to comparing neural response between the disorders, neuroimaging studies of patients with comorbid eating and SUD would be helpful in identifying overlapping/differing circuitry. For example, if patients with comorbid addiction and ED displayed similar patterns of neural activation to food and drug cues, this may provide evidence of shared underpinnings. Unfortunately, there is limited research on this topic. One neuroimaging study of participants with remitted bipolar disorder did examine the association of addiction and disordered eating-spectrum scores with brain activation during exposure to affective faces (Hassel et al., 2009). Substance use severity was related to reduced activation in the right PFC to happy faces and the right caudate to neutral faces, whereas elevated disordered eating was linked to elevated right ventral putamen activation to happy and neutral faces (Hassel et al., 2009). Yet, very few participants in the study had concurrent eating

and addictive disorders. Further research on comorbidities will be important in understanding the relationship between disordered eating and SUD.

4.8 "Food Addiction"

"Food addiction" may be another factor potentially linking addiction and problematic patterns of eating. Recent studies have suggested that certain types of foods may be capable of triggering an addictive process in vulnerable individuals (Avena, Rada, & Hoebel, 2008; Johnson & Kenny, 2010). In a sample of young women who did not meet criteria for any ED, greater endorsement of addictive-like eating (e.g., tolerance, withdrawal, continued use despite consequences) was related to increased activation in the ACC, medial OFC, amygdala, DLPFC, and caudate in response to anticipated food receipt, but lower activation in the lateral OFC to food receipt (Gearhardt, Yokum, et al., 2011). This activation is similar to patterns of neural response associated with SUD, namely elevated reward/motivation-related activation to cues and diminished control-related activation during consumption. Thus, an addictive-like response to food may occur outside of the context of traditional ED (e.g., BED).

Clinical research found that approximately half of patients seeking treatment for BED met the threshold for "food addiction," which was associated with more frequent binge eating, greater emotion dysregulation, and elevated pathology (Gearhardt, White, Masheb, & Grilo, 2013; Gearhardt et al., 2012). Future research examining the neural correlates of "food addiction" in ED populations would be useful in evaluating the possible contribution of an addictive process to disordered eating.

4.9 Important Differences Between Eating and Addictive Disorders

There are also important factors that are unique to eating and addictive disorders. For example, body and shape concerns are hypothesized to be important factors in causing and maintaining disordered eating (Fairburn et al., 2008). There have been some investigations that examine the neural correlates of body concerns in eating pathology. For instance, participants with AN exhibit greater amygdala response to body image words, images of their body, and pictures of their body morphed to be heavier, which suggests greater emotional response (Miyake et al., 2010; Seeger, Braus, Ruf, Goldberger, & Schmidt, 2002). In contrast, AN participants displayed greater activation in a reward-related region (i.e., the ventral striatum) to underweight images (Friederich et al., 2010) and also displayed greater activation in the insula and lateral PFC (Mohr et al., 2010). In contrast, participants with BN did not exhibit greater amygdala activation in response to images of their body morphed to look heavier (Miyake et al., 2010).

In addition to increased emotionality in response to body image, ED is also associated with avoidance of body image-related cues. For example, Vocks et al. (2011) found that AN and BN participants had less activation in the inferior parietal lobe (which is related to processing of emotions and sensory information) when viewing pictures of their own body, which the authors interpreted as avoidance. Patients with BN exhibited less activation than healthy control subjects in the middle frontal gyrus and lateral occipital cortex when viewing distorted images of their body (Mohr et al., 2011). Uher et al. (2005) also found that when AN and BN subjects rated body types, they demonstrated hypoactivity in the lateral fusiform gyrus and parietal cortex relative to healthy control subjects. This pattern of results has been interpreted as avoidance of the discomfort caused by activities associated with body image. Little neuroimaging research has been conducted on BED and body image.

Another important difference to consider is the role of the substance being consumed (or avoided) in eating and addictive disorders. Food is necessary for survival, and AN is related to significant malnutrition, which can impact the brain substantially (Delvenne et al., 1997). For example, patients with AN appear to have decreased gray matter in the hypothalamus, inferior parietal lobe, lentiform nucleus, and caudate (Titova, Hjorth, Schiöth, & Brooks, 2013), which may be related to starvation. In contrast to AN, addictive disorders are related to excess consumption of a substance to the point of intoxication, which may also impact the brain. For example, acute alcohol intoxication impacts the functioning of the cerebellum (Volkow et al., 1988) and the OFC, ACC, and primary motor cortex (Calhoun, Pekar, & Pearlson, 2004). Acute cocaine intoxication impacts the ventral tegmental area, substantia nigra, nucleus accumbens, basal forebrain, globus pallidus, amygdala, and subcallosal cortex (Breiter et al., 1997), and acute heroin administration alters cerebral blood flow in the amygdala (Guyer et al., 2007). Thus, food restriction and intoxication both have marked, but different, relationships to neural functioning.

4.10 Summary and Future Directions

In sum, the neuroimaging literature on BED/BN has many similarities with addiction neuroimaging findings, and AN appears to exhibit a more distinct pattern of results in multiple domains (see Table 4.1). These commonalities between BED/BN and addiction speak to the current debate about the potential role of an addictive process in certain types of eating problems (Avena et al., 2012; Ziauddeen et al., 2012). Yet, it is also important to consider the relatively small number of neuroimaging studies focusing on certain types of ED (i.e., BED) and in certain domains (e.g., motivation, reward, interoceptive awareness). More research is needed before strong conclusions can be drawn. An essential future direction is to evaluate the impact of binge consumption of highly processed, calorie-dense foods on neural systems. Neuroimaging studies of individuals with comorbid eating and addictive disorders would also contribute to our understanding of the relationship between

these conditions. Additionally, exploring the association between "food addiction" characteristics and neural responses in patients with ED will be helpful in evaluating how addictive tendencies may contribute to disordered eating. Finally, a greater emphasis on longitudinal designs to parse out preexisting versus later developing (i.e., "causes vs. consequences") events in neurobiology will be important for understanding both ED and addictions.

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Genetic Vulnerability to Eating Disorders and Substance Use Disorders

5

Jessica H. Baker and Melissa A. Munn-Chernoff

Abstract

It is well established that there is substantial comorbidity between eating disorders and substance use disorders. However, it is unclear why these two disorders frequently co-occur. It has been hypothesized that the two disorders may share a common etiology, which could be genetic in nature. There is ample evidence that the eating disorders, specifically anorexia nervosa and bulimia nervosa, and a variety of substance use disorders have a genetic component, yet little research has explored whether these genetic factors are shared. This chapter reviews the current empirical literature indicating that anorexia nervosa and bulimia nervosa and substance use disorders are influenced by genetic factors, as well as preliminary findings exploring whether these disorders indeed share a genetic architecture. We close with suggestions for future research to further elucidate the shared genetic risk between eating disorders and substance use disorders.

Keywords

Anorexia nervosa • Bulimia nervosa • Comorbidity • Eating disorders • Genetics • Substance use disorders

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5.1 Genetic Vulnerability to Eating Disorders and Substance Use Disorders

There is ample evidence suggesting that eating disorders (ED) and substance use disorders (SUD) have a genetic component. In order to discern the familial nature and genetic architecture of these disorders, family, adoption, and twin study designs are utilized. In a family study, risk for a disorder is determined among first-degree relatives of individuals with the disorder (or probands), and this risk is then compared with risk for the disorder in first-degree relatives of individuals without the disorder (or controls). Family studies are often the first step in genetic epidemiology. However, these studies cannot delineate whether a disorder that runs in families is due to genetic or environmental factors. In contrast, adoption studies can explicate the genetic and environmental contributions to observed familiality by comparing similarity for a disorder in biological versus adoptive relatives. If the observed correlations for the disorder are higher among biological relatives, this suggests genetic factors; if the observed correlations are higher among adoptive relatives, this suggests environmental factors.

Twin study designs are also able to elucidate genetic and environmental contributions to familiality. Twin studies decompose the variance of a disorder into genetic and environmental components by comparing the concordance rates of the disorder among identical and fraternal twins. This variance is broken down into additive genetic (i.e., heritability), shared environmental (i.e., environments that increase similarity between twins), and unique environmental factors (i.e., environments that create dissimilarity between twins). However, twin studies are unable to identify which genes are involved in vulnerability towards a disorder.

Finally, molecular genetic approaches can identify specific genes that influence vulnerability towards a disorder. The dominant approach in the molecular genetics field changes rapidly and has included linkage, candidate gene, and genome-wide association studies (GWAS). Linkage studies are used to identify regions in the genome that may harbor genes that predispose individuals to a disorder and are advantageous in that they can be useful for narrowing down the search of the entire human genome to specific regions. Candidate gene studies explore the association between a specific genetic variant and a disorder. If the variant and disorder are correlated, an association is assumed between the two; however, candidate gene association studies require investigators to select a specific gene for analysis based on a hypothesized association between the pathophysiology of the trait and the gene of interest. Due to the necessity of this a priori hypothesizing, candidate gene studies are limited by existing knowledge of the underlying biology of a disorder.

More recent advances in molecular genetic technology have enabled GWAS, which do not focus on one specific gene or set of genes but examine the entire genome. GWAS are able to explore 300,000–1,000,000 genetic markers across the entire human genome; therefore, they have the ability to identify novel genetic variants that may be involved in the vulnerability to a disorder without needing a priori knowledge. An important consideration in GWAS, however, is that very large sample sizes are necessary. Because an entire sweep of the genome is

conducted, a large number of comparisons are made, requiring a greater level of statistical significance ($p < 10^{-8}$).

Here we review the latest empirical evidence suggesting that there is a genetic vulnerability to ED, SUD, and their comorbidity. We provide an overview of family, twin, and molecular genetic study findings for each disorder, as well as initial findings exploring the genetic overlap between ED and SUD. For ED, we focus on anorexia nervosa (AN) and bulimia nervosa (BN); for SUD, we focus on alcohol use disorder (AUD), nicotine dependence, and illicit drug use disorders because these disorders are most commonly explored for their comorbidity and genetic vulnerability. We conclude by discussing burgeoning approaches in the field and ways these approaches can answer important questions about the genetic etiology of this comorbidity.

5.2 Family Studies of Eating and Substance Use Disorders

Initial family studies revealed that ED and SUD aggregate in families. For example, family members of probands with AN are approximately 11 times more likely to develop AN (Strober, Freeman, Lampert, Diamond, & Kaye, 2000). Further, there is a shared familial transmission between AN and BN, such that there is increased risk for BN in relatives of those with AN and vice versa (Lilenfeld et al., 1998; Strober et al., 2000; Walters & Kendler, 1995). The relative risk for BN in females with a relative with AN has been reported at 12.3, whereas females who have a relative with BN have a reported relative risk of 4.2 for developing AN (Strober et al., 2000).

SUD also aggregate within families (Wang, Kapoor, & Goate, 2012). Relatives of individuals with an illicit drug use disorder are at a 4.5-fold greater risk for having an illicit drug use disorder compared with controls, whereas relatives of probands with AUD are twice as likely to have AUD (Merikangas et al., 1998). The familial aggregation of illicit drug use disorders also appears greater among relatives of females than relatives of men, which may suggest a greater familial loading among females (Merikangas et al., 1998). Similar to ED, a cross-familial transmission is suggested between SUD classes (Rietschel & Treutlein, 2013). The prevalence of certain illicit drug use and nicotine disorders is greater in relatives of probands with alcohol dependence than relatives of controls (Bierut et al., 1998; Nurnberger et al., 2004).

Family studies have provided convincing evidence that ED and SUD aggregate within families. Several reports have also suggested cross-familial transmission between ED types and SUD classes. This indicates the familial vulnerability towards these disorders may exist at a general level, while additional factors may play an important role in which ED or substance class disorder emerges.

5.3 Adoption Studies of Eating and Substance Use Disorders

To date, no adoption studies of ED diagnosis have been conducted. In contrast, several adoption studies have examined the familiality of SUD and, in general, suggest that genetic factors play an important role in their familial aggregation (Wang, Kapoor, et al., 2012). However, when exploring the number of diagnostic criteria met for substance dependence in probands, genetic effects were null for familial transmission for alcohol dependence until five criteria were met, whereas only one criteria was necessary for genetic effects to be evident for drug dependence (Yates, Cadoret, Troughton, & Stewart, 1996).

5.4 Twin Studies of Eating and Substance Use Disorders

Twin studies have corroborated the familial nature of ED and SUD. Heritability estimates have ranged from 28 to 74 % for AN and from 28 to 83 % for BN, with the remaining variance attributable to unique environmental factors (Trace, Baker, Penas-Lledo, & Bulik, 2013). For SUD, results have suggested that genetic factors are highly involved. For example, genetic factors have been implicated for alcohol, nicotine, cannabis, stimulant, and cocaine abuse or dependence. In regard to alcohol dependence, heritability estimates typically range between 40 and 60 % (Wang, Kapoor, et al., 2012), with an average heritability of 57 % (Sullivan, Daly, & O'Donovan, 2012). The average heritability for nicotine dependence is similar, estimated at 67 % (Sullivan et al., 2012), whereas heritability estimates range between 30 and 80 % for illicit substance dependence (Wang, Kapoor, et al., 2012).

Twin research has also examined whether the genetic risk factors for an SUD are specific or nonspecific. If these factors are nonspecific, they would predispose individuals to misuse a range of substances, whereas specific factors would predispose an individual to misuse a specific substance or substance class. In general, results suggest that the genetic risk for an SUD is not substance specific, but is a general factor predisposing individuals to a range of SUD (Kendler, Jacobson, Prescott, & Neale, 2003; Kendler, Myers, & Prescott, 2007; Tsuang et al., 1998). Thus, environmental factors (e.g., peer group, access to substances) likely impact which substance is used.

5.5 Molecular Genetic Studies of Eating and Substance Use Disorders

A significant number of reports have examined the association between specific genetic variants and ED and SUD independently. The following section will review molecular genetic studies that have been conducted within the ED field and within the SUD field, with candidate gene studies focusing on those genetic variants that may be most relevant for the comorbidity between ED and SUD.

5.5.1 Linkage Studies

Few linkage studies of ED exist, although there are more reports for SUD. For ED, studies have found several linkage peaks on various chromosomes: for AN, chromosomes 1 (Devlin et al., 2002; Grice et al., 2002), 2, and 13 (Devlin et al., 2002) have been implicated, whereas for BN, chromosomes 10 and 14 (Bulik et al., 2003) have been implicated. The greater number of linkage studies for SUD compared with ED is due, in part, to the higher prevalence of some SUD. As discussed in a review (Wang, Kapoor, et al., 2012), a number of linkage peaks have been identified for alcohol dependence including regions in chromosomes 4 and 14.

Over 20 linkage studies have been conducted for nicotine dependence and have been reviewed in detail elsewhere (Li, Ma, & Beuten, 2004). A recent meta-analysis implicated a region on chromosome 17 for a combined measure of smoking-related behaviors, whereas suggestive linkage was identified at chromosome 5 for a measure of nicotine dependence (Han, Gelernter, Luo, & Yang, 2010). Additional linkage studies have been reported for cannabis, including linkage to regions on chromosomes 1, 2, 3, 8, 9, and 14 for cannabis dependence and related constructs (Agrawal, Hinrichs, et al., 2008; Agrawal, Pergadia, et al., 2008; Ehlers, Gizer, Vieten, & Wilhelmsen, 2010; Han et al., 2012; Hopfer et al., 2007). Finally, other SUD linkage peaks have been identified including chromosomes 9 and 12 for cocaine dependence (Gelernter et al., 2005) and chromosome 14 for opioid dependence (Lachman et al., 2007).

5.5.2 Candidate Gene Studies

5.5.2.1 Dopamine Genes

The dopamine system has been extensively studied in SUD, as it is known to be involved in reward, motivation, motor activity, cognition, emotion, and food intake. For ED, studies have suggested that the A1 allele in the TaqIA polymorphism (i.e., rs1800497) in the dopamine D2 receptor gene/ankyrin repeat and kinase domain containing 1 (*DRD2/ANKK1*) gene are associated with sensation-seeking among women with bulimia-spectrum disorders who also experienced childhood sexual abuse (Groleau et al., 2012). In contrast, other research has suggested that as the number of A2 alleles of the TaqIA polymorphism increases, the greater the association with purging AN (Bergen et al., 2005). Additional research has focused on the association between ED and polymorphisms in the catechol-O-methyltransferase (*COMT*) gene; however, results are inconclusive. Some studies suggest that the Val allele of the Val158Met polymorphism (i.e., rs4680) is significantly associated with AN (Frisch et al., 2001; Mikolajczyk, Smiarowska, Grzywacz, & Samochowiec, 2006) and BN (Yilmaz, Kaplan, Zai, Levitan, & Kennedy, 2011), whereas others suggest that the Met allele is associated with AN (Michaelovsky et al., 2005) or that there is no association between alleles in this polymorphism and ED (Brandys et al., 2012). Although there are other genes in the dopamine system, less research has investigated their association with ED.

Numerous studies have demonstrated an association between genetic variants in the dopamine system and alcohol dependence. Two meta-analyses (Munafo, Matheson, & Flint, 2007; Smith, Watson, Gates, Ball, & Foxcroft, 2008) on approximately 40 studies investigating the TaqIA polymorphism, the most widely studied polymorphism in the addiction literature, indicated that the A1 allele was significantly associated with alcohol dependence. On the other hand, a recent meta-analysis of the Val158Met polymorphism did not find a significant association with alcohol dependence (Tammimaki & Mannisto, 2010).

For nicotine dependence, meta-analyses have indicated that the A1 allele increases risk for nicotine dependence (Li et al., 2004; Munafo, Clark, Johnstone, Murphy, & Walton, 2004), whereas the Val allele significantly increases risk for smoking (Tammimaki & Mannisto, 2010). Two specific groups of alleles across multiple polymorphisms (i.e., haplotype) in the *COMT* gene have also been shown to be differentially protective against smoking in African-American women and European-American men (Beuten, Payne, Ma, & Li, 2006).

No study has examined associations between cannabis abuse or dependence and variants in dopamine genes, but there is evidence to suggest that the TaqIA polymorphism increases risk for a cannabis-related "high" at an earlier age in adolescent boys compared with their peers who did not have the A1 allele (Conner et al., 2005). Furthermore, a meta-analysis has suggested that the Val allele of the Val158Met polymorphism is associated with increased risk for cannabis abuse (Tammimaki & Mannisto, 2010). For other illicit drugs, the A1 allele of the TaqIA polymorphism is associated with greater heroin consumption and resistance to treatment outcome; however, findings on psychostimulants are conflicting. No significant associations between the Val158Met polymorphism and opioid addiction or stimulant abuse have been observed (Tammimaki & Mannisto, 2010). However, other variants in the *COMT* gene, as well as additional genetic variants in the dopamine system (*DRD2/ANKK1*, dopamine transporter (*SLC6A3*), and dopamine β hydroxylase (*DβH*)), have been associated with cocaine abuse or dependence (Haile, Kosten, & Kosten, 2007).

Taken together, candidate gene studies on genetic variants in the dopamine system and their association with ED and SUD implicate similar genetic risk factors. Although several replication studies have been conducted for alcohol and nicotine dependence, more studies are needed to understand whether genetic variants in the dopamine system confer risk for other SUD, as well as AN and BN.

5.5.2.2 Serotonin Genes

One of the most widely studied systems in psychiatric research is the serotonin system because it has been associated with aggression, sleep, personality, mood and appetite regulation. Extant research within the ED and SUD fields has examined genetic variants on two separate serotonin genes. For ED, most studies have focused on a single polymorphism in the promoter region of the serotonin transporter gene (*SLC6A4*), *5-HTTLPR*. Two meta-analyses reported that individuals who had at least one copy of the short allele were more likely to have AN compared with individuals who had two copies of the long allele (Calati, De Ronchi, Bellini,

& Serretti, 2011; Lee & Lin, 2010). There were no significant associations between this polymorphism and BN (Lee & Lin, 2010). Several studies have also examined the association between a promoter region polymorphism (-1438G/A, rs6311) of *HTR2A* and AN. Although an initial meta-analysis reported an absence of an association between this genetic variant and AN (Ziegler et al., 1999), a more recent meta-analysis indicated an association (Gorwood, Kipman, & Foulon, 2003).

For AUD two meta-analyses have reported that the short allele of *5-HTTLPR* is significantly associated with alcohol dependence (Feinn, Nellissery, & Kranzler, 2005; McHugh, Hofmann, Asnaani, Sawyer, & Otto, 2010). The G allele of the -1438G/A polymorphism in the *HTR2A* gene was also significantly associated with alcohol dependence in a sample of Japanese individuals whose *ALDH2* gene, a gene with established connections to alcoholism, was inactive (Nakamura et al., 1999). Moreover, the presence of the G allele in -1438G/A distinguished alcohol-dependent patients from heroin-dependent patients when the individuals were also carriers of the *5-HTTLPR* short allele (Saiz et al., 2009). Although findings with *5-HTTLPR* appear robust, additional work is necessary to confirm the association between this *HTR2A* polymorphism and alcohol dependence.

Studies examining *5-HTTLPR* and nicotine dependence or related phenotypes have been mixed. As reviewed elsewhere (Herman & Balogh, 2012), some studies report an association with the short allele, others with the long allele, and still others report no association. However, the A allele of the -1438G/A polymorphism in the *HTR2A* gene is associated with tobacco smoking (Polina, Contini, Hutz, & Bau, 2009). Finally, minimal work has explored associations between these two serotonin genes and other drug disorders, and those that do exist are mixed (Herman & Balogh, 2012).

In sum, genetic variants in the serotonin system may influence risk for ED and some SUD. However, replication is essential to fully understand the extent to which these variants contribute to individual vulnerability to these disorders.

5.5.3 Genome-Wide Association Studies

To date, only four GWAS of ED exist. In general, these studies have not found genome-wide significant *p*-values for any single-nucleotide polymorphism (SNP) and AN (Nakabayashi et al., 2009; Wang et al., 2011) or eating disorder symptomatology (Boraska et al., 2012; Wade et al., 2013). Although no GWAS for BN diagnosis currently exist, a GWAS examining a BN spectrum phenotype, which included items asking about self-induced vomiting, binge eating, and bulimia, also showed no significant findings (Wade et al., 2013). These negative findings are likely due to small sample sizes, limiting the ability to detect significant associations. Lastly, one report did identify a region, which included a recurrent 13q12 deletion, only observed among AN women of European ancestry and not control women (Wang et al., 2011).

There are many more GWAS for SUD than for ED. Studies of alcohol dependence have yielded findings that survived genome-wide significance and included SNPs in or near the *C12orf51* gene (Baik, Cho, Kim, Han, & Shin, 2011; Wang, Foroud, et al., 2012) and a SNP (rs1789891) that lies between the *ADH1B* and *ADH1C* genes (Frank et al., 2012; Treutlein et al., 2009). However, some GWAS have not reported significant associations between common SNPs and measures of alcohol dependence (Bierut et al., 2010; Heath et al., 2011; Kendler et al., 2011).

GWAS of nicotine dependence have been the most successful of any psychiatric disorder. The most robust finding comes from three GWAS of smoking-related phenotypes, where SNPs located in nicotinic acetylcholine receptor subunit genes (e.g., *CHRNA5*, *CHRNA3*, and *CHRNB3*) reached genome-wide significance (Liu et al., 2010; Thorgeirsson et al., 2010; The Tobacco and Genetics Consortium, 2010). GWAS of illicit drugs have reported a significant association between two genetic variants in the *ANKFN1* gene (rs1019238 and rs1431318) and cannabis dependence (Agrawal et al., 2011), and differential associations by ethnicity for heroin addiction such that rs10494334 was significantly associated with heroin addiction among European-Americans, whereas in African-Americans, rs950302 in the *DUSP27* gene was associated with heroin addiction (Nielsen et al., 2010).

In general, with the exception of smoking-related phenotypes, GWAS have not been successful in identifying genetic risk factors for ED and SUD. Those SNPs that have emerged have not been in dopaminergic or serotonergic genes. Differences between candidate gene and GWAS findings could result from multiple factors, including the fact that the threshold for significance in GWAS is so high.

5.6 Genetic Vulnerability to Eating and Substance Use Disorder Comorbidity

Given the significant comorbidity between ED and SUD and the strong familiality involved in each, it has been hypothesized that the disorders share a familial vulnerability, albeit genetic or environmental. Similar to the approaches described above, family, twin, and molecular genetic study designs can be used to elucidate whether ED and SUD share a genetic etiology.

5.6.1 Family Studies of Eating and Substance Use Disorder Comorbidity

Family studies have assessed whether ED and SUD aggregate together within families. Initial reports observed an increased likelihood of an SUD in first-degree relatives of probands with BN (Holderness, Brooks-Gunn, & Warren, 1994). However, when proband SUD was controlled for, findings indicated that BN and SUD were transmitted independently (Kaye et al., 1996; Lilenfeld et al., 1997, 1998; Schuckit et al., 1996). Limited reports have explored the prevalence of ED in relatives of those with an SUD or the impact of family history of ED on SUD

vulnerability, but in general, findings also suggest independent transmission (Harrell, Slane, & Klump, 2009; Nurnberger et al., 2004; Schuckit et al., 1996; von Ranson, McGue, & Iacono, 2003).

5.6.2 Twin Studies of Eating and Substance Use Disorder Comorbidity

Similar to the twin design discussed above, bivariate twin designs are used to decompose the correlation between two disorders into genetic and environmental components. These models can also yield the genetic and environmental correlations between the disorders. These correlations represent the correlation between the genetic and environmental factors influencing disorder one (e.g., ED) and the genetic and environmental factors influencing disorder two (e.g., SUD). If the correlations are estimated at 1.0, this would indicate complete overlap.

To date, four studies have explored the genetic overlap between BN and SUD, including AUD and illicit drug use disorders, and, in contrast to family studies, indicate a shared familial association. The first report applied a multivariate twin model to the lifetime history of six psychiatric disorders including BN and alcoholism (alcohol dependence or problem drinking) to elucidate the genetic overlap among these six disorders (Kendler et al., 1995). Although findings revealed that a majority of the genetic liability to alcoholism was independent from the other five disorders (including BN), there was evidence of a small amount of genetic overlap with BN (6 %).

Expanding on this first investigation, Baker, Mitchell, Neale, and Kendler (2010) explored the genetic correlation between a BN symptom count and several SUD including AUD, regular smoking (defined as ever engaging in an average of at least seven episodes of smoking per month), and illicit drug use disorder. Findings suggested small-to-moderate overlap in the genetic factors contributing to the BN symptom count and all SUD examined. The strongest genetic correlation was observed between BN and AUD, estimated at 0.53, whereas the genetic correlation between BN and any illicit drug use disorder was estimated at 0.37 and regular smoking estimated at 0.35.

Examining shared genetic liability between broadly defined BN diagnosis, any illicit drug use disorder, and AUD corroborates findings. Utilizing this broader definition of BN diagnosis, a genetic correlation of 0.39 was observed between BN and an illicit drug use disorder (Baker, Mazzeo, & Kendler, 2007), which is quite similar to the genetic correlation reported above. Confirming previous findings in a large, population-based study of female twins from Sweden, the genetic correlation between BN and AUD was estimated at 0.23 (Trace, Thornton, et al., 2013). Although this correlation is lower than previously reported, the confidence intervals overlap.

Providing further evidence of a shared genetic component between ED and SUD, shared genetic risk has been observed between binge eating, inappropriate compensatory behaviors, and alcohol misuse. Specifically, a genetic correlation of

0.61 and 0.31 was estimated between problematic alcohol use and inappropriate compensatory behaviors and binge eating, respectively (Slane, Burt, & Klump, 2012). A larger investigation corroborated these findings and found significant genetic overlap between alcohol dependence and binge eating and between alcohol dependence and inappropriate compensatory behaviors (estimated genetic correlations of 0.26 and 0.32, respectively) (Munn-Chernoff et al., 2013). These findings suggest that the comorbidity between ED and SUD may be more related to specific ED symptoms as opposed to a specific diagnosis, which would explain why SUD are more common in individuals with a binge-purge-type ED. Clearly, further work is needed exploring the genetic relationship between specific ED symptoms and SUD and to further delineate the inconsistencies in findings across family and twin studies.

5.6.3 Molecular Genetic Studies of Eating and Substance Use Disorder Comorbidity

To date, no molecular genetic studies have explored whether there is a cross-disorder association between ED and SUD. Linkage studies for these disorders independently have shown overlap in linkage peaks (e.g., chromosomes 1 and 14), whereas candidate gene association studies have shown independent associations between similar genetic variants in the dopamine and serotonin systems and ED and SUD. However, in the absence of cross-disorder analyses, we are unable to discern whether the observed overlap in linkage peaks and genetic variants contributes to the comorbidity between ED and SUD. Nonetheless, despite this lack of cross-disorder association analyses, it is likely that at least some of the same genetic variants in dopamine and serotonin genes influence liability to both ED and SUD and their comorbidity. Clearly, this is an area worth further exploration.

5.7 Conclusion and Future Directions

Despite an extensive amount of literature showing that the comorbidity between certain ED and SUD is high, the reasons for this comorbidity are unclear. It has long been hypothesized that ED and SUD share a common etiology, which may include shared genetic influences. Although the results of family studies have been inconsistent, twin studies uniformly suggest there is at least a small amount of overlap in genetic risk. Moreover, two promising studies suggest that the symptoms of binge eating and inappropriate compensatory behaviors may be the "genetic link" between ED and SUD—which may account for some of the inconsistencies observed across the literature.

Further, although numerous studies have investigated whether genetic variants contribute to ED and SUD, more work is needed to identify genetic variants that contribute to their comorbidity. Genes in the dopamine and serotonin systems may be important, as well as other genes that have accumulated less evidence (e.g.,

genes in the opioid system, such as *OPRD1*). Future studies should focus on genetic variants in these key neurotransmitter systems and the shared association between binge eating and/or inappropriate compensatory behaviors and SUD.

An additional important next step in identifying the genetic factors influencing comorbidity between ED and SUD is to implement cross-disorder association analyses, specifically cross-disorder GWAS. Cross-disorder GWAS would allow for the identification of shared genetic variants that transcend diagnostic categories and are shared between comorbid disorders—an approach that has been highly successful in identifying shared genetic variants between schizophrenia, bipolar disorder, and major depression (Smoller et al., 2013). This type of cross-disorder analysis would answer important questions as to whether certain genetic variants contribute to the liability to both ED and SUD.

Finally, understanding the genetic factors that increase vulnerability for the comorbidity between ED and SUD not only have important implications for our understanding of etiology, but also have important implications for prevention, detection, and treatment. For example, prevention efforts can be developed for those at genetic risk (e.g., those with first-degree relatives with an ED or comorbid ED-SUD). Ultimately, a thorough understanding of the genetic architecture of this comorbidity will enrich our ability to prevent, detect, and treat these disorders independently, as well as their comorbidity.

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Dimensions of Personality and Neuropsychological Function in Eating Disorders, Substance Use Disorders, and Addictions

6

Carolyn M. Pearson, Leila Guller, and Gregory T. Smith

Abstract

Substance abuse, eating disorders, and other addictive behavior problems tend to be comorbid with personality disorders and with dimensions of personality pathology. Substance abuse and pathological gambling tend to be highly comorbid with antisocial personality disorder, whereas eating disorders are often comorbid with both Cluster B and Cluster C personality disorders. These comorbidities can increase symptom severity and compromise treatment. Negative emotionality, negative urgency, and sensation seeking correlate with most forms of addictive behavior, although anorexia nervosa appears quite different and is associated with lower levels of novelty seeking and high levels of self-directedness and perfectionism. Well-developed neurobiological models of brain system functioning and neurotransmitter plasticity paint a picture of how the initially impulsive engagement in addictive behaviors can become compulsive over time. Behavioral and pharmacological treatment strategies emphasize renewed engagement in nondrug pleasurable activities, and new treatments are being developed that target the specific personality pathology of the patient.

Keywords

Addiction • Anorexia nervosa • Bulimia nervosa • Eating disorders • Pathological gambling • Personality • Personality disorders • Substance use • Treatment

The focus of this chapter is on the relationships among personality disorders, dimensions of personality and substance use, eating disorders (ED), and other addictive behaviors. The chapter is organized as follows. First, we review evidence concerning the comorbidity of personality disorders (PD) and substance use; comorbidity of PD and the ED of bulimia nervosa (BN), binge eating disorder

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(BED), and anorexia nervosa (AN); and comorbidity between PD and the additional addictive behavior of pathological gambling. We then consider research on the topic of whether the presence of PD influences the severity of symptoms and the treatment responsiveness of individuals suffering from addictive disorders. Next, because of evidence that it may be more fruitful to focus on relationships between specific dimensions of personality, rather than PD, and addictive disorders, we summarize key findings in that domain. In the following section of the chapter, we provide an overview of the predominant neurobiological account of drug addiction and review evidence that a similar neurobiological process applies to some forms of ED and to pathological gambling. Another characteristic shared by substance abuse, addiction, and ED is the apparent developmental progression of the disorder from initial, impulsive engagement in the addictive behavior toward compulsive reliance on the addiction over time. We provide a brief introduction to a model explaining that progression. Lastly, we consider treatment implications of the observed relationships between personality and addictive behaviors.

6.1 Comorbidity of Personality Disorders and Addictive Behaviors

In general, Cluster B PD, most prominently antisocial personality disorder (ASPD) and borderline personality disorder (BPD), have high rates of comorbidity with substance abuse, pathological gambling, BN, and BED. Cluster C PD tend to co-occur more frequently with AN. We next summarize specific findings on this topic.

6.1.1 PD and Substance Use

Using a large, nationally representative sample, Compton, Conway, Stinson, Colliver, and Grant (2005) documented the comorbidity between ASPD and numerous substance use disorders (SUD). Strikingly, there is a relationship between abuse of several different categories of drugs and ASPD. For example, having an alcohol use disorder (AUD) was associated with an eightfold increase in the odds of also having ASPD. Those diagnosed with abusing sedatives were 12.5 times as likely to have ASPD. Similar increased odds were present for virtually all substances studied: For opioid abuse, an odds ratio of 12.8; for amphetamine abuse, an odds ratio of 11.9; for hallucinogen abuse, an odds ratio of 14.0; for cocaine abuse, an odds ratio of 10.9; and for inhalant abuse, an odds ratio of 18.7. Considering behaviors relevant to ASPD, Compton et al. (2005) also examined adult engagement in antisocial behavior. The results were similar: For the same set of substance abuse diagnoses, the odds of also being diagnosed with ASPD ranged from being 4.4 times as great as those without the disorder to 8.8 times as great.

6.1.2 PD and Eating Disorders

There appear to be high rates of comorbidity between PD and each of the most common ED: AN, BN, and BED. Estimates for the overall rate of comorbidity vary widely, ranging from 27 to 93 % (Vitousek & Manke, 1994). Complicating the estimation process is that comorbidity appears higher when PD are assessed by questionnaire and lower when they are assessed by interview. Cassin and von Ranson (2005) concluded that questionnaire assessment overestimates comorbidity; it is not known whether interview assessments also underestimate comorbidity. Nonetheless, even by interview, rates of comorbidity are high (Cassin & von Ranson, 2005).

Concerning specific types of PD, it is the case that among individuals with AN, particularly AN restricting type (ANR), the Cluster C disorders of dependent personality disorder (DPD), avoidant personality disorder (APD), and obsessive–compulsive personality disorder (OCPD) co-occur the most frequently (Cassin & von Ranson, 2005; Halmi et al., 2005). The magnitude of comorbidity is estimated to be much higher when questionnaire assessment is used (prevalence rates range from 42 % for OCPD to 50 % for APD) than when interviews are used (average prevalence rates across studies were 7 % for DPD, 14 % for APD, and 15 % for OCPD). For individuals with ANR, questionnaire assessments identify high comorbidity with BPD from Cluster B (39 %), but that finding was not observed with interview assessment (3 %). In general, the rates of ANR comorbidity with Cluster A and Cluster B PD are lower than those for Cluster C, regardless of assessment method.

Individuals with BN appear to have high rates of comorbidity with most forms of PD, and particularly with Cluster B and C disorders. When assessed by questionnaire, a meta-analysis found comorbidity estimates of 41 % with DPD, 40 % with APD, 33 % with histrionic personality disorder (HPD), 32 % with BPD, and 28 % with OCPD (Cassin & von Ranson, 2005). Again, assessment by interview produced much lower estimates of comorbidity: 21 % with BPD from Cluster B; 19 % with APD from Cluster C; 10 % with DPD from Cluster C; and 9 % each with Cluster B's HPD and Cluster C's OCPD (Cassin & von Ranson, 2005; also see Halmi et al., 2005, on comorbidity between BN and OCPD). Regardless of which assessment method provides the most accurate estimates, it is clear that individuals with BN often have Cluster B and/or Cluster C personality disorders as well.

To date, few studies have investigated comorbidity between BED and PD. Cassin and von Ranson's (2005) meta-analysis was able to aggregate data across studies only for the interview assessment method. They found substantial comorbidity with three PD: Cluster C's APD, 11 %; Cluster C's OCPD, 10 %; and Cluster B's BPD, 9 %. To summarize, Cluster C personality disorders co-occur frequently with each type of ED and are perhaps most likely to co-occur with AN; Cluster B disorders tend to co-occur most frequently with BN and BED.

6.1.3 PD and Pathological Gambling

In a clinical study, Blaszczynski and Steel (1998) assessed the PD status of 82 consecutive admissions to a treatment program for gambling problems. Fully 93 % of the participants met diagnostic criteria for at least one PD, with an average of 4.6 PD per participant. The most common PD were from Cluster B: BPD (69.5 % of participants), HPD (65.9 %), and narcissistic personality disorder (NPD: 57.3 %). Not surprisingly, the presence of these disorders was associated with high levels of affective instability and impulsivity. Interestingly, there was also a high rate of Cluster C disorders in this sample: DPD (48.8 %) and APD (36.6 %) were the two most frequent. Research on possible mechanisms by which these different personality disorder tendencies transact to increase risk for gambling addiction is indicated.

In an extensive study using 4,497 twin pairs, Slutske and colleagues (2001) found a strong association between pathological gambling and ASPD (and also with conduct disorder and ASPD). It does not appear to be a simple case in which ASPD causes pathological gambling or the reverse, because the association between the two was predominantly explained by common genetic factors. Thus, current evidence highlights the comorbidity between pathological gambling and each Cluster B PD, as well as implicating some role for Cluster C PD.

6.2 The Impact of Personality Disorders on Severity and Treatment Responsiveness for Substance Use, Eating Disorders, and Pathological Gambling

6.2.1 Substance Use

In a sample of 606 substance abuse patients, Westermeyer and Thuras (2005) found the following. Substance abuse patients who also had ASPD reported more substance-related family problems, interpersonal problems, legal problems, financial problems, and occupational problems. They also had more psychological and pharmacological symptoms. In addition, the substance abuse–ASPD comorbid patients had more family members who had been diagnosed with SUD. They were more likely to use tobacco, amphetamines, cannabis, opioids, and cocaine than were other substance abuse patients. Concerning treatment, they had 50 % more treatment admissions and spent 64 % more days in treatment. They were also exposed to more treatment modalities, and the accumulated cost of treating them was 58 % higher than it was for other SUD patients.

6.2.2 Eating Disorders

Several studies have found that the presence of BPD among individuals with ED is associated with greater psychopathology and poorer functioning (Bruce & Steiger,

2005; Wilfley et al., 2000; Wonderlich & Mitchell, 2001). In some studies, BPD has also been found to be associated with greater ED symptomatology in particular (Johnson, Tobin, & Dennis, 1990; Johnson, Tobin, & Enright, 1989; Wilfley et al., 2000). For example, Johnson et al. (1989) found that more ED patients with BPD used laxatives, and Johnson et al. (1990) found that ED patients with BPD were less likely to have experienced remission of their ED symptoms after 1 year and, at that 1 year follow-up, were higher than non-BPD patients on several important risk factors, such as drive for thinness, distorted body image, personal ineffectiveness, interoceptive awareness difficulties, interpersonal distrust, and maturity fears. Wonderlich et al. (2007) developed a typology of individuals with BN and found that the subset high in interpersonal–emotional distress (and thus similar to those high in BPD) were more likely than others to have comorbid mood and anxiety disorders and to have cognitive, affective, impulsive, and interpersonal dysfunction. They were also the group with the highest rates of ED symptoms and they underwent more therapy for more months than did others. Concerning treatment, the presence of BPD, or of other Cluster B PD, is associated with poor treatment response in anorexia nervosa–binge purge type (ANBP), BN (Wonderlich & Mitchell, 1997, 2001), and BED (Wilfley et al., 2000). Interestingly, a study of the natural course of BN and of eating disorder not otherwise specified (EDNOS) found that course was not influenced by the presence or severity of BPD, OCPD, or APD (Grilo et al., 2003).

6.2.3 Pathological Gambling

In a large ($n = 237$) sample of individuals seeking outpatient treatment for pathological gambling, 16.5 % met diagnostic criteria for ASPD (Pietrzak & Petry, 2005). Compared to other pathological gamblers, this comorbid group began gambling earlier in life and reported greater severity of gambling problems, greater severity of substance use problems, more medical problems, and higher levels of paranoid ideation, phobic anxiety, and somatization (Pietrzak & Petry, 2005). Pathological gamblers with comorbid ASPD are also more likely to commit criminal offenses (Blaszczynski & McConaghy, 1994). Clinical case descriptions suggest that ASPD complicates gambling treatment, reducing treatment efficacy (Law, 2013).

6.3 The Relationship Between Specific Dimensions of Personality and Substance Abuse, Eating Disorders, and Pathological Gambling

While the comorbidity of PD and substance use, ED, and gambling is important and informative, some researchers argue that it is more fruitful to focus on relationships between specific dimensions of personality and addictive behaviors. Generally, negative emotionality and impulsivity/behavioral disinhibition tend to be the two

specific dimensions of personality that are commonly associated with the addictive behaviors of interest. We next summarize those findings.

6.3.1 Personality and Substance Use

In a large 7-year longitudinal study, Sher, Bartholow, and Wood (2000) found that the personality traits most clearly related to disinhibition, such as novelty seeking, most consistently predicted a variety of SUD both cross-sectionally and prospectively. Negative emotionality, as measured by neuroticism and harm avoidance, were both predictors cross-sectionally, but did not demonstrate robust predictions of SUD diagnoses prospectively under stringent statistical analyses (Sher et al., 2000). Compared to SUD, which seems to be specifically associated with problems in constraint and behavioral control (McGue, Slutske, & Iacono, 1999), AUD tend to be characterized specifically by the trait of negative emotionality and some problems with impulsivity-like traits (McGue et al., 1999; McGue, Slutske, Taylor, & Iacono, 1997). In fact, the rate of alcoholism was found to be especially high among those who scored extreme on both negative emotionality (high scores) and constraint (low scores, thought to be analogous to a form of impulsivity) (McGue et al., 1997).

Given the importance of both emotion and behavioral disinhibition/impulsivity, researchers have extensively studied many different traits that are related to impulsive behavior, two of which include emotional components. One is positive urgency, the tendency to act rashly when experiencing a positive mood, and the other is negative urgency, the tendency to act rashly when experiencing a negative mood. Longitudinal research with college freshmen has shown that positive urgency predicts (a) engagement in illegal drug use, (b) increases in quantity of alcohol consumption, and (c) problems associated with alcohol use (Cyders, Flory, Rainer, & Smith, 2009; Settles, Cyders, & Smith, 2010; Zapolski, Cyders, & Smith, 2009). Similarly, negative urgency has been shown to be associated with drinking quantity and negative outcomes associated with alcohol consumption (Cyders et al., 2009; Settles et al., 2010). Thus, the personality traits that link emotionality and impulsivity may be of particular importance for substance use.

6.3.2 Personality and Eating Disorders

For AN, self-oriented and socially prescribed perfectionism prospectively predicts onset (Cassin & von Ranson, 2005), and obsessive–compulsive traits tend to increase the odds of developing the disorder (Anderluh, Tchanturia, Rabe-Hesketh, & Treasure, 2003; Cassin & von Ranson, 2005) as does narcissism (Cassin & von Ranson, 2005). Although this profile is similar to those with BN (Cassin & von Ranson, 2005), individuals with ANR (anorexia nervosa, restricting type) tend to be higher in self-directedness (Klump et al., 2000), higher in persistence, and lower in novelty seeking than those with BN or no disordered eating (Cassin & von Ranson,

2005). Individuals who binge eat and/or purge, on the other hand, tend to be characterized by more impulsive and emotion-based traits. First, those who are diagnosed with ANBP, BN, and BED tend to score higher on measures of sensation seeking than ANR or controls (Rossier, Bolognini, Plancherel, & Halfon, 2000; Steiger, Jabalpurwala, Champagne, & Stotland, 1997), and BN inpatients and outpatients seem to be more impulsive than individuals with ANR type and nonpsychiatric controls (Cassin & von Ranson, 2005). Second, negative emotionality and neuroticism are associated with binge eating and purging symptoms (Cassin & von Ranson, 2005; Lilenfeld, Wonderlich, Riso, Crosby, & Mitchell, 2006), and a meta-analysis found that negative affect and neuroticism had a modest average effect size of .09 for BED and BN (Stice, 2002). Research suggests that negative mood precedes binge eating and that binge eating may in fact be an attempt to regulate or modulate negative mood (e.g., Smyth et al., 2007). When examining the role of specific impulsivity traits, negative urgency concurrently and prospectively predicts BN and BED symptoms above and beyond all other impulsivity-related traits, including sensation seeking, lack of perseverance, and lack of planning (Fischer, Smith, & Cyders, 2008; Pearson, Combs, Zapolski, & Smith, 2012).

6.3.3 Personality and Gambling

Similar to the other addictive behaviors, researchers have focused on traits related to impulsivity and emotionality. In one large, 3-year longitudinal study, researchers found that even after controlling for substance use and sex, young adults with pathological gambling scored significantly higher on negative emotionality and significantly lower on constraint (Slutske, Caspi, Moffitt, & Poulton, 2005). However, when examining a subgroup involved only in gambling, negative emotionality was no longer elevated, suggesting that the high levels of negative affect that predicted problem gambling may be due, in part, to comorbid alcohol or nicotine dependence and that pure gambling may rather be a result of significantly low constraint, or problems in impulsivity and behavioral inhibition (Slutske et al., 2005). Another study that investigated the longitudinal influence of impulsivity traits on gambling among college freshman found that while sensation seeking, positive urgency, and lack of planning all concurrently predicted gambling behavior, only positive urgency uniquely predicted increased gambling behavior prospectively (Cyders & Smith, 2008a). Thus, it seems that positive mood and impulsivity are both important predictors of future problem gambling.

6.4 Neurobiological Account of Drug Addiction

It seems that personality traits that predispose one to emotion-based impulsive action are common among addictive behaviors. One explanation for this commonality can be found in a neurobiological model to explain emotion-driven rash or impulsive action.

There appears to be a functional brain system involved in the processing of emotion-laden experiences and preparing for action involving interconnections between the amygdala and the orbitofrontal cortex (OFC) and its medial sector (the ventromedial prefrontal cortex or VMPFC) (Barbas, 2007; Bechara, Tranel, & Damasio, 2000; Ghashghaei & Barbas, 2002; LeDoux, 2000; Lewis & Todd, 2007). The amygdala is responsive to emotionally salient stimuli; in particular, to distressing stimuli. There are numerous "bottom-up" connections between the amygdala and the OFC/VMPFC. These connections are understood to serve the function of orienting these areas of the PFC to attend to, and prepare behavioral responses to, important or emotionally salient stimuli (Cardinal, Parkinson, Hall, & Everitt, 2002; Lewis & Todd, 2007). The OFC and VMPFC, in turn, have numerous "top-down" connections back to the amygdala; these connections enable the PFC to modulate amygdala functioning. Specifically, they appear to carry information about possible consequences of actions and a bias toward long-term interests back to the amygdala. Essentially, recognition of consequences of prospective behaviors and a concern for long-term goals inhibit amygdala functioning, thus reducing the degree of emotion-driven action following amygdala activation. Thus, in a well-functioning system, individuals recognize emotionally salient stimuli and attend to them. Their responses to those stimuli involve attempts to meet the need underlying the emotion, but in a way that does not harm one's long-term interests and goals.

However, reduced functioning of the OFC/VMPFC can result in fewer cautioning signals to the amygdala, resulting in increased levels of emotion-driven action uninformed by one's long-term interests. On the most basic level, individuals with damaged OFC/VMPFC display an impaired ability to effectively anticipate the potential risks of their actions (Bechara, Damasio, Tranel, & Damasio, 2007). Other studies have shown that OFC/VMPFC-damaged individuals are sensitive to immediate reinforcement and punishment and oblivious to future consequences of their actions (Bechara, Damasio, Damasio, & Anderson, 1994). They seem unable to inhibit emotion-driven actions in light of their own and others' interests and well-being; instead, they are guided by immediate consequences only. Put differently, they lack effortful control or purposeful inhibition of emotional responses.

Another way in which this system can malfunction, leading to affect-driven action, is through neuromodulators like serotonin (5HT) and dopamine (DA; Lewis & Todd, 2007). Experimental and correlational designs have demonstrated that low levels of 5HT are associated with greater rates of reckless behaviors that often involve risk, together with greater levels of negative affect (Cools et al., 2005; Depue & Collins, 1999; Frankle et al., 2005; Krakowski, 2003; Morgan, Impallomeni, Pironi, & Rogers, 2006; Spoont, 1992; Winstanley, Dalley, Theobald,

& Robbins, 2004; Winstanley, Eagle, & Robbins, 2006; Winstanley, Theobald, Dalley, Glennon, & Robbins, 2004; Zald & Depue, 2001). Considering the brain system of interest, low levels of 5HT result in lower levels of OFC/VMPFC functioning and therefore fewer top-down transmissions from the OFC/VMPFC back to the amygdala in the presence of strong emotion. This results in lesser inhibition of the amygdala, a stronger and more lasting emotional response, and reduced ability to consider the possible long-term consequences of actions. One result is an increased disposition toward disinhibited or rash action (Cyders & Smith, 2008b).

For some receptor subtypes and in some brain areas, 5HT modulates dopamine (DA) activity (Morelli et al., 2011). In those cases, when low levels of 5HT are present, DA levels are less inhibited, thereby increasing levels of DA in the brain. Thus, areas of one's brain can have low levels of 5HT and high levels of DA. Elevated levels of DA are associated with a tendency to act (Depue, 1995; Depue & Collins, 1999; Zald & Depue, 2001) and with increasing reward-seeking and risk-taking behaviors (Spear, 2000), including emotional dysregulation, impulsivity, and cognitive–perceptual impairment (Friedel, 2004). High levels of DA activity in the amygdala–OFC/VMPFC circuit are associated with high rates of rash or reckless acts (Floresco & Tse, 2007). One result of the combination of low 5HT and high DA in this functional brain system is an overall reduction in OFC/VMPFC modulation of amygdala-driven responses, or a reduction in effortful control/affect-guided planning and an increased likelihood of engaging in rash, ill-considered acts (Cyders & Smith, 2008b; Depue, 1995). It is important to appreciate that the combination of low 5HT and high DA is one of many different patterns of neurotransmitter elevation that can occur in different regions of the brain, and there are circumstances in which 5HT and DA covary positively (Rothman, Blough, & Baumann, 2008).

6.4.1 Neurobiology of Substance Use

The brain system described above explains how one is at risk, neurobiologically, to engage in a rash, impulsive action while experiencing a strong emotion. Next, we briefly describe how this individual is at risk for continued engagement of that behavior by describing the most prominent neurobiological model of drug addiction. Each time an individual uses a drug, there is an increase in dopaminergic activity, which has been shown to be associated with subjective perceptions of reward (Volkow & Wang, 1996). These feelings of pleasure reinforce the drug use so the individual comes to choose it as a way to increase positive affect and decrease negative affect. However, the neuroplasticity associated with the drug use causes an elevation in the brain reward threshold, thereby creating a greater and greater elevation in "baseline" thresholds (Koob & Volkow, 2010). Because the reward function fails to return within normal homeostatic range, the individual need more of the drug more often to achieve the same effect of immediate reward and pleasure.

When the reward system is chronically activated by drug use, the brain adapts by decreasing the number of dopamine D_2 receptors, which results in a decreased sensitivity of the reward circuits to stimulation by natural reinforcers (Martin-Solch et al., 2001; Volkow & Fowler, 2000). Consequently, after extensive, ongoing drug use, one comes to find little pleasure in everyday activities and instead craves the drug and focuses on seeking the drug. Furthermore, the chronic activation of the reward system also activates an anti-reward circuit that has the opposing actions of limiting the drug's reward function (Koob, 2005). Together, these processes decrease sensitivity to rewards (Volkow et al., 2010). The combination of the loss of function of natural reward systems and the recruitment of the brain stress or anti-reward systems creates a situation of chronic loss of pleasure and ongoing negative affect; these states can only be alleviated by ever-increasing amounts of the drug on an escalating frequency of occasions. Compounding this difficulty is that, over time, substance abusers experience enhanced sensitivity of memory circuits to conditioned expectations of reward from drug use; these conditioned expectations then drive further drug use (Volkow et al., 2010). Use of the drug becomes a powerful source of negative reinforcement and of anticipated pleasure. Individuals choose to continue using the drug to achieve negative reinforcement, despite knowing that the use has negative consequences.

6.5 Neurobiology of BN

There is evidence that this process operates for BN, as well. Women with BN tend to have low levels of the major metabolite of 5HT (Kaye, Gwirtsman, George, & Ebert, 1991; Steiger & Bruce, 2007). Impaired serotonergic responsiveness is often cited during the acute illness state of BN (e.g., Brewerton, 1995; Brewerton et al., 1992; Goldbloom, Garfinkle, Katz, & Brown, 1990; Kaye et al., 1998) and is also associated with BN symptom severity (Jimerson, Lesem, Kaye, & Brewerton, 1992). Moreover, higher levels of impulsivity in women with BN are associated with low levels of 5HT measures (Steiger, Israel, Gauvin, Kin, & Young, 2003). The rash BN behaviors of binge eating and purging therefore appear associated with alterations in brain systems that are associated with reduced effortful control or reduced affect-guided planning.

Research on DA levels in BN is typically conducted in the brain's reward systems since DA is the neurotransmitter most associated with reward (e.g., Broft et al., 2012; Jimerson et al., 1992). Women with BN tend to have a lower number of DA receptors, and there is a significant negative association between the frequencies of binge eating and purging and the striatal DA response (Broft et al., 2012). Because of this decreased response, more DA need to be released to achieve the same rewarding response. An increase in DA is associated with reward value (Volkow et al., 2002, 2003). Thus, similar to what is observed in individuals with SUD (Volkow et al., 2002, 2003), individuals with BN tend to have lower baseline levels of DA responding, which is associated with reduced baseline levels of

pleasure or reward, thereby experiencing less frequent positive affect and also requiring larger upsurges in DA to experience reward.

6.6 Neurobiology of AN

Although the focus of this section of the chapter concerns behaviors that are often described as impulsive, there is considerable evidence that AN should be regarded quite differently. As noted above, AN, particularly ANR, is highly comorbid with OCPD, and attributes such as cognitive and behavioral inflexibility (Zastrow et al., 2009), rigidity, preference for symmetry and exactness, low impulsivity, avoidance of novel situations, and obsessiveness (Cassin & von Ranson, 2005; Fassino et al., 2002) tend to characterize the personalities of individuals with AN. Zastrow et al. (2009) found that women with AN had particular deficits in behavioral response shifting, i.e., they had trouble changing their behavior when changing task demands required it. This deficit was associated with reduced activation in the left and right thalamus, ventral striatum, anterior cingulate cortex, sensorimotor brain regions, and cerebellum.

Another intriguing possibility for understanding the neurocognitive underpinnings of some aspects of AN was provided by Jarry and Vaccarino (1996), who observed that high levels of 5HT in the same functional brain system described above (i.e., variation in 5HT levels in the opposite direction from what characterizes BN) are associated with the preference for symmetry and exactness shared by AN and obsessive–compulsive disorder. High levels of 5HT in this functional system may also be associated with unusually high levels of effortful control.

An additional dimension of AN is body distortion, and there is evidence of impaired spatial orientation skills in women with AN (Nico et al., 2010). Possibly, one thing that contributes to distorted body image in individuals with AN is a deficit in basic spatial orientation abilities. A number of researchers have identified deficits localized to the right parietal lobe as possible contributing factors (Grunwald et al., 2002; Nico et al., 2010).

6.7 Neurobiology of Pathological Gambling

A neurobiological risk process similar to that for BN may be present in individuals identified as pathological gamblers (Potenza, 2008; Zack & Poulos, 2009). Zack and Poulos (2009) review extensive evidence supporting the possibility that the neurochemical basis for pathological gambling is very similar to that for substance abuse and BN. For example, anticipated rewards and uncertainly of reward delivery, both of which are characteristic of gambling, are known to stimulate DA release. Researchers have found cross-priming of motivation for gambling by amphetamine use. In addition, there is some evidence for reduced 5HT, perhaps in the same functional brain system involving the OFC/VMPFC, among

pathological gamblers. DA agonist treatments for Parkinson's disease result in an increase in pathological gambling (Potenza, 2008), a finding consistent with DA facilitating risk-taking or impulsive behavior.

6.8 Developmental Process of Addiction: From Impulsive Behavior to Compulsive Reliance

Just as these addictive behaviors are described as impulsive, they can also be described as compulsive: Individuals with substance use, alcohol use, eating disorders, and/or pathological gambling diagnoses repeatedly engage in the behavior despite knowing and experiencing the harms associated with the behavior. Continued engagement in a behavior despite knowing that doing so is harmful is referred to as compulsive. Although impulsivity and compulsivity have historically been described as existing on opposite poles of a continuum (Skodol & Oldham, 1996), examination of the body of DSM disorders suggests that these disorders have features of both impulsivity and compulsivity simultaneously, which has led some to conclude that the two terms refer to separate, but partially overlapping constructs (e.g., Engel et al., 2005). We next briefly summarize a model that describes how a behavior is initially impulsive in nature and then becomes compulsive over time (Pearson, Guller, Spillane, & Smith, 2011).

Due in part to the functional brain system described above, some individuals have a tendency to respond to intense emotion with rash, ill-advised action that provides the immediate negative reinforcement of relief from the distress but that otherwise works against the individual's ongoing interests and need. This tendency is described as the personality trait of negative urgency. When one who is high in negative urgency experiences negative mood, such a person feels the urge to act rashly in response to the distress, thereby increasing his or her risk to engage in a risky behavior like using drugs, drinking, gambling, or binge eating and purging. The behavior involves the pursuit of short-term, immediate solutions to one's distress as opposed to resolution of the source of one's distress, which would involve problem-solving over a longer period of time.

The first time one engages in one's impulsive behavior of choice, the act is reinforced as one experiences a reduction in distress. One did not choose a problem-solving, more adaptive way to cope with the distress. Because of the strong, immediate reinforcement, the behavior is chosen more and more frequently over time, thus setting the stage for compulsive engagement in risky behavior. Compulsive reliance on such behavior likely occurs for at least two reasons. First, the immediately reinforcing rash action comes to be consistently chosen when distressed. Second, on each occasion in which one chooses that behavior, one has missed an opportunity to take a different approach to managing one's distress. One has missed a chance to engage in responses that focus on solving the problem that led to the initial distress. By repeatedly missing such chances, one tends not to develop problem-solving coping responses to distress. Over time, one has less and

less access to responses other than the addictive behavior and thus engages in the behavior despite knowing that it will bring harm; it is now compelled.

6.9 Treatment Implications

There are many well-studied, valid behavioral treatments for addictions, such as motivational interviewing (Miller & Rollnick, 2002) and the community reinforcement approach (Meyers & Miller, 2006). There is also reason to believe that a combination of behavioral and pharmacological treatments may work best for many forms of addictive disorder (Potenza, Sofuoglu, Carroll, & Rounsaville, 2011). It is beyond the scope of this chapter to review the treatment literature for each of multiple forms of addiction in depth (see Potenza et al., 2011 for one recent review). Instead, we highlight specific treatment considerations in light of the research we have reviewed.

Given the fact that negative emotions tend to precede the engagement of many addictive behaviors, treatments need to focus on ways in which to reduce negative affect and increase positive affect. This is especially important considering the neurobiological model which suggests that chronic reliance on such behaviors leads to a decrease in DA activity, and therefore chronic states of negative affect. With respect to psychotherapy, incorporating behavioral activation strategies, which are interventions designed so that clients return to engaging in formerly pleasurable activities, seems beneficial. When individuals do return to formerly pleasurable activities, over time they come to find pleasure in them again (Dimidjian et al., 2006), thus providing an addiction-free source of reinforcement. With respect to pharmacotherapy, a focus on enhancing DA functioning and restoring brain circuits altered by chronic drug use may also help individuals engage in nondrug pleasurable behaviors (Volkow, Fowler, Wang, & Swanson, 2004).

An important development is the recognition that it may be valuable for clinicians to distinguish among different personality dispositions as they develop treatment strategies. A client engaging in drug use or alcohol use may be doing so as a function of his or her intense affect, but he or she may, instead, be expressing an intense need to seek new, thrilling stimulation. Making these distinctions is useful, because different preventive or treatment interventions are needed for the different personality underpinnings to this set of problem behaviors.

When treating substance abusers, individuals with ED, and those suffering from pathological gambling, who are also high in negative urgency, clinicians might benefit from using interventions that reduce impulsive actions undertaken while experiencing intense negative emotions. One such therapy, dialectical behavior therapy (DBT; Linehan, 1993), was developed to treat BPD, which is characterized by high emotional vulnerability and lability as well as impulsiveness and low distress tolerance (American Psychiatric Association, 2013). DBT tends to focus on reducing urgency or emotion-driven impulsive acts by teaching skills for emotion regulation and distress tolerance. For instance, DBT includes training on understanding one's emotions, how to tolerate one's distress or one's intense

emotions without engaging in immediate action, and stopping and adjusting one's emotional reactions by considering the context; these skills have all been proven helpful.

Adaptations of DBT have been created for BN (Safer, Robinson, & Jo, 2010; Safer, Telch, & Agras, 2001; Telch, Agras, & Linehan, 2001) and have been shown to be at least somewhat effective for reducing bulimic symptoms. Similar to DBT, integrative cognitive affective therapy (ICAT; Wonderlich et al., 2010) for BN also focuses on regulating emotions in the moment; however, it also includes a goal-focused component to help individuals identify long-term goals and engage in behaviors that help them to meet their long-term goals and need as opposed to a focus on the immediate. Although this therapy is relatively new, preliminary findings suggest its effectiveness in reducing negative urgency type BN behavior and increasing positive behaviors that help one achieve future goals (Wonderlich et al., 2008, 2014).

For those who are high in sensation seeking but not high in negative urgency, the interventions described above are likely to miss the mark. Such individuals get involved in addictive behaviors not to alleviate distress, but instead to meet a strong need for novelty and stimulation. For such clients, interventions that provide individuals with safe alternatives for seeking high levels of stimulation have proven valuable. For example, the use of media messages with high sensation value that encourage alternative, safe means of seeking stimulation, and development of a repertoire of stimulating activities that are safer, provides clients with behavioral options at key choice points that appears to be effective in reducing drug use (Stephenson, 2003).

A difficult challenge for clinicians treating addictive behaviors is their comorbidity with other disorders, particularly personality disorders. We cannot provide a full review of strategies for each possible form of comorbidity. We do note, however, the problem that many forms of addictive behavior can be comorbid with ASPD. This comorbidity is likely to cause difficulties in treatment, because, to date, researchers have not produced evidence for successful treatment of ASPD (Duggan, Mason, Banerjee, & Milton, 2007; Salekin, 2002). Individuals with ASPD tend not to understand the impact of their behaviors on others and they tend not to generalize lessons learned from one experience to new experiences, thus compromising treatment. A treatment focus on the addictive behavior and its negative impact on the client's goals, rather than a focus on ASPD symptoms, might prove most beneficial. There is a clear need for ongoing collaborations between clinicians and researchers to fashion the most effective, person-specific treatments for these difficult problems.

In this chapter, we have reviewed the literature on the comorbidity of PD and substance use, ED, and pathological gambling. We have discussed the more specific personality underpinnings of these addictive disorders and a common neurobiological model that may explain the similar addictive properties of these problem behaviors, including how they may become compulsive over time. We concluded by briefly discussing treatment implications and future directions for interventions given the personality underpinnings and comorbidity of PD.

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Exercise Addiction and Compulsive Exercising: Relationship to Eating Disorders, Substance Use Disorders, and Addictive Disorders

7

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Abstract

The recent addition of a behavioral addictions category in the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) has provided a framework to examine when behaviors progress from normal to pathologically excessive. Several behaviors that are either typically harmless (e.g., gambling, shopping) or even healthy (e.g., alcohol consumption, sex) when performed in moderation have been identified as having addictive potential. Exercise is one such behavior that may be viewed as an addiction. However, the literature is confused by several terms such as compulsion, addiction, and dependence that all may refer to similar, if not the same, pathological patterns of problematic exercise. This chapter will first review the various terms that are widely used and provide a clear distinction among exercise addiction, dependence, and compulsion. Next, we will review assessment measures that are commonly used in research and in clinical practice to screen for problematic exercise patterns. Finally, we will discuss the emerging literature that examines the co-occurrence of problematic exercise and eating pathology, substance use disorders, and other behavioral addictions.

Keywords

Behavioral addiction • Eating disorders • Exercise addiction • Exercise compulsion • Exercise dependence • Substance use disorders

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Behavioral addictions have recently become a topic of increasing interest in light of the addition of gambling disorder as the sole condition in a new category on behavioral addictions in the Diagnostic and Statistical Manual of Mental Disorders (DSM-5; American Psychological Association [APA], 2013). While the DSM-5 identifies only gambling as a behavioral addiction, other behaviors such as sex, shopping, and Internet gaming may have addictive potential. Exercise is another behavior that has been described as an addiction (Freimuth, Moniz, & Kim, 2011), compulsion (Meyer & Taranis, 2011), and dependency (Hausenblas & Symons Downs, 2002). The varied terms used to describe excessive exercise collectively indicate the pathological potential of physical activity. Although a great deal of recent research on pathological patterns of exercise exists, our understanding of problematic exercise is limited by the use of several terms that may be describing one singular phenomenon (Cook et al., 2013; Freimuth, 2009; Mónok et al., 2012).

Because exercise can serve as an effective method of weight control, and it is a common feature of eating disorders (ED) (Shroff et al., 2006), much of the excessive exercise literature has been conducted within the context of ED. The association among exercise and ED is so strong that some argue that problematic exercise does not exist independent of an ED (Mond, Hay, Rodgers, Owen, & Beumont, 2004). Nevertheless, problematic exercise has been observed clinically in the absence of ED (de Coverley Veale, 1987; Veale, 1995) and diagnostic guidelines for problematic exercise exclusive of ED have been proposed (Bamber, Cockerill, & Carroll, 2000, 2003). To come to such a conclusion requires clear operational definitions that describe and define pathological exercise and guide assessments. Therefore, this chapter will first review the large body of research examining all major nomenclatures of problematic exercise. Second, we will examine current tools available to quantify problematic exercise. We will conclude the chapter with a brief review of problematic exercise in conjunction with ED, substance use disorders (SUD), and other addictions. (For further information on the use of exercise in treatment, see Chap. 28.)

7.1 Distinguishing Exercise Addiction and Compulsion

Definitions for addiction and compulsion share much in common. Specifically, both addictions and compulsions are associated with adverse consequences across many domains (e.g., interpersonal, work), describe urge-driven repetitive behaviors that are difficult to control, and may also serve to manage affect. Thus, considerable overlap in features associated with exercise addiction and compulsion has contributed, in part, to the inconsistent use of terms describing pathological exercise. However, there is evidence that the scope of affect regulation, the developmental sequence, and presence of tolerance and withdrawal symptoms has the potential to distinguish exercise addiction from compulsion.

7.2 Addiction

The American Society of Addiction Medicine (ASAM, 2011; Smith, 2012) has recently updated their definition of addiction to reflect a more accurate description of both substance use *and* behaviors (Karim & Chaudhri, 2012) that have been conceptualized as addictions. Addiction is characterized by an inability to consistently **A**bstain, impaired **B**ehavioral control, **C**raving or increased hunger for drugs or rewarding experiences (i.e., behavioral addictions), **D**iminished recognition of significant problems with one's behaviors and interpersonal relationships, and a dysfunctional **E**motional response. Moreover, addictions have a genetic basis that leads to a primary, chronic disease of brain reward, motivation, memory, and related circuitry that manifests as a bio-psycho-social-spiritual dysfunction (ASAM, 2011). Simply stated, individuals with an addiction have a genetic predisposition that results in either substance use or behaviors producing hypersecretion of dopamine in the mesolimbic reward pathway (Smith, 2012). Consequently, repeated engagement in a behavior (e.g., drug use, exercise) becomes compulsive and without regard for the bio-psycho-social-spiritual consequences.

Exercise addiction research has begun to examine most aspects included in the ASAM definition of addiction. Much of this work has focused on examining addictive behaviors ability to manage affect. For example, exercise either reduces or alleviates a variety of negative affective states such as anger, depression, stress, and boredom (USDHHS, 2008). Where compulsive behavior is primarily a means to either escape or reduce a specific negative affect, anxiety, a distinctive feature of addiction is a dual potential to reduce negative affect while also creating positive feelings (Goodman, 2008). Therefore, addictive behaviors are intrinsically motivated through a substance or behavior's ability to increase positive affect and/or good feelings sometimes described as a high or buzz (e.g., runner's high, β-endorphin release; see Hamer & Karageorghis, 2007, for a review). A recent ecological momentary assessment study examined the relationship between daily activities and mood (i.e., valence, energetic arousal, and calmness) over a 10-week period and found that participants felt more content (valence), awake (energetic arousal), and calm (calmness) after being physically active compared to when they were inactive (Kanning & Schlicht, 2010). This study also found that the positive mood-exercise relationship was affected by the individual's baseline mood level, with the greatest effect seen when mood was depressed. This dual capacity of a behavior to escape negative affect while bringing relief, pleasure, or a high characterizes a potential addiction in its early stage of development. When a person discovers that a behavior provides a significant benefit (e.g., the behavior is a means to cope, improve self-esteem) that is not readily obtainable through other means, the behavior begins to increase in frequency.

With time, the addictive behavior can begin to have a compulsive urge-driven quality (not to be confused with a compulsive disorder). That is, as the behavior increases in frequency, the individual continues to engage in the behavior despite it creating havoc in the person's life. For example, the addicted runner may put this activity above work and family. Ceasing or cutting back on this routinized

repetitive behavior is not an option because to do so is associated with cravings and dysphoria. The addictive behavior is then "compulsive" in the sense that it is difficult to control, and the primary motivation is escape from distress (Freimuth et al., 2011). At this point, the addicted exerciser may miss out on family events or work deadlines because taking a break from the gym means experiencing the discomfort of withdrawal.

Tolerance and withdrawal are also often included in definitions that are unique to addiction. The presence of each may offer another way to distinguish addiction from compulsion. Specifically, tolerance refers to the need to do more of a behavior to get the desired effect. A person who once needed four beers to get "high" now consumes six beers or what began as a 1 h workout three times a week has extended into daily workouts of no less than 2 h to achieve the desired sense of accomplishment. Ceasing any addictive behavior is also associated with withdrawal syndrome. In the case of exercise, withdrawal is associated with a sense of being uneasy, irritability, sluggishness, sleep disturbances, decreased sexual drive, and increased negative affect (Aidman & Woollard, 2003; Berczik et al., 2012).

Finally, exercise addiction has also been presented as a form of dependence. In general, dependence occurs in response to taking a substance that affects the central nervous system and represents a normal physiological adaptation to repeating a dose of a substance. Functionally, dependence and addiction refer to the same pattern of behavior. Moreover, some types of substance use (e.g., prescription drug use, nonproblematic alcohol use) may show tolerance and/or mild withdrawal symptoms but would not reflect the compulsion nor the severity of a disorder necessary to satisfy a diagnosis of addiction. Therefore, the use of dependence as the preferred nomenclature over addiction as a diagnostic term originated in the DSM-III-R with the intention of avoiding a potentially stigmatizing and pejorative term such as addiction and to better describe a wide range of problematic substance use (O'Brien, Volkow, & Li, 2006). Because patterns of exercise display many similarities with SUD (i.e., tolerance, withdrawal symptoms, ability to be performed by most individuals without progressing to addiction), a compelling argument has been made that exercise dependence is the preferred term. The basis for this nomenclature, in large part, was to more closely reflect the diagnostic criteria in the DSM-IV (Hausenblas & Symons Downs, 2002). The DSM-5 criteria are similar, although the term "dependence" has been dropped in favor of "use disorders," and "craving" has been added to the list of potential symptoms of SUD (American Psychiatric Association, 2013).

7.3 Compulsion

Compulsive has also been used as a descriptive term to indicate problematic exercise. Compulsivity is defined as an urge to perform a behavior to relieve anxiety arising from the perception of a negative consequence if the behavior is not performed (American Psychiatric Association, 2000; Meyer, Taranis, Goodwin, & Haycraft, 2011). Examples of compulsive disorders include ritualized and

stereotyped behaviors such as frequent checking or hand washing (de Coverley Veale, 1987). For example, an individual with obsessive–compulsive disorder who, after leaving home, worries that the stove was left on will be consumed by thoughts that there will be an explosion until he returns home to check. Thus, compulsions are a response to an urge to take action with the intent of escaping unpleasant affect, usually anxiety. This anxiety arises from imagined negative consequences (i.e., obsessions) if the action is not taken. As we have previously described, patterns of exercise in an addicted individual may begin to exhibit compulsive tendencies. However, one key distinction of compulsion from addiction is that compulsions do not show tolerance. That is, a given behavior, be it hand washing or checking, alleviates anxiety until the next time the urge arises, but the individual does not need to wash more or check more often to quell a given urge.

Compulsive behavior is also associated with obsessive thinking. For example, the compulsive exerciser is consumed with thoughts of adverse events if exercise does not occur. While both the addicted and compulsive exercisers obsess about the adverse effects of forgoing the behavior, the addicted person's obsessions are focused more on realistic negative consequences of doing the behavior, while those of the compulsive are based on unrealistic consequences arising from not doing the behavior (Yates, 1991).

Review of both the exercise addiction and compulsion literatures reveals major overlap among each nomenclature as the preferred term to describe problematic exercise patterns. This may be simplistically summarized as a pattern of exercise that is deeply engrained in the individual's daily rituals, increased time spent in exercise behavior and/or exercise-related activities (e.g., planning for future episodes, recovering from previous episodes, shopping for equipment), and continuing to exercise despite serious consequences such as injury and/or weight-related health detriments (e.g., ED). Moreover, this pattern of exercise is performed with a zeal that is beyond the scope of a "normal" exerciser and is resistant to change. Notable differences in these literatures are that the addiction literature provides a more dynamic and inclusive framework for defining exercise as a behavioral addiction and has presented various theories on the etiology and biopsychosocial implications of exercise as an addiction. Alternatively, descriptions of exercise as a compulsion offer better evidence of exercise's role in affect regulation and in ED. A further key distinction among addiction and compulsion terminology is that compulsive behaviors do not show the same tolerance or withdrawal symptoms that are hallmarks of addiction.

Case studies (de Coverley Veale, 1987) and correlational research (Cook et al., 2013) support the importance of distinguishing forms of problematic exercise. Primary exercise addiction is defined as continually exercising solely for the psychological gratification resulting from the exercise behavior (de Coverley Veale, 1987). Secondary exercise addiction occurs when an individual uses exercise to accomplish some other end in conjunction with other pathologies, specifically ED (Hausenblas & Symons Downs, 2002). While compulsion may exist in primary exercise addiction (Freimuth et al., 2011), there is clear evidence that compulsion is the key factor of secondary addiction that may distinguish beneficial

from detrimental exercise in ED (Cook & Hausenblas, 2011; Cook, Hausenblas, Tuccitto, & Giacobbi, 2011; Hausenblas, Cook, & Chittester, 2008; Meyer & Taranis, 2011). Thus, addiction terminology represents all forms of problematic exercise, while compulsive terminology is a preferable term for secondary exercise addiction (Meyer & Taranis, 2011). Simply stated, all problematic patterns of exercise may be described as an addiction. However, addictions include compulsive urge-driven qualities, and compulsions are the key facet that may explain the relationship among exercise and ED. Therefore, compulsive exercise is the preferred terminology for what has been referred to as secondary exercise addiction.

Given the current state of the literature where exercise addiction and compulsion are often not distinguished and to avoid further confusion, we will use the term *problematic exercise* throughout the remainder of this chapter unless we are referring to a study that clearly makes the distinction or measurement tool.

7.4 Measuring Exercise Addiction and Compulsion

The various conceptualizations and definitions previously described have generated several measurement tools that quantify the various forms of problematic exercise. Each tool has its own advantages and disadvantages in measuring facets of addiction or compulsion that are pertinent to either general or specific populations (e.g., eating-disordered individuals, body builders, runners). Additionally, it is important to note that most measures contain items that continue to confuse conceptualizations of problematic exercise (e.g., compulsion scales that contain items assessing tolerance or withdrawal). We have focused our review to reflect the tools that are most commonly used, demonstrate superior psychometrics, and present an approach to quantifying problematic exercise that may be broadly applicable to multiple fields of research. Therefore, we have included the *Exercise Addiction Inventory* (EAI) and *Exercise Dependence Scale* (EDS) because they are the two most widely used measures of exercise addiction (Mónok et al., 2012). Similarly, we have included the *Compulsive Exercise Test* (CET) and *Obligatory Exercise Questionnaire* (OEQ) because they are two popular scales of exercise compulsion that have been used extensively in research examining exercise in ED. Examples of specific items for each facet of all measurements are provided in parenthesis.

7.5 Exercise Addiction Inventory

The EAI (Terry, Szabo, & Griffiths, 2004) is a quick and simple screening tool measure that is based on theoretical constructs of behavioral addiction (Griffiths, 1996). The EAI includes one item for each of the following six components of behavioral addiction:

- Salience (“Exercise is the most important thing in my life”)

- Mood modification ("I use exercise as a way of changing my mood (e.g., to get a buzz, to escape, etc.)")
- Tolerance ("Over time I have increased the amount of exercise I do in a day")
- Withdrawal ("If I have to miss an exercise session, I feel moody and irritable")
- Conflict ("Conflicts have arisen between me and my family and or my partner about the amount of exercise I do")
- Relapse ("If I cut down on the amount of exercise I do, and then start again, I always end up exercising as often as I did before")

Responses to the items are on a 5-point Likert scale ranging from 1 (strongly disagree) to 5 (strongly agree). Item scores are summed, and higher total scores reflect problematic exercise. The EAI can also be used categorically. Total scores of 0–12 are indicative of an "asymptomatic individual," 13–23 are indicative of a "symptomatic individual," and scores above 24 are indicative of an "at risk of exercise addiction individual." A cutoff score of 24 reflects the top 15 % of responses (Terry et al., 2004).

7.6 Exercise Dependence Scale

The EDS (Hausenblas & Symons Downs, 2002) is a 21-item measure of exercise dependence symptoms that was developed to reflect the criteria for substance dependence listed in the DSM-IV (American Psychiatric Association, 2000). The EDS includes seven subscales consisting of three items each that assess:

- Tolerance ("I continually increase my exercise frequency to achieve the desired effects/benefits")
- Withdrawal effects ("I exercise to avoid feeling tense")
- Continuance ("I exercise despite persistent physical problems")
- Lack of control ("I am unable to reduce how intense I exercise")
- Reductions in other activities ("I think about exercise when I should be concentrating on school/work")
- Time ("I spend a lot of time exercising")
- Intention ("I exercise longer than I expect")

Responses to the items are on a 6-point Likert scale ranging from 1 (never) to 6 (always). A lower score reveals less exercise dependence symptoms. Responses can be summed for a total continuous score of exercise dependence symptoms. Additionally, the scale can be used categorically. Individuals endorsing scores of five to six on items for at least three subscales are categorized as "at risk for exercise dependence," scores of three to four on at least three subscales are categorized as "nondependent symptomatic," and scores of one to two are categorized as "nondependent asymptomatic." The psychometric properties of this scale are good (Symons Downs, Hausenblas, & Nigg, 2004). This scale has not yet been updated to reflect the recent addition of cravings in the SUD criteria in DSM-5.

7.7 Compulsive Exercise Test

The CET (Taranis, Touyz, & Meyer, 2011) is a 24-item measure that assesses compulsive features of exercise that are implicated in the maintenance of excessive exercise in ED. The CET includes the following five subscales:

- Avoidance and rule-driven behavior ("I usually continue to exercise despite injury")
- Weight control exercise ("I exercise to burn calories and lose weight")
- Mood improvement ("I feel less anxious after I exercise")
- Lack of exercise enjoyment ("I find exercise a chore")
- Exercise rigidity ("My weekly pattern of exercise is repetitive")

Responses to the items are on a 6-point Likert scale ranging from 0 (never true) to 5 (always true). An item on the "weight control exercise" subscale is reverse scored (e.g., "I do not exercise to be slim") as is an item on the "lack of exercise enjoyment" subscale (e.g., "I enjoy exercising"). All items may then be summed to provide a total score. Items included in each subscale may also be summed. The CET has demonstrated good psychometric properties (Taranis et al., 2011).

7.8 Obligatory Exercise Questionnaire

The OEQ (Thompson & Pasman, 1991) is a 20-item scale that assesses how often individuals experience various emotions, attitudes, and behaviors related to exercise and compulsion to continue exercising. The OEQ has three distinct dimensions (Ackard, Brehm, & Steffen, 2002) that assess:

- Exercise fixation ("When I miss a scheduled exercise session, I may feel tense, irritable, or depressed")
- Exercise frequency ("I engage in physical exercise on a daily basis")
- Exercise commitment ("When I miss an exercise session, I feel concerned about my body possibly getting out of shape")

Responses are indicated on a 4-point Likert scale ranging from "never" to "always." The psychometric properties of this scale are good (Thompson & Pasman, 1991). The OEQ has been used extensively in research examining the association among exercise compulsion and ED.

7.9 Prevalence of Problematic Exercise

Prevalence estimates for problematic exercise are limited by the following three reasons. First, measures that have been developed to quantify problematic exercise are largely driven by continuous assessments of symptom severity rather than a theoretical approach (Terry et al., 2004). Second, most problematic exercise research has reported symptom severity scores rather than prevalence rates of a fully developed condition (Sussman, Lisha, & Griffiths, 2011). Third, because problematic exercise prevalence is higher in eating-disordered individuals (Shroff

Table 7.1 Prevalence of exercise pathology

Scale type	Authors	Year	Exercise measure	Prevalence (%)	Sample	*N*
Addiction	Griffiths et al.	2005	Exercise Addiction Inventory	3.0	College students	279
Addiction	Mónok et al.	2012	Exercise Addiction Inventory	3.2	Hungarian nationwide—regular exercisers	474
Addiction	Mónok et al.	2012	Exercise Addiction Inventory	0.5	Hungarian nationwide—point prevalence	2,710
Addiction	Szabo and Griffiths	2007	Exercise Addiction Inventory	6.9	College students	355
Addiction	Villella et al.	2011	Exercise Addiction Inventory	8.5	Italian high school students	2,853
Addiction	Modolo et al.	2011	Negative Addictions Scale	33.2	Amateur athletes	300
Compulsion	Garman et al.	2004	Commitment to Exercise Scale	21.8	College students	268
Compulsion	Mond et al.	2006	Commitment to Exercise Scale	16.5	Australian sample of female exercisers	3,472
Compulsion	Mond et al.	2008	Commitment to Exercise Scale	22.6	Adult women primary care patients	257
Compulsion	Guidi et al.	2009	Consumptive Habits Questionnaire	18.1	College students	589
Compulsion	Dalle Grave et al.	2008	Eating Disorder Examination	45.5	Eating disorder patients	165
Compulsion	Ackard et al.	2002	Obligatory Exercise Questionnaire	8.9	College students—female only	586
Compulsion	Slay et al.	1998	Obligatory Exercise Questionnaire	25.9	Runners in a 4-mile recreational road race	324
Compulsion	Schroff et al.	2006	Structured Interview for Anorexic and Bulimic Disorders	38.8	Eating disorder patients	1,857
Dependence	Lejoyeaux et al.	2008	Author-developed questionnaire	42.0	French gym users	300
Dependence	Lejoyeaux et al.	2012	Author-developed questionnaire	30.0	French gym users	500

(continued)

Table 7.1 (continued)

Scale type	Authors	Year	Exercise measure	Prevalence (%)	Sample	*N*
Dependence	Bamber et al.	2000	Exercise Dependence Questionnaire	22.8	College students, fitness classes, runners, and eating disorder patients	291
Dependence	Blaydon and Lindner	2002	Exercise Dependence Questionnaire	52.0	Triathletes	171
Dependence	Grandi et al.	2011	Exercise Dependence Questionnaire	36.4	Fitness club users and regular exercisers	79
Dependence	Weik and Hale	2009	Exercise Dependence Questionnaire	24.9	Adult exercisers	204
Dependence	Zmijewski and Howard	2003	Exercise Dependence Questionnaire	45.9	College students	237
Dependence	Allegre et al.	2007	Exercise Dependence Scale	3.2	Ultramarathoners	95
Dependence	Cook and Hausenblas	2011	Exercise Dependence Scale	1.9	College students—females only	387
Dependence	Cook et al.	2013	Exercise Dependence Scale	1.4	Community sample of runners	2,660
Dependence	Cook et al.	2011	Exercise Dependence Scale	2.7	College students	539
Dependence	Hausenblas and Symons Downs	2002 (Study 1)	Exercise Dependence Scale	3.4	College students	266
		(Study 2)	Exercise Dependence Scale	13.4	College students	553
		(Study 3)	Exercise Dependence Scale	3.1	College students	862
		(Study 4)	Exercise Dependence Scale	9.6	College students	366
		(Study 5)	Exercise Dependence Scale	9.8	College students	419
Dependence	Mónok et al.	2012	Exercise Dependence Scale	1.9	Hungarian nationwide—regular exercisers	474

(continued)

Table 7.1 (continued)

Scale type	Authors	Year	Exercise measure	Prevalence (%)	Sample	*N*
Dependence	Mónok et al.	2012	Exercise Dependence Scale	0.3	Hungarian nationwide—point prevalence	2,710
Dependence	Symons Downs et al.	2004	Exercise Dependence Scale	5.0	College students	1,263
Dependence	Weik and Hale	2009	Exercise Dependence Scale	11.9	Adult exercisers	204

et al., 2006) and athletes (Allegre, Therme, & Griffiths, 2007; Blaydon & Lindner, 2002; Modolo et al., 2011), prevalence estimates have been biased by sample characteristics (Cook et al., 2013; Mónok et al., 2012). We have summarized the major studies that reported exercise addiction, compulsion, and dependence prevalence in Table 7.1.

7.10 Problematic Exercise and Eating Disorders, Substance Use Disorders, and Other Addictive Disorders

Individuals who display some form of problematic exercise typically exhibit personality characteristics and psychological distress such as perfectionism, inhibition of anger, high self-expectations, tolerance of physical discomfort, denial of severity, depression, and anxiety that are similar to features of anorexia nervosa. These similarities initially suggested that increased exercise amount is associated with ED and may explain the increased prevalence of ED in athletes (Sundgot-Borgen & Klungland Torstveit, 2004).

Animal research has provided some support for the ED and problematic exercise association. That is, the activity-based anorexia model is based on the observation that when rats are restricted from feeding, but have unhindered access to a running wheel, they engage in increased amounts of physical activity (Epling & Pierce, 1996). These rats subsequently voluntarily restrict feeding when food is available and continue to increase physical activity amount that, if not intervened upon, results in death. Similar to humans, athletic-based anorexic rats self-restricted food intake and increased physical activity more rapidly and to a more severe extent than rats of the same litter without athletic-based anorexia (Boakes, Mills, & Single, 1999). Animal research has the advantage of being able to more closely examine possible mechanistic pathways in the brain that may be responsible for this effect. Specifically, the association between hypothalamic–pituitary–adrenal (HPA) axis activity and food restriction increased in physical activity, and this relationship is mediated through body fat levels (Adan et al., 2011; Duclos, Bouchet, Vettier, & Richard, 2005; Hamer & Karageorghis, 2007). However,

research examining athletic-based anorexia is difficult to replicate in humans, and explanations of this effect typically include some recognition of culture (Epling & Pierce, 1996).

Current research on exercise and eating behaviors suggests that within ED populations exercise is more like a compulsion than an addiction (Meyer & Taranis, 2011). That is, an individual with an ED may be consumed by an unrealistic worry that an immediate unpleasant change in appearance will occur if an exercise session is missed and that exercise behavior may serve as a means to escape the unpleasant affect associated with an ED. This aspect of affect regulation has supported the role of exercise as a compensatory behavior as part of an ED (American Psychiatric Association, 2013; Meyer et al., 2011).

Affect regulation is a key aspect of understanding ED and has been implicated as a central construct in the development and maintenance of these disorders (Haynos & Fruzzetti, 2011). With regard to exercise in ED, compulsive exercise in ED patients has been associated with elevated levels of negative affect (Brewerton, Stellefson, Hibbs, Hodges, & Cochrane, 1995; Vansteelandt, Rijmen, Pieters, Probst, & Vanderlinden, 2007), anxiety (Brewerton et al., 1995; Klein, Mayer, Schebendach, & Walsh, 2007), and depression (Peñas-Lledó, Leal, & Waller, 2002). This research points to problematic exercise in ED populations to be more like a compulsion than an addiction (Meyer & Taranis, 2011) and that exercise may be used to cope with these kinds of adverse emotional states (Fairburn, Cooper, & Shafran, 2003). Also like a compulsion, a person with an ED is consumed by the unrealistic worry that an immediate unpleasant change in appearance will occur if an exercise session is missed. As such, exercise behavior may serve as a means to escape unpleasant affect. This aspect of affect regulation has supported the role of exercise as a compensatory behavior as part of an ED (American Psychiatric Association, 2013; Meyer et al., 2011).

In two recent ecological momentary analysis studies, LePage, Price, O'Neil, and Crowther (2012) found that compulsive exercise is related to lower positive affect on nonexercise days than exercise days and negative affect was stronger on exercise days. Furthermore, increased compulsive exercise was associated with more thoughts about eating on nonexercise days in individuals with scores above 13 on the Eating Attitudes Test-26. Thus, the ability for exercise to provide an escape from unpleasant internal cues such as negative affect supports the social-cognitive/cognitive-behavioral view of compulsive exercise as a symptom, but not an antecedent, of an ED (Fairburn et al., 2003).

The association of exercise with ED fails to account for the well-established physiological and psychological benefits of exercise. That is, several narrative reviews and meta-analyses have shown that in the absence of an ED, exercise imparts positive improvements in factors associated with ED such as anxiety, depression, stress reactivity, and self-esteem (USDHHS, 2008). Similarly, the physiological benefits conveyed by exercise may also help to counteract the negative consequences of ED. For example, cardiovascular benefits such as increased cardiac mass, increased stroke volume and cardiac output at rest and during exercise, lower resting heart rate and blood pressure, and a decreased

tendency for blood clotting are pertinent to ED (Pearson, Goldklang, & Streigel-Moore, 2002). Exercise also has the ability to reduce adiposity, thus, contributing to a leaner and fit body type (Thompson, Heinberg, Altabe, & Tantleff-Dunn, 1999). The metabolic benefits of exercise, beyond contributing to decreased adiposity, include decreased triglycerides and increased high-density cholesterol, increased insulin-mediated glucose uptake, and possible increase in resting metabolism (Haskell, 1994). Finally, exercise increases skeletal muscle mass and bone density in youth and is related to the retention of bone mineral density in older adults. This has implications in the development of osteoporosis, a common consequence of prolonged ED behaviors (Klump, Bulik, Kaye, & Treasure, 2009). Thus, examining pathological motivations for exercise (e.g., compulsions) is a key factor in understanding the relationship among exercise amount and ED (Adkins & Keel, 2005).

Hausenblas et al. (2008) presented a model that may reconcile why problematic exercise is associated with ED, while nonproblematic exercise is associated with several factors that may protect against ED. This model posits that exercise may impart mental health benefits (e.g., reduced anxiety and depression) which serve as protective factors in the development of eating pathology. However, when cognitions and motivations for exercise become pathological (i.e., exercise dependence), exercise may exacerbate eating pathology, thus supporting clinical observations and animal model research.

Recent research has begun to support this model. In a series of studies, Cook and colleagues found that regular exercise positively affects psychological well-being, which in turn reduces ED risk. In each study, problematic exercise, defined as exercise dependence, negated this relationship and mediated the exercise and ED relationship (Cook & Hausenblas, 2008, 2011; Cook et al., 2011). Moreover, exercise dependence may exacerbate the detrimental impact of an ED on health-related quality of life (Mond, Myers, Crosby, Hay, & Mitchell, 2008). Further research is needed to prospectively examine these associations that have received initial support and continue to examine physiological effects specified in the original model (Hausenblas et al., 2008).

While most of the research investigating problematic exercise and co-occurring disorders has focused on ED, researchers have recently begun to examine the association with other addictions. Initial studies suggest that other addictions, particularly behavioral addictions, are quite common in individuals with problematic exercise. This may superficially suggest common neurological pathways in addictive disorders (Lynch, Peterson, Sanchez, Abel, & Smith, 2013). Sussman and colleagues' (2011) review of the literature revealed that 15 % of exercise-addicted individuals are also addicted to smoking, alcohol, or illicit drugs and that an additional 25 % of exercise addicts exhibit behavioral addictions such as gambling, Internet, love, sex, work, and/or shopping addictions. Other studies have reported higher prevalence rates of co-occurring disorders. For example, in a study of patrons of a Parisian gym, prevalence of compulsive buying (63 % vs. 38 %), bulimia nervosa (70 % vs. 47 %), and hypochondria (28 % vs. 20 %) was higher in the exercise dependence group than the nonexercise dependence group (Lejoyeux, Avril, Richoux, Embouazza, & Nivoli, 2008). However, these elevated rates may

have been due to poor measurement and overly broad operational definitions. Finally, in a study of individuals described as having a sexual addiction (Carnes, Murray, & Charpentier, 2005), 11.4 % of the men and 13.2 % of the women were identified as also having "addictive athleticism" (presumably problematic exercise).

Further research is needed to examine the etiology and relationships among exercise and other addictions. For example, several recent reviews have concluded that moderate amounts of exercise may be an effective therapy or adjunct to therapy for both ED (Hausenblas et al., 2008; Ng, Ng, & Wong, 2013; Wolff et al., 2011; Zschuckle, Gaudlitz, & Ströhle, 2013; Zunker, Mitchell, & Wonderlich, 2011) and other addictions (Fontes-Ribeiro, Marques, Pereira, Siva, & Macedo, 2011; Lynch et al., 2013). Moreover, exercise is also a widely recommended and accepted behavior that may foster a norm of inclusion to the larger healthy community as opposed to the stigmatization of being labeled with an addiction. Thus, it is not yet clear whether problematic exercise occurs with other forms of addiction or if the individual is compulsively exercising to self-treat the original addiction.

7.11 Summary

For over 40 years researchers have described problematic patterns of exercise (Baekland, 1970). A major goal of our chapter was to provide definitional clarity of exercise dependence, addiction, and compulsion that can be used to guide the next generation of research in this area. Addiction terminology represents all forms of problematic exercise, while compulsive terminology is a preferable term for problematic exercise that is secondary to an ED. Definitional clarity, both in nomenclature and in measurement tools that attempt to quantify aspects of addiction or compulsion, is needed as we move forward examining the antecedents and consequences of excessive exercise.

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Nutritional Aspects of Eating Disorders, Addictions, and Substance Use Disorders

8

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Abstract

Substance use disorders and eating disorders can lead to unhealthy eating and malnutrition that is associated with varying degrees of medical morbidity and increased risk of death. Vitamin and mineral deficiencies are most common in patients with anorexia nervosa, bulimia nervosa, and alcohol use disorders but can be seen in other substance use disorders as well. These disorders may share some underlying pathophysiology and can co-occur, which further increases the risk of malnutrition. The specific nutritional deficits in eating disorders and substance use disorders, their clinical (especially neurological) manifestations, how commonly they occur, and their mechanisms of action will be discussed in this chapter.

Keywords

Eating disorders • Substance use disorders • Nutritional deficiency • Alcohol use disorders • Anorexia nervosa • Binge eating disorder • Bulimia nervosa

8.1 Introduction

The importance of nutrition for healthy living was discussed as early as the fifth century when Hippocrates suggested that many health problems could be prevented or alleviated with healthy diet (and exercise) (Simopoulos, 2001). Nutrition refers

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to the supply of foods or supplements for the growth and maintenance of a living organism. There are six major classes of nutrients. These include carbohydrates, proteins, fats, water, vitamins, and minerals. Over the centuries, specific nutritional deficiencies have been shown to carry deleterious health effects. One of the earliest discoveries was made by Dr. James Lind who in 1747 treated scurvy among British sailors and seafarers with lime juice. In the early 1930s it was found that scurvy is a disease due to deficit of the essential nutrient vitamin C. Similarly, Japanese sailors whose diets consisted primarily of white rice developed beriberi (a constellation of neurological and cardiac deficits) from which they often died. This condition was later found to be due to thiamine deficiency. Since then, numerous medical disorders have been linked to malnutrition or specific nutrient deficiencies. Many governments throughout the world have set forth specific guidelines for food consumption and daily requirements of vitamins and minerals.

There are a number of psychiatric conditions that predispose people to develop malnutrition, most commonly addiction to substances substance use disorders (SUD) or disordered eating from eating disorders (ED) anorexia nervosa (AN), bulimia nervosa (BN), binge eating disorder (BED). Malnutrition can be seen in other psychiatric conditions such as schizophrenia when delusions are related to food intake (McCormick, Buchanan, Onwuameze, Pierson, & Paradiso, 2011) or in children with attention-deficit hyperactivity disorder when appetite is decreased from stimulant use (Davis et al., 2007; Schachter, King, Langford, & Moher, 2001). This chapter will primarily focus on the mechanisms of malnutrition and their sequelae occurring among individuals suffering with SUD and ED and especially when these two conditions co-occur.

8.2 Mechanisms and Clinical Manifestations of Malnutrition in SUD

The first descriptions of addiction to substances were found in Sumerian writings as early as 6,000 years BC (Davenport-Hines, 2003; Krikorian, 1975). SUD have become known today as a cluster of cognitive, behavioral, and psychological symptoms indicating that an individual continues using substances despite substance-related problems (American Psychiatric Association, 2013). The most recent version of the Diagnostic and Statistical Manual of Mental Illness (DSM-5) considers what was formerly indicated as substance abuse and dependence as a continuum of disorders involving the use of substances from mild to severe, according to the number of endorsed symptoms (American Psychiatric Association, 2013). The threshold for diagnosing mild substance use disorders is set at two symptoms (or criteria) from a list of 11. Craving is a symptom not present in the previous version of the Manual (i.e., a strong desire to use a substance). Problems with law enforcement are eliminated due to cultural considerations.

Malnutrition and nutritional deficiencies are common among people with SUD (Abd El Gawad, Hassan, Ghanem, Awad, & Ali, 2011; Gueguen et al., 2003; Islam,

Hossain, Ahmed, & Ahsan, 2002; Saeland et al., 2008). SUD impact nutrition and diet in at least two ways (1) through reduced food intake and by the deleterious effects of the used substances on the body. It is plausible that combination effects may be multiplicative rather than simply additive. Perhaps because of the alteration in reward and impulse control pathways, people who suffer with SUD show poor decision-making in nutritional choices that can create a variety of deficiencies and/or excesses of certain nutrients. Diminished and/or poor nutritional choices may result in noxious effects on the body irrespective of the substance abused. With poor nutrition, the most common nutrients impacted are the vitamins and rare minerals that are supplied from fresh vegetables and fruits including vitamin A (e.g., carotene), B vitamins (thiamine, niacin, pantothenic acid, and biotin), vitamin C, vitamin E, folic acid, copper, potassium, manganese, magnesium, molybdenum, and selenium. Some drugs including cocaine, marijuana, amphetamine, and heroin may impact food intake by reducing appetite (Anglin, Burke, Perrochet, Stamper, & Dawud-Noursi, 2000; Mohs, Watson, & Leonard-Green, 1990). Some substances of abuse have been shown to be directly neurotoxic such as ecstasy, inhalants, hallucinogens, and methamphetamine (Ares-Santos, Granado, & Moratalla, 2013; Cairney et al., 2013; Capela et al., 2013; Cowan, Roberts, & Joers, 2008; Marona-Lewicka, Nichols, & Nichols, 2011; Takagi, Lubman, & Yucel, 2011). The specific nutritional impact of some of the more commonly used substances will be discussed in more detail below.

A nationwide study in 2008 showed that alcohol is the most commonly abused substance in the USA, followed by cannabis and methamphetamine abuse (Substance Abuse and Mental Health Services Administration, 2011). Excess use of this substance is known to cause deficiency of several vitamins and minerals through impaired absorption, metabolism, and utilization (Lieber, 2003; McClain, Barve, Barve, & Marsano, 2011; Strohle, Wolters, & Hahn, 2012). Alcohol can exert direct effects on metabolism or effects due to the progression of medical conditions associated with alcohol overuse (e.g., hepatitis and dilated cardiomyopathy). Impaired absorption is in the early stages secondary to the effects of alcohol to the gastric mucosa. In later stages of the illness, poor absorption develops due to gastritis secondary to cirrhosis and the relative venous stasis in the portal splanchnic district. Metabolism is generally altered in various degrees according to the severity of accompanying hepatic pathology. Vitamin deficiencies in alcohol use disorders (AUD) include vitamin A, vitamins of the B family (thiamine, riboflavin, niacin, pantothenic acid, pyridoxine, and cobalamin), folic acid, vitamin C, vitamin D, and vitamin E (Clugston & Blaner, 2012; Strohle et al., 2012). The mineral deficiencies associated with AUD include calcium, magnesium, and zinc (Devgun, Fiabane, Paterson, Zarembski, & Guthrie, 1981). These deficiencies are thought to play a role in some forms of dementia and depression as well as poor appetite (Coppen, 2005; Glick, 1990; Ramsey & Muskin, 2013; Rasmussen, Mortensen, & Jensen, 1989; Tiemeier et al., 2002). Men with AUD have been shown to have low levels of vitamin D and are at an increased risk of osteoporosis due to direct and indirect effects on bone remodeling and formation, which leads to increased risk for fractures (González-Reimers et al., 2011). Similarly, men with AUD may be at

risk for bleeding problems secondary to vitamin K deficiency by disturbed absorption through the liver, which is often impaired from heavy alcohol use (Iber, Shamszad, Miller, & Jacob, 1986).

The most serious and life-threatening deficiency that occurs in AUD is a deficiency of B vitamins including thiamine and folic acid. These deficiencies are known to be associated with dementia (Goebels & Soyka, 2000). Wernicke's encephalopathy and Korsakoff's syndrome are severe life-threatening conditions that can occur in a thiamine-deficient state induced by poor nutrition often caused by severe alcoholism (Lough, 2012). There are times in which the lack of one B vitamin impairs the utilization of another. For example, folic acid cannot be utilized in the body in the absence of B12 and when deficient folic acid can mask vitamin B12 deficiency as well.

The impact of addiction to tetrahydrocannabinol (THC) in cannabis on nutrients has not been studied to the same extent as alcohol. Results from the third national health and nutrition examination survey showed higher cigarette-smoking rates and higher consumption of sodas and alcohol, including beer, among current cannabis users than among non-current cannabis users (Smit & Crespo, 2001). Cannabis users also consumed more sodium, fewer fruits, and more pork, cheese, and salty snacks, which resulted in lower serum carotenoid levels (i.e., vitamin A deficiency) (Smit & Crespo, 2001). Vitamin A deficiency can result in night blindness (complete blindness in severe deficiency), impaired immunity to infections, predisposition to cancer, and even birth defects. While cannabis can increase appetite in some people, several studies and two national surveys have found that active cannabis users (using at least three times a week) actually have a lower body mass index (BMI) than persons who do not use cannabis (Le Strat & Le Foll, 2011; Penner, Buettner, & Mittleman, 2013; Rodondi, Pletcher, Liu, Hulley, & Sidney, 2006). The studies revealed that smoking cannabis was also associated with a smaller waist circumference and lower insulin levels than those who do not use cannabis (Penner et al., 2013; Rodondi et al., 2006).

Illicit stimulant substances such as cocaine and amphetamine (methamphetamine) have been shown to decrease appetite when using and when withdrawing from substance (Walsh, Donny, Nuzzo, Umbricht, & Bigelow, 2010). Calorie and protein malnutrition has been found in people who use cocaine chronically (Santolaria-Fernández et al., 1995). Rodent models of cocaine addiction have also found that to cocaine continued use of the substance and neglect food (Sanchis-Segura, & Spanagel, 2006). Cocaine and methamphetamine have been found to be especially neurotoxic to dopaminergic neurons in the basal ganglia and hippocampus in animal models (Aksenov et al., 2006; Ares-Santos et al., 2013; Capela et al., 2013; Olsen, 1995) and have been linked to myocardial infarctions in humans (Galasko, 1997; Isner et al., 1986; Lange & Hillis, 2001). More data is needed to fully understand the specific nutritional deficiencies that occur with addiction to stimulant substances, but there appears to be at least indirect effects of poor appetite causing reduced consumption of nutritious food.

Poor appetite leading to poor intake of nutrients is also the likely cause of malnutrition in people primarily addicted to opiates (Meleger, Froude, & Walker, 2013; Morabia et al., 1989; Neale, Nettleton, Pickering, & Fischer, 2012). In a study of 149 people addicted to heroin 25 % were found to have hypovitaminemia, primarily of B vitamins, which are water soluble and are most vulnerable during poor intake of regular food (el-Nakah, Frank, Louria, Quinones, & Baker, 1979). Hyperkalemia (or increased blood potassium) has been reported in heroin addiction, while calcium deficiency has been reported in morphine abusers (Mohs et al., 1990). Hyperkalemia can lead to deadly arrhythmias, while calcium deficiency can cause altered bone metabolism and reduced trabecular bone mass resulting in osteoporosis (Katz & Norman, 2009; Roberts, Finch, Pullan, Bhagat, & Price, 2002).

In summary, common nutritional deficiencies associated with SUD occur early in the addiction process and continue as the illness progresses, poor consumption of a balanced diet is but one mechanism of malnutrition. There is evidence that the acute intoxicating effects and long-term craving of particular substances also impair insight and decision-making leading to poor self-care and malnutrition. There is also an association between chronic substance addiction and unemployment as well as homelessness (Johnson, Freels, Parsons, & Vangeest, 1997), which may contribute to a lack of access to nutritious food (Beharry, 2012; Rehm et al., 2009). Studies suggest that having a SUD increases the chances of being addicted to more than one substance (Aggrawal, 2001; Perkonigg, Lieb, & Wittchen, 1998), which may theoretically increase the chances of malnutrition. More studies are needed to fully understand all the deleterious effects of SUD on the body and brain to improve strategies for treatment and prevention.

8.3 Mechanisms and Clinical Manifestations of Malnutrition in ED

Disordered eating that leads to clinical or subclinical malnutrition of macronutrients (e.g., carbohydrates, proteins, fats) and/or micronutrients (e.g., vitamins, minerals, trace elements) is often present to varying degrees in people suffering from AN and can be seen in some patients with BN (Casper, Kirschner, Sandstead, Jacob, & Davis, 1980; Rock & Vasantharajan, 1995; Setnick, 2010; Winston, 2012). Typical nutritional problems that occur with BED include excessive intake of highly caloric foods that can lead to significant weight gain but will not be discussed in detail here (Raymond, Bartholome, Lee, Peterson, & Raatz, 2007; Schneider, 2003). There are a number of medical consequences of vitamin and mineral deficiencies as well as severe electrolyte disturbances that have been described in patients with AN and BN, which can occur in SUD as well, particularly AUD (see Table 8.1). The most life-threatening electrolyte abnormality is hypokalemia, which can lead to life-threatening arrhythmias and cardiac arrest in AN and BN (Hofland & Dardis, 1992; Winston, 2012). This condition develops primarily due to metabolic alkalosis from purging behaviors that include vomiting, laxative

Table 8.1 Malnutrition and associated medical symptoms/complications in people with eating and/or substance use disorders

	ED	SUD	Medical conditions	Common symptoms
Protein	AN	AUD, cocaine, methamphetamine use disorders	Kwashiorkor, marasmus, anasarca, liver failure (fatty infiltrates)	Muscle wasting, enlarged abdomen, thinning hair, loss of teeth, skin depigmentation, dermatitis, edema
Glucose	AN	AUD	Hypoglycemic coma, central nervous system problems	Shakiness, anxiety, nervousness, tachycardia, palpitations. Perspiration, pallor, coldness, clamminess, dilated pupils, paresthesia
Sodium	AN	AUD	Central nervous system problems[a]	Nausea, vomiting, headache, confusion, lethargy, fatigue, anorexia, restlessness and irritability, muscle weakness, spasms or cramps, seizures, and decreased consciousness or coma
Potassium	AN and BN	AUD	Cardiac arrest	Muscle weakness, myalgia, and muscle cramps
Magnesium	AN and BN	AUD	Cardiac arrhythmias, central nervous system problems	Weakness, muscle cramps, tremors, athetosis, jerking, nystagmus, confusion, disorientation, hallucinations, depression, epileptic fits, hypertension, tachycardia, tetany
Calcium	AN and BN	AUD, opiate abuse	Cardiac arrhythmias	Muscle tetany, bruising, paresthesias
Zinc	AN	AUD	Anorexia	Poor appetite, growth retardation, acrodermatitis enteropathica, diarrhea, taste disorders, hypogonadism, increased risk of cancer
Vitamin B1 (thiamine)	AN	AUD	Wernicke's encephalopathy/ Korsakoff's syndrome/ dementia, beriberi heart failure	Mental confusion, amnesia, poor insight, confabulation, weakness, shortness of breath
Vitamin B2 (riboflavin)	AN	AUD	Angular cheilitis	Cracked and red lips, inflammation of the lining of mouth and tongue, mouth ulcers, cracks at the corners of the mouth, and a sore throat

(continued)

Table 8.1 (continued)

	ED	SUD	Medical conditions	Common symptoms
Vitamin B3 (niacin)	AN	AUD	Pellagra, dilated cardiomyopathy, dermatitis	Diarrhea, confusion, skin rash and sun sensitivity, mouth and tongue inflammation
Vitamin B6 (pyridoxine)	AN	AUD	Sideroblastic anemia, peripheral neuropathy	Lethargy, nerve damage, seizures, skin problems, and sores in the mouth
Vitamin B9 (folic acid)	AN	AUD	Macrocytic anemia	Diarrhea, weakness or shortness of breath, peripheral neuropathy, pregnancy complications, mental confusion, forgetfulness, depression, sore or swollen tongue, peptic or mouth ulcers, headaches, heart palpitations, behavioral disorders. Can lead to homocysteinemia and increased risk of cancer
Vitamin B12 (cobalamin)	AN	AUD	Pernicious anemia, dementia	Fatigue, depression, poor memory, symptoms of mania and/or psychosis
Vitamin C (ascorbic acid)	AN	AUD	Scurvy	Brown spots on the skin (thighs and legs), spongy gums, and bleeding from all mucous membranes. Pallor, depression, suppurating wounds, tooth loss
Vitamin D	AN	AUD	Rickets (children), osteopenia/osteoporosis (adults)	Muscle aches and/or twitching (secondary to hypocalcemia), bending of the spine, bowing of the legs, proximal muscle weakness, bone fragility, and increased risk for fractures
Vitamin E (tocopherol)	AN	AUD	Spinocerebellar ataxia, myopathies, anemia	Poor nerve conduction, lethargy, muscle pain

[a]Corrected too quickly can cause pontine myelinolysis

use, and/or diuretic use. Poor intake of calories and particularly of protein has been associated with liver damage, anemia, and neutropenia in AN (Sabel, Gaudiani, Statland, & Mehler, 2013; Tomita et al., 2013).

Patients suffering with AN are also at high risk of developing osteopenia or osteoporosis, conditions whose mechanisms are multifactorial including low estrogen and poor intake of calcium and/or vitamin D (Fonseca et al., 1988; Legroux-Gerot, Vignau, Collier, & Cortet, 2005; Misra et al., 2004). One study in Japan found that 40 % of adolescent patients with AN met criteria for osteoporosis and that an

earlier onset of AN increase the risk, since adolescence is such an important time for acquisition of peak bone mass development (Maesaka & Hasegawa, 2003). Vitamin D is a fat-soluble vitamin responsible for enhancing intestinal absorption of calcium and phosphate. It can be synthesized in the skin and is also absorbed in the gastrointestinal tract primarily from dairy products but is converted into its biologically active form through the liver and kidneys, and thus the presence of liver or kidney damage in AUD and/or AN can contribute to deficiency of this vitamin (De Caprio et al., 2006). Excess vitamin D taken in the form of supplements can lead to hypercalcemia and cause symptoms of anorexia, nausea, and vomiting, frequently followed by polyuria, polydipsia, pruritus, central nervous manifestations of insomnia, depression, nervousness, weakness, and ultimately renal failure (Vieth, 1999).

The B vitamins are water soluble and are present in many foods but can easily become depleted in the context of ED. Poor nutrition leading to vitamin B1 (thiamine) deficiency can occur in AN as with AUD and can lead to life-threatening Wernicke's encephalopathy and/or Korsakoff's syndrome in some patients (Handler & Perkin, 1982; McCormick, Buchanan, Onwuameze, Pierson, & Paradiso, 2011; Peters, Parvin, Petersen, Faircloth, & Levine, 2007; Saad, Silva, Banzato, Dantas, & Garcia, 2010; Sharma, Sumich, Francis, Kiernan, & Spira, 2002). Thiamine can be stored in the body for only 9–18 days and is an essential cofactor for glucose to enter the Krebs cycle for energy utilization. The brain and heart require a constant supply of glucose and are prone to severe damage when glucose by-products accumulate in the tissues and cannot be used for energy. Wernicke's encephalopathy occurs during the acute thiamine-deficient state and can be rapidly fatal especially when glucose is given without pretreatment with thiamine (Watson, Walker, Tomkin, Finn, & Keogh, 1981). The long-term sequela for those who survive thiamine deficiency is Korsakoff's syndrome. This syndrome is characterized by confabulatory amnesia (dementia), apathy, poor insight, and oculomotor manifestations such as eye-muscle weakness and/or nystagmus. Most cases of Korsakoff's syndrome occur in the absence of any clear encephalopathic episodes (Ogershok, Rahman, Nestor, & Brick, 2002). Both conditions are underrecognized in the medical setting, especially in the setting of starvation without AUD. One study of AN patients being hospitalized for treatment found that 1/3 were deficient in thiamine (Winston, Jamieson, Madira, Gatward, & Palmer, 2000). Thiamine deficiency is known to cause damage primarily to the thalamus (Zuccoli et al., 2009) even before any neurological signs are present in animals (Langlais & Zhang, 1997). In a sample of 14 patients with AN undergoing longitudinal brain imaging, two subjects had thiamine deficiency (thiamine diphosphate <70 nmol/L) and showed magnetic resonance imaging evidence of significantly increased thalamic volume (McCormick, McCann, & Keel, 2012), suggestive of osmotic damage that occurs in acute Wernicke's encephalopathy (Jung, Chanraud, & Sullivan, 2012).

Although rare, pellagra from vitamin B3 (niacin) deficiency has been shown to occur in AN and has a characteristic desquamation rash that primarily affects the face and periphery and is worsened with sun exposure (Jagielska, Tomaszewicz-Libudzic, & Brzozowska, 2007; MacDonald & Forsyth, 2005; Prousky, 2003; Rapaport, 1985). Riboflavin and/or biotin deficiency can also occur in AN and

AUD and can cause dermatitis that is similar to that in pellagra (Capo-Chichi et al., 1999; Gehrig & Dinulos, 2010; Rock & Vasantharajan, 1995). Similarly, vitamin B6 (pyridoxine) deficiency has been shown to occur with ED or AUD and can cause seborrheic dermatitis as well as peripheral neuropathy (Majumdar, Shaw, O'Gorman, Aps, Offerman, & Thomson, 1982; Schlosser, Pirigyi, & Mirowski, 2011). Folate and vitamin B12 deficiencies have been shown to occur in AN and BN (Eedy, Curran, & Andrews, 1986; Moyano, Vilaseca, Artuch, Valls, & Lambruschini, 1998) and can lead to megaloblastic anemia (Miller et al., 2005; Misra et al., 2004) and cognitive impairment (Katzman, Christensen, Young & Zipursky, 2001; McDowell et al., 2003; Tchanturia et al., 2004). The effects of folate and thiamine deficiency that lead to anemia and cognitive impairment have been well described in elderly patients who sometimes have dietary deficiencies in these vitamins as well (Morris, Jacques, Rosenberg, & Selhub, 2007; Roberts, Martin-Clavijo, Winston, Dharmagunawardena, & Gach, 2007; Selhub, Morris, Jacques, & Rosenberg, 2009). Vitamin E and other antioxidants have also been found to be reduced in some people with AN and are thought to be due to poor nutrition and oxidative stress from starvation (Moyano et al., 1999). Zinc deficiency can occur in AN and AUD, which has been associated with a number of physiological problems, including anorexia (poor appetite), growth retardation, acquired acrodermatitis enteropathica (peripheral and perioral dermatitis), taste disorder, diarrhea, and hypogonadism (Kim et al., 2010; Roberts, Martin-Clavijo, Winston, Dharmagunawardena, & Gach, 2007; Suzuki et al., 2011). Secondary zinc deficiency can occur in the presence of low levels of vitamins A and D (Potocnik et al., 2006). Vitamin K deficiency has been described in a patient with BN and was due to poor intake of green leafy vegetables (Niiya et al., 1983). Several studies have assessed whether iron, vitamin A, or essential fatty acids are deficient in ED and SUD and these appear to be normal in most patients with these conditions (Forbes & Parsons, 2012; Langan & Farrell, 1985; Lieb et al., 2011; Sabel et al., 2013).

Macronutrient deficiency in the presence of calorie restriction in its most severe form causes marasmus and has been seen in children and adults and is still prevalent in countries with poor access to food (Román, 2013). Protein deficiency in the presence of normal calories has been shown to cause Kwashiorkor in AN characterized by severe edema, muscle wasting, enlarged abdomen, thinning hair, loss of teeth, skin depigmentation, dermatitis, and edema (Esca, Brenner, Mach, & Gschnait, 1979; Grillet & Harms, 1980). Hypoglycemia can occur in AN and AUD, especially at very low weight (average BMI of 13) and/or in the presence of liver damage (Gaudiani, Sabel, Mascolo, & Mehler, 2012), which can lead to ketoacidosis, coma, and/or death (Fulop, Ben-Ezra, & Bock, 1986; Rich, Caine, Findling, & Shaker, 1990; Yanai, Yoshida, Tomono, & Tada, 2008). Cardiomyopathy or heart failure can occur in AN and can be due to deficiencies in protein, thiamine, phosphorus, magnesium, selenium, and/or ipecac poisoning (Birmingham & Gritzner, 2007). Some of the behaviors associated with ED can cause exposure to toxins. One such example was reported by one of the authors of this chapter. A 47-year-old woman with a 30-year history of AN who for 7 years had reduced her food intake to almost exclusively canned tuna was hospitalized at

53 % of her expected body weight showing marked depression and confusion and was found to have frank mercury poisoning (Ravneet & Paradiso, 2008).

Some of the complications from nutritional deficiencies do not emerge until refeeding efforts are instituted. For example, in AN it is rare for a person to have phosphate deficiency on admission for hospitalization, but this nutrient can be rapidly depleted and lead to heart failure within a few days of refeeding (O'Connor & Nicholls, 2013). A study of 50 hospitalized AN patients revealed that 60 % of patients had low magnesium and that magnesium levels should also be assessed and replaced when necessary during the first 3 weeks of refeeding (Birmingham, Puddicombe, Hlynsky, 2004). Low magnesium levels can also occur in BN and AUD and exacerbate hypocalcemia and prolong muscle tetany, cardiac arrhythmias, and even heart failure (Abbott, Nadler, & Rude, 1994; Fonseca & Havard, 1985; Hall, Beresford, & Hall, 1989). Similarly, hyponatremia is known to occur in AN and BN (Caregaro, Di Pascoli, Favaro, Nardi, & Santonastaso, 2005) and replacing sodium too quickly can lead to anasarca or refeeding edema (Rigaud, Boulier, Tallonneau, Brindisi, & Rozen, 2010). Rapid correction of hyponatremia in ED and AUD, especially in the context of hypokalemia, can also cause central pontine myelinolysis, which can manifest as acute paralysis and dysphagia (Amann, Schäfer, Sterr, Arnold, & Grunze, 2001; Heng et al., 2007; Malhotra & Ortega, 2013; Patel, Matthews, & Bruce-Jones, 2008). Guidelines on monitoring and supplementation of macro- and micronutrients during early refeeding are discussed in Chap. 23. In addition, many of these nutritional deficiencies can have adverse effects on a developing fetus, and malnutrition in AN and BN and obesity in BED can all lead to reduced fertility (Linna et al., 2013).

8.4 Co-occurrence of SUD and ED May Increase the Incidence of Malnutrition

SUD and ED have a high rate of co-occurrence (Bulik et al., 2004; Lilenfeld et al., 1998), especially between AUD and ED with bulimic features (Baker, Mitchell, Neale, & Kendler, 2010; Duncan et al., 2006; Hudson, Hiripi, Pope, & Kessler, 2007; Munn-Chernoff et al., 2013; Slane, Burt, & Klump, 2012). A meta-analysis of 21 studies reported a median rate of 23.0 % of alcohol use among individuals with BN (Holderness, Brooks-Gunn, & Warren, 1994). Another study found an AUD rate of 21 % in people with AN, most of which developed AN prior to an AUD (Baker et al., 2010). While the majority of people with AN do not develop AUD, when it does occur, there is a significantly increased risk of death and thus it is recommended that clinicians inquire about alcohol use in this population (Bulik et al., 2004). Alternatively, in a study of 140 people with SUD without organic pathology, compared to 50 healthy adults, over 90 % weighed under the mean weight for the population and over half had experienced weight loss and reduced appetite, while nearly 20 % met criteria for severe malnutrition (Santolaria-Fernández et al., 1995). While there are no studies that have specifically assessed increased risk of malnutrition when ED and SUD co-occur, it is likely that there is

an increased risk of malnutrition, electrolyte abnormalities, and/or vitamin and mineral deficiencies due to poor intake, absorption, and utilization of essential nutrients. More research in this area is greatly needed.

Conclusions

ED and SUD can lead to unhealthy eating and malnutrition that is associated with varying degrees of medical morbidity and increased risk of death. These disorders likely share some aspects of underlying pathophysiology related to reward processing in the brain as well as common medical sequelae due to nutritional deficits. Vitamin and mineral deficiencies are most common in patients with AN, BN, and AUD but can be seen in other SUD as well. The most widely appreciated nutritional deficiency is for B vitamins, particularly thiamine in AUD, but several research studies have shown that this occurs in AN as well and can cause severe neurological sequelae and/or death. More research is needed to fully understand the neurobiological consequences of malnutrition in SUD and ED and to determine the extent of the additive effects when these conditions co-occur.

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Bariatric Surgery and Substance Use Disorders, Eating Disorders, and Other Impulse Control Disorders

9

James E. Mitchell, Astrid Müller, Gavin Meany, and Cindy Sondag

Abstract

As the prevalence of obesity continues to increase worldwide, bariatric surgery is increasingly being utilized for the severely obese. The most commonly performed surgery is Roux-en-Y gastric bypass, which usually results in significant and sustained weight loss. However, increasingly, problems are being recognized after such procedures. One such problem is the development of alcohol use disorders, which may at least partially result from changes in the pharmacokinetics of alcohol after surgery. Eating problems can also develop, including binge eating or loss of control eating and, rarely, full-blown eating disorders. Other addictive disorders can occur as well, but have been much less studied. Clinicians need to be alert to the development of such problems and to institute proper evaluation and treatment if they occur.

Keywords

Alcohol use disorders • Bariatric surgery • Complications • Compulsive buying • Eating disorders • Loss of control eating • Obesity

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9.1 Introduction

As the obesity epidemic continues to grow worldwide, bariatric surgery is increasingly being utilized as a treatment for the severely obese. It is now widely recognized that about two out of three adults in the USA are overweight (BMI = 25–29.9 kg/m^2) or obese (BMI > 30 kg/m^2), and rates of obesity continue to rise across the age spectrum. Given this increase, and the growing awareness that obesity is associated with a variety of untoward health consequences, including cardiovascular disease, asthma, liver dysfunction, obstructive sleep apnea, various malignancies, and infertility (Bray, 2003; Shah & Ginsburg, 2010), bariatric surgery continues to grow as a treatment for the severely obese. This reflects several additional factors, including an increased awareness that most psychosocial treatments and many of the available medications impact only modestly on body weight and often provide only a temporary improvement. Also, increasingly, bariatric surgery has been found to be a safe and effective approach, with the subsequent significant weight loss frequently resulting in reversal or marked improvement in medical comorbidities (Latifi, Kellum, De Maria, & Sugerman, 2002).

9.2 Bariatric Surgery Procedures

In considering bariatric surgery, there are three procedures that are commonly used in the USA, and increasingly worldwide. There are other procedures that are used, though rarely, and new procedures are under development. The most commonly used procedure in the USA is the Roux-en-Y gastric bypass (RYGB), which involves the creation of a small gastric pouch in the upper stomach and bypass of the first portion of the small intestine, resulting in both a restriction of food intake and a degree of malabsorption. Also widely used is the laparoscopic adjustable gastric band (LAGB), which involves the placement of a band around the upper stomach creating a small pouch. In this procedure, there is restriction in the amount that can be eaten, but no malabsorption. The most recent procedure to be introduced, which is gaining popularity, is the gastric sleeve (sleeve), which was originally envisioned as the first of a two-stage procedure and is now increasingly being utilized as a single-stage operation. This involves creating a sleeve out of the stomach and removing a large portion of the stomach. In general, the amount of weight loss is superior with the RYGB and the sleeve to that achieved following the LAGB, and the majority of patients eventually lose 50 % or more of their excess body weight, while weight losses following LAGB placement are generally more modest. However, it must be noted that bariatric surgery procedures are not without complications, including complications during the intraoperative period (e.g., the risks of anesthesia, trauma to other internal organs), short-term complications (e.g., intestinal leaks, bowel obstruction, pulmonary embolism), and late complications (e.g., internal hernias, vitamin and mineral deficiencies, and dumping syndrome) (Byrne, 2001; Elliot, 2003; Koch & Finelli, 2010; Latifi et al., 2002). However, for

the majority of severely obese patients, the benefits of these procedures clearly outweigh the risks. The presurgical mentality rate is 0.3 % while the adjusted long-term mortality rate at a mean of 7.1 years was reduced 40 % (Adams et al., 2007; Flum et al., 2009).

In addition to the focus on gastric restriction and malabsorption as the mechanisms resulting in weight loss following RYGB, it is increasingly thought that various normal changes, including alterations of various peptides, including glucagon-like peptide 1 (GLP-1), an important regulator of glucose homeostasis, are involved. Also changes in binding to certain receptor sites are now considered possible mechanisms, including the binding to nuclear receptor Farnesoid XR (FXR) in the gut, which is also involved in glucose regulation as well as regulation of lipids and a variety of other metabolic processes. Therefore, some of the changes actually result from physiological and endocrinological changes rather than purely anatomical modifications to the gut.

Although obesity is generally not considered to be primarily an impulse control or addictive disorder, this has become an area of intense interest and debate in recent years (DiLeone, Taylor, & Picciotto, 2012; Volkow, Wang, Tomasi, & Baler, 2013). However, there is considerable overlap in the behaviors associated with obesity and these disorders. There is some literature to support the idea that patients who undergo bariatric surgery may go on to develop a new addiction or new impulse control disorder (ICD) to replace a food "addiction," depending on how this addiction is defined. Recent data have also shown that patients who are status-post bariatric surgery have an increased prevalence of alcohol disorders. Despite earlier suggestions that support the development of other addictive disorders, there are very few published data in this area. Such disorders are summarized in Table 9.1. We will first review the data on problems with alcohol before turning to other potential problems.

9.3 Bariatric Surgery and Alcohol Abuse/Dependence

Ertelt and colleagues (2008) surveyed post-bariatric surgery patients to investigate self-reported alcohol abuse and dependence before and 6–10 years after bariatric surgery. Of the 70 respondents, six individuals were identified who met self-reported criteria for a diagnosis of alcohol dependence and one participant reported alcohol abuse after surgery. In this sample, two patients developed new, postoperative alcohol dependence. Additionally, two patients reported that they did not drink alcohol prior to surgery, but did consume alcohol after surgery. Two other patients reported that alcohol consumption had increased after surgery. However, most of those in the sample reported no change in their alcohol consumption. Reports in this area should be interpreted with the knowledge that active alcohol use disorders (AUDs) are usually considered exclusionary for these surgeries, but patients may not admit to such problems prior to surgery in order to not jeopardize their chances of receiving the surgery

Table 9.1 Bariatric surgery and addictive disorders

	Disorder	Evidence	Possible mechanisms
1.	Alcohol abuse/dependence	Substantial	– Changes in pharmacokinetics – Changes in EtOH reinforcement – Dissatisfaction with psychosocial outcome
2.	Eating disorders	Substantial	– LOC eating may emerge postsurgery – May be preceded by BE/BED prior to surgery
3.	Other addictive disorders/impulse control disorders/obsessive-compulsive disorders	Anecdotal	– Addiction transfer
	– Pathological skin picking – Trichotillomania – Kleptomania – Nonparaphilic compulsive sexual behavior – Compulsive buying – Pathological gambling – Pathological Internet use – Compulsive exercise		

Sogg, Hatoum, and Turbett (2011) surveyed 340 RYGB patients about their alcohol use frequency before and at various durations after surgery and found that 73 participants reported consuming alcohol at a level that was defined as "problem drinking." Thirty-two participants reported problem drinking after surgery, and importantly, 19 of these participants did not report preoperative problem drinking.

Another published study by Suzuki, Haimovici, and Chang (2012) provided some additional relevant data. These authors surveyed 530 post-bariatric surgery patients about their alcohol consumption. Of these 530 patients, 51 agreed to be interviewed and assessed more thoroughly at a mean of 43.4 months postsurgery. Two key findings from this study were as follows (1) individuals with a lifetime history of an AUD were more likely to have an AUD after surgery compared to those who did not have such a history and (2) patients who underwent RYGB were more likely to have an AUD after surgery than those who received an LAGB.

Welch and colleagues (2011) assessed 75 RYGB patients 2 years after surgery using the CAGE, a screening tool for alcohol problems. Although not the primary focus of the study, the authors provided data on self-reported levels of alcohol abuse in this sample. Of the 75 participants who completed follow-up assessments, only 1.3 % of them responded in a manner suggesting that they abused alcohol.

Östlund (2011) presented data from 12,277 patients who had undergone bariatric surgery. She reported that patients who underwent bariatric surgery were at greater risk for inpatient treatment for alcoholism than a comparison group from the general population in Sweden, who were matched for age and gender. Importantly, she further reported that those who underwent gastric bypass were 2.3 times more likely to develop alcohol problems than those who received LAGB.

Most recently, our group along with other investigators from the NIDDK-funded Longitudinal Assessment of Bariatric Surgery-2 (LABS-2) consortium published a paper on this issue (King et al., 2012). Among 1,945 patients who underwent bariatric surgery and completed baseline (presurgery), and follow-up assessments 1 and 2 years postsurgery, there was a significant increase in AUD noted between baseline and year 2. The percentages of patients with an AUD, as defined using the AUDIT, were 7.6 % at baseline, 7.3 % at 1 year (a nonsignificant change from baseline), and 9.6 % at 2 years ($p = 0.01$ vs. baseline). The odds of a postoperative AUD were increased among males and younger patients and those who reported presurgery smoking, regular alcohol consumption, AUD, recreational drug use, a lower sense of belonging, and among those who underwent a RYGB as opposed to an LAGB. Another recent paper (Fogger & McGuinness, 2012) reported that 14 % of a series of 173 participants in a state monitoring system for nurses with addiction problems had undergone bariatric surgery, and 10 % had developed an addiction after surgery, most commonly to alcohol or hydrocodone.

Therefore, several lines of evidence, most recently the large data set collected by the LABS-2 consortium, suggest that patients who undergo RYGB have a higher risk of developing an AUD versus those who receive an LAGB. There is currently insufficient evidence regarding the development of AUD in patients who undergo sleeve gastrectomy. In summary, the RYGB is the most common bariatric surgery performed in the USA, and the majority of available data suggest that RYGB leads to an increase in the risk of a postoperative AUD.

A number of other studies have explored pharmacokinetic changes that occur following bariatric surgery. These studies are particularly relevant as pharmacokinetic changes inevitably may act as a causal mechanism for increased alcohol use problems longitudinally. The studies in this area have included a variety of methodologies including pre- and postsurgery designs, weight-based versus fixed dosing of alcohol, and alcohol concentrations measured via both breath (breath alcohol concentrations; BrAC) and blood sampling (blood alcohol concentrations; BAC). Despite the variety of methodologies used, the data have shown an accelerated and higher peak alcohol concentration in postsurgery patients. Some of these studies have also shown differences in alcohol metabolism following surgery, although this finding has been inconsistent.

In 2002, Klockoff, Naslund, and Jones published a study that examined the pharmacokinetics of alcohol in women who had undergone RYGB. They compared 12 healthy control participants to 12 patients who had undergone surgery on BAC after administration of a bolus, weight-based dose of alcohol (0.3 g kg^{-1}). They found that compared to the control group, the RYGB group experienced significantly higher BAC at the first (10 min) and second (20 min) measurement points. Groups were markedly and significantly different at the first assessment point: at the 10-min measurement, the operated group had a BAC of 0.713 g l^{-1} and the control group had only reached a level of 0.171 g l^{-1}.

In 2007, Hagedorn, Encarnacion, Brat, and Morton reported on the metabolism of alcohol in 17 control and 19 post-gastric bypass patients. They measured BrAC every 5 min from 15 to 140 min following ingestion of a fixed, 5-oz drink of red

wine. The main findings from this study were that the RYGB patients had a greater peak BrAC and, unlike the findings of Klockhoff, Naslund, and Jones (2002), a longer time for alcohol levels to reach 0 compared to controls. Notably, at the first assessment point (15 min), RYGB patients experienced higher BrAC than control participants. Limitations of this study included differing BMIs of the surgical and nonsurgical groups and a fixed dose that did not control for differences in body weight.

Woodard, Downey, Hernandez-Boussard, and Morton (2011) studied 19 RYGB patients' BrAC prior to surgery as well as 3 and 6 months after surgery. Patients drank a fixed dose (5 oz) of red wine and provided BrAC recordings at multiple time points in the study. They found that peak BrAC was higher and it took greater time to return to a BrAC of 0 in the postoperative conditions than the preoperative condition. Additionally, patients reported more dizziness and double vision in the postoperative conditions. Finally, it is important to note that the peak BrAC was highest at the first assessment point (15 min post-drink). Like the study by Hagedorn and colleagues (2007), this study is also limited by the administration of a fixed, non-weight-based dose of alcohol. This is also true for another paper published by this group (Changchien, Woodard, Hernandez-Boussard, & Morton, 2012), which failed to find evidence of changes in kinetics 3 months and 6 months after LAGB or laparoscopic sleeve, using a fixed dose, non-weight-based amount of alcohol.

Maluenda et al. (2010) studied 12 patients who had undergone laparoscopic sleeve gastrectomy. These patients drank a weight-based dose (3.6 ml per liter of water body mass) on two occasions, once prior to surgery and once 30 days after surgery. They found that BrAC was higher and it took longer to return to a BrAC value of 0 in the postsurgery condition. Again, the peak time BrAC values were collected at the first measurement point was 10 min post-alcohol consumption.

The findings from the studies reviewed above are important in that they show that postoperative bariatric surgery patients reach higher levels of intoxication compared to their presurgery state or weight-matched controls. These data, combined with the prevalence of the data cited earlier, suggest that a subgroup of patients, some of whom have a history of AUD but some of whom do not, abuse alcohol after bariatric surgery. A number of variables may presumably contribute to this outcome, including the pharmacokinetic changes in alcohol after surgery.

9.4 Bariatric Surgery and Eating Disorders

One of the areas of interest in terms of eating pathology after bariatric surgery is what is termed "loss of control" (LOC) eating. Questions remain as to whether LOC eating should include either binge eating (i.e., a quantifiably large amount of food is consumed) or subjective overeating (when a patient feels as though they are unable to stop eating but the amount of food ingested is not necessarily large) or whether both should be considered. Some studies have noted that there is a subgroup of people who indicate that they have difficulty with a sense of LOC at times while

eating after bariatric surgery and that this population is more likely to regain weight after RYGB. There is, however, a concern that LOC may be the self-labeling of an epiphenomenon associated with depression. Also, gradations of LOC are likely, as opposed to it being simply a dichotomous variable.

This issue of LOC eating following bariatric surgery will be discussed in detail given that this is the only eating-related variable which has consistently been shown to impact on weight outcomes across a variety of studies. In 1992, Rowston et al. reported follow-up data on 16 patients 2 years after biliopancreatic diversion (BPD) using the Bulimic Inventory Test, Edinburgh (BITE) questionnaire. They found that patients overall lost a significant amount of weight and that eating disordered behavior usually improved within 3 months after surgery. However, those who reported binge eating (BE) and/or LOC eating lost less weight over the longer follow-up period, although preoperative BE was not predictive of postoperative LOC. Pekkarinen, Koskela, Huikuri, and Mustajoki (1994) studied 27 vertical banded gastroplasty (VBG) patients 5 years after surgery. They found that patients who reported BE had a statistically significant greater weight regain, and very few had what they defined as a successful outcome at the end of follow-up. They defined BE as eating large amounts of food, accompanied by a sense of LOC, and subsequent feelings of guilt and shame. They used the Binge Eating Scale (BES) and BITE to identify BE behavior postoperatively and identified it as the "main predictive factor for poor outcome."

Eating disturbances in patients undergoing VBG (a practice no longer widely used in the USA) were studied by Hsu, Betancourt, and Sullivan (1996). They retrospectively analyzed the eating behavior of 24 female patients 3.5 years after surgery using the Eating Disorder Examination (EDE). A trend was found for those with an eating disturbance before surgery to regain more weight compared to those without such a disturbance. However, this study was retrospective and cross-sectional in design, of short duration, and had low statistical power owing to the sample size.

In 2001, our group did a structured long-term (13–15 year) follow-up of patients after RYGB (Mitchell et al., 2001). Seventy-eight patients were interviewed, and it was found that BE/LOC was associated with more weight regain, if present after surgery, but not before surgery. Limitations of this study included its retrospective design and the long time to follow-up that may have reduced the veracity of the data obtained. Also, some patients were lost during the follow-up interval.

Kalarchian et al. (2002) studied BE among RYGB patients at long-term follow-up. They studied 99 patients between 2 and 7 years after RYGB using the Eating Disorder Examination Questionnaire (EDE-Q) and found that BED or LOC eating was associated with less weight loss, which occurred in 46 % of patients. Another study was completed by Guisado Macias and Vaz Leal (2003). They followed up 140 patients after VBG for 18 months postoperatively. They assessed patients using the BES and did find a correlation between those who reported LOC eating postoperatively (about 18 %) and poor outcomes. However, they did not assess predictive factors.

Larsen et al. (2006) followed 157 patents approximately 34 months after undergoing LAGB. They administered the BES and found that those patients who reported engaging in BE were more likely to have regained weight. They also found that patients who engaged in what was labeled "emotional eating" or "external eating" were also more likely to meet the BE criteria, but they did not assess preoperative characteristics that might be predictive of those outcomes.

Colles, Dixon, and O'Brien (2008) studied "grazing" and LOC eating in 129 patients 12 months after undergoing banding. They used the Questionnaire on Eating and Weight Patterns-Revised (QEWP-R) and found that BE and LOC were associated with less weight loss. They also assessed preoperative characteristics. They found that only 50 % of patients who exhibited preoperative BE went on to evidence LOC eating and weight regain.

In 2010 White, Kalarchian Masheb, Marcus, and Grilo assessed 361 patients preoperatively and 1–2 years after RYGB. They used the EDE-Q, and their data revealed that preoperative LOC eating was associated with less weight loss/more weight regain. They found a high prevalence of LOC (approximately 61 % preoperatively, and about 36–39 % postoperatively). They also found that preoperative LOC was predictive of postoperative LOC. It also appears to be important as to when the LOC eating patterns returns, since reoccurrence shortly after surgery was associated with a better outcome than that reoccurring longer term after surgery in this study. The reason for this difference is not obvious.

de Zwaan and colleagues (2010) studied 59 patients after RYGB 2 years postoperatively. They used a comprehensive interview, the Eating Disorder Examination-Bariatric Surgery Version (EDE-BSV), as well as QEWP-R preoperatively, and found that preoperative LOC was associated with less weight loss. In their study population, 29 % of patients preoperatively met criteria for BE/BED and 25 % did so postoperatively. They also found that preoperative LOC was predictive of postoperative LOC in 47 % of patients. Beck, Mehlsen, and Stoving (2012) also found that BE postsurgery, at a follow-up mean of 2 years, produced less weight loss in a sample of 45 previous RYGB patients using an instrument designed for this study.

A review of these data raises several important points: first, the studies reveal that BE/LOC postoperatively is associated with less weight loss or more weight regain. However, only half of the studies evaluated preoperative eating disordered behavior, with only one study examining what was labeled preoperative LOC per se, although LOC must have been assessed in several other studies as well given that some patients satisfied criteria for BE. Second, the studies that evaluated postoperative BE/BED or LOC found rates varying widely from 3 % in Colles et al. (2008) to 46 % in the Kalarchian et al. (2002) papers. Possible contributors to this wide variance include type of surgery (LAGB vs. RYGB) and type of assessment (e.g., QEWP-R vs. EDE-Q), length of follow-up, and whether LOC or BE/BED were both assessed.

Despite this body of data, important questions remain unanswered. Why do some people experience LOC eating after surgery, while others do not? What are the predictive factors for the emergence or reemergence of this LOC eating? Is the

core psychopathology of an eating disorder present in this patient group, despite the fact that these people may not meet criteria for a current eating disorder diagnostic category? Further study is warranted to address these issues.

9.5 Bariatric Surgery and Other Addictive Disorders

We will now discuss literature regarding the presence of other ICDs and behavioral addictions. These disorders have undergone a shift in the DSM-5, which now includes kleptomania, pyromania, and intermittent explosive disorder as specific ICDs. Gambling disorder is now included as a non-substance-related addictive disorder and trichotillomania in the obsessive-compulsive and related disorders section.

"Healthy" behaviors that are performed excessively and thus may become harmful are sometimes grouped as "behavioral addictions" (see Chap. 18) and include compulsive buying, pathological skin picking, nonparaphilic compulsive sexual behavior, pathological internet use, and excessive exercising (Berczik et al., 2012; Coleman, Raymond, & McBean, 2003; Dell'Osso, Altamura, Allen, Marazziti, & Hollander, 2006; Karim & Chaudhri, 2012; Kuzma & Black, 2008; Meyer, Taranis, Goodwin, & Haycraft, 2011; Odlaug & Grant, 2010).

Corresponding to the relatively sparse research on non-substance addictions and ICDs-NOS in general, only a few studies have investigated their role in bariatric surgery patients. As obesity is associated with increased impulsivity and reduced impulse control (Davis, 2010; Gruss, Mueller, Horbach, Martin, & de Zwaan, 2012; Lent & Swencionis, 2012; Mobbs, Crepin, Thiery, Golay, & Van der Linden, 2010; Müller et al., 2012; van Hout, van Oudheusden, & van Heck, 2004), one might expect elevated prevalence rates of ICDs or behavioral addictions among bariatric surgery patients. ICDs have been shown to be relatively prominent in obese, pre-bariatric surgery candidates. In a sample of 100 presurgery individuals, 19 % met criteria for at least one current ICD and 27 % had a lifetime history of at least one ICD (Schmidt, Korber, de Zwaan, & Müller, 2012). The most prevalent ICDs were excoriation disorder and compulsive buying, with current prevalence rates of 8 % and 6 % and lifetime rates of 9 % and 8 %, respectively. With regard to other ICDs, the following current/lifetime prevalence rates were reported: intermittent explosive disorder 5 % and 10 %, pathological gambling 1 % and 3 %, and pathological internet use 2 % and 5 %. No participant was diagnosed with pyromania, kleptomania, trichotillomania, or hypersexual behavior. Of note, the prevalence rates in this obese sample differed barely from those among consecutive psychiatric inpatients who were assessed with the same structured interview (Müller et al., 2011). The pre-bariatric surgery sample consisted of consecutive morbidly obese individuals who were seen for a routine psychosocial pre-bariatric surgery evaluation but not for psychiatric treatment like the psychiatric inpatients. Considering this, it is surprising that there were no differences found between psychiatric inpatients and obese individuals who were not seeking psychotherapy or psychopharmacological treatments.

There is an increasing literature on excoriation disorder, which is now listed in DSM-5's section on obsessive-compulsive and related disorders (Grant & Odlaug, 2009; Odlaug & Grant, 2010). Excoriation disorder is characterized by repetitive picking of the skin, commonly on the head and face, although virtually any area of the body can be involved. Compulsive picking results in tissue damage and often leads to scarring, with resultant significant body image concerns. This is a widely recognized and relatively common psychiatric problem, with a lifetime prevalence estimated to be between 1.4 % and 5.4 % (Hayes, Storch, & Berlanga, 2009; Keuthen, Koran, Aboujaoude, Large, & Serpe, 2010). The aforementioned findings of Schmidt et al. (2012) are in line with earlier reports that indicate a much higher prevalence of skin picking in clinical samples than in the general population, including patients with obsessive-compulsive disorders (Grant, Mancebo, Pinto, Eisen, & Rasmussen, 2006), trichotillomania (Odlaug & Grant, 2010), and eating disorders (Favaro, Ferrara, & Santonastaso, 2007). It is important to note that pathological skin picking should be carefully delineated from non-suicidal self-injury.

Compulsive buying (see Chap. 19), also referred to as compulsive shopping or shopping addiction, also seems to be prevalent in obese individuals who are seeking bariatric surgery. This behavioral addiction is characterized by repetitive maladaptive purchasing behavior that is difficult to control and results in distress and significant social, psychological, and financial consequences. Compulsive buying is common in consumer societies with prevalence estimates of about 5–8 % (Koran, Faber, Aboujaoude, Large, & Serpe, 2006) and often is associated with other psychiatric illnesses, particularly mood and anxiety disorders, other ICDs, and BED. Whether or not it is associated with substance use disorders is unclear (Mueller et al., 2010).

The prevalence of other ICDs and behavioral addictions has not been reported in the literature following bariatric surgery. Some have argued that the problematic behaviors may increase or transfer to another problem in order to replace the presurgery "food addiction" through the development of other non-substance addictions (Buffington, 2007; Moorehead & Alexander, 2007; Spencer, 2006). Thus far, only anecdotal reports have examined the development of postoperative behavioral addictions. For example, instead of overeating/binge eating, some patients who have undergone bariatric surgery engage in compulsive buying of clothing, hypersexual behavior, or other extreme activities that appear to be triggered by the massive weight loss. Excessive exercising (see Chaps. 7 and 28) that is often linked to eating disorder pathology and extreme fear of regaining weight may also result (Blum et al., 2011; Larsen et al., 2006). Post-bariatric surgery patients frequently suffer from redundant skin. Beyond appearance-related issues, hanging skin after substantial weight loss can result in skin irritation and provoke or aggravate pathological skin picking. To our knowledge, the "addiction-transfer" hypothesis has yet to be empirically investigated adequately in terms of non-substance addictions. However, we have seen individuals within our clinical practice that suffered from behavioral addictions, and we think that the potential

link between behavioral addictions and bariatric surgery should be further addressed.

Conclusions

The hypothesis that bariatric surgery may eventually result in the development of new addictive disorders, or the reemergence of a disorder in remission, has received a great deal of attention in the media and increasing attention in the bariatric surgery literature. A growing literature suggests that AUD may reemerge or develop de novo after bariatric surgery, in particular after RYGB. Additional literature suggests that some patients postsurgery will develop so-called LOC eating. Some of these individuals will have a history of BE or BED prior to surgery. The emergence of LOC eating seems to be associated with less weight loss or greater weight regain, making it a clinically important variable in long-term outcome. Of great interest, it is currently unclear whether or not the patients that develop AUD after surgery are the same ones who develop BE/LOC eating, or how a history of either problem presurgery contributes to the development of the other problems after surgery. Currently data on other substance abuse problems after surgery, including opioids and amphetamine, are limited to a small number of case reports. Other "cross addictions," such as skin picking and excessive exercising, have been suggested as possible sequelae of bariatric surgery, although data demonstrating this cross addiction potential empirically are currently lacking.

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Prevention of Eating Disorders and Substance Misuse in Adolescence: Toward a Developmental Contextual Perspective

10

Michael P. Levine

Abstract

This chapter reviews the conceptual and empirical foundations for efforts to prevent, simultaneously, substance misuse and eating disordered behavior in adolescents. The focus is universal and selective prevention in middle and high schools, although Stice's dissonance-based form of indicated prevention is also considered, particularly its potential for application to adolescents not already at high risk. Seven converging lessons from prevention research and reviews in each field are presented as guidelines for further investigations of the joint prevention of substance misuse and the spectrum of disordered eating. The potential value of these guidelines is illustrated by a review of the ATLAS and ATHENA programs, which constitute the only project to date that has demonstrated the ability to prevent, simultaneously, attitudes and behaviors related to body image, eating pathology, and substance misuse. It is argued that simultaneous prevention of substance misuse and disordered eating should incorporate Piran's critical social perspective and an ecological model, both of which can be usefully understood and applied within Lerner's theory of developmental contextualism.

Keywords

Comorbidity • Critical social perspective • Developmental contextualism • Disordered eating • Eating disorders • Media literacy • Positive youth development • Prevention • Substance misuse

Inspection of literature reviews addressing prevention of eating disorders (ED) (e.g., Levine & Smolak, 2006; Stice, Becker, & Yokum, 2013) and prevention of substance misuse (e.g., Botvin & Griffin, in press-a; Griffin & Botvin, 2010;

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Karki et al., 2013) reveals that only one project has demonstrated the ability to prevent, simultaneously, attitudes and behaviors related to body image, eating pathology, and substance misuse. Review of the ATLAS and ATHENA programs (Elliot & Goldberg, 2008) constitutes the third goal of this chapter; the first is providing a conceptual foundation for simultaneous prevention, while the second is distillation of empirical guidelines, based on overlapping lessons from each field.

This chapter focuses on universal-selective prevention in middle and high schools. Adolescence is one high-risk period for both ED and substance misuse (Griffin & Botvin, 2010; Levine & Smolak, 2006); and, according to the Rose Paradox in public health, in a large population the clear majority of new cases of a disorder come from those at low-to-moderate risk, *not* the relatively few people who are at high risk (Austin, 2001). The final goal is to use those guidelines to identify directions for further investigations of the joint prevention of substance misuse and the spectrum of disordered eating.

10.1 Conceptual Foundations: Defining the Problems to Be Addressed

10.1.1 Substance Misuse

Drawing on a publication from Ontario's Ministry of Health Promotion (2010), *substance misuse* is defined as self-administration (ultimately by choice) of psychoactive substances (e.g., nicotine, alcohol, amphetamine, marijuana, oxycodone) in a manner—and especially in a pattern—that (1) is de jure illegal, (2) departs from culturally approved developmental expectations, and (3) generates at least one of the following: (a) danger for self or others, (b) disability in fulfilling roles and developmental tasks, (c) suffering or misery for self or others, (d) alienation from others or from one's core values, and (e) increased probability of further substance use. As more of criteria (a)–(e) are met, the likelihood increases that use is misuse.

10.1.2 Eating Disorders and Disordered Eating

For prevention purposes, it is useful to conceptualize DSM-5 ED as extremes of six intertwined continua (Levine & Smolak, 2006): (1) negative body image; (2) unhealthy forms of weight management; (3) overvaluing the self in terms of perceived weight and shape in relation to unrealistic standards of beauty, fitness, and muscularity; (4) irrational fear and loathing of body fat and fat people, all feeding drives for thinness and leanness; (5) harsh self-surveillance and self-criticism, in a reciprocal relationship with shame, anxiety, depression, and difficulties in self-regulation; and (6) binge eating. People who have eating attitudes and behaviors that generate mild-to-moderate problems [see (a)–(e) above] and who have moderate-to-high levels of (1), plus (2) or (6), and at least one of (3)–(5) fall into the broad category of *disordered eating* (Levine & Smolak, 2006).

Each of these continua, while unhealthy, is ordinary to the point of being normative and culturally syntonic.

10.2 Conceptual Foundations

10.2.1 Symptom Comorbidity and Shared Etiology

Misuse of alcohol and other drugs is comorbid with ED that revolve around binge eating and purging (see Chaps. 11 and 12). Although it is surprising the Wolfe and Maisto's (2000) review of putative risk factors for the comorbidity of bulimic behaviors and substance abuse found very little evidence supporting shared etiology, substance misuse is significantly associated with the psychopathology often accompanying ED: anxiety disorders, trauma- and stressor-related disorders, depression, the personality dimension of neuroticism, and deficits in expressing negative affect and controlling impulses (Ferriter & Ray, 2011; O'Brien & Vincent, 2003; Chaps. 6, 16, and 17). In high school and college students, substance misuse is also correlated with some of the continua constituting the definition of disordered eating (see, e.g., Parkes, Saewyc, Cox, & MacKay, 2008; Wolfe & Maisto, 2000).

There is increasing evidence that negative body image, disordered eating and ED, substance misuse, and non-suicidal self-injury are elements in a larger spectrum of significant problems in self-care reflecting "disembodiment" (Piran & Teall, 2012) and "body disregard" (Muehlenkamp, 2012). Misuse of alcohol, marijuana, or oxycodone may serve many of the same purposes as binge eating and/or self-initiated dieting, including peer acceptance, experimentation with "adult" behaviors, and, most notably, escape from painful self-awareness and other forms of distress. Misuse of laxatives, diuretics, diet pills, caffeine, and other stimulants (e.g., nicotine, amphetamine) may be undertaken to control hunger and/or control and manage weight in those with significant weight and shape concerns. Both substance misuse and the continuum of disordered eating are comprehensible and functional in the context of adolescent development within a culture haunted, if not powered, by long-standing tensions between asceticism, delay of gratification, and self-control versus intolerance of discomfort, "cutting loose," and "just do it!" (Griffin & Botvin, 2010; Levine & Smolak, 2006).

10.2.2 The Nonspecific Vulnerability-Stressor Model

It has long been known that many different types of physical and mental disorders in adolescence and adulthood have overlapping risk factors. These include vulnerability to negative affect, the experience of cumulative life stress, behavioral incompetencies (e.g., in coping skills, self-management, goal setting, and problem solving), and the juxtaposition of poor social skills and lack of social support (Levine & Smolak, 2006). Consequently, according to a nonspecific vulnerability-stressor (NSVS) model, problems will be prevented and resilience

will be promoted when stakeholders in community health, including adolescents, collaborate to make multiple environments (e.g., school, the Internet, athletics, the family) more predictable, safer, more respectful, and more responsive while strengthening opportunities for adolescents to have multifaceted lives. Stressors to be minimized range from normative developmental challenges (e.g., issues of autonomy, relatedness, identity, competence at work, sexuality) to common threats (e.g., sexual harassment, family conflicts such as divorce) to major losses and trauma (Levine & Smolak, 2006).

10.3 Conceptual Foundations: Why Prevention and What Is It?

10.3.1 Why Prevention?

The prevalence of comorbid substance misuse and ED (or disordered eating) will not be substantially reduced, beyond minimization of behavioral "contagion," by applying a detect-and-treat approach (Levine & Smolak, 2006). Even if people were open about their problems and received strong support from family and friends who were impervious to the stigma of mental illness, there would still be many psychological, sociological, and economic barriers to identification, referral, and specialized, evidence-based treatment, including an imbalance between the large number of people suffering and the small number of professionals with proper training. The detect-and-treat approach, while humane and necessary in the necessarily limited ongoing efforts to curb the *prevalence* of disordered eating + substance misuse, will never reduce its *incidence* (i.e., the number of *new* instances of the comorbid condition in a given time period divided by the number of people in a population who could reasonably be considered at risk).

10.3.2 Definitions and Assumptions Related to Prevention

10.3.2.1 Prevention

To prevent a disorder is to understand and anticipate the conditions that foster the problem and trigger its onset and to intervene in systematic ways so as to forestall or delay the disorder. One way to accomplish this is to protect health and reinforce resilience and adaptive functioning, including the ability to cope effectively with developmental challenges and life's unpredictable but inevitable hardships (Committee on the Prevention of Mental Disorders, 2009; Guerra & Bradshaw, 2008; Levine & Smolak, 2006).

10.3.2.2 The Prevention Spectrum

This chapter applies the "mental health intervention spectrum" of prevention proposed by the National Research Council (NRC) and the Institute of Medicine (IOM), both of the USA's National Academy of Sciences (Committee on the Prevention of Mental Disorders, 2009). This is a continuum from general health

and resilience promotion → universal prevention → selective prevention → indicated or "targeted" prevention, the latter shading into treatment (case identification, then intervention, and then aftercare).

The spectrum begins with policies and programs intended to improve the health and hardiness of an entire population. *Universal prevention* programs transform and strengthen public policies, social institutions, and common cultural attitudes and practices in order to prevent designated conditions (e.g., substance misuse) from developing in circumscribed by extremely large groups of people, such as all youth ages 11 through 14 in California.

Selective prevention also involves changes in public policy and group practices, but the primary audience is a large group of people who do not yet have the condition of interest (i.e., they are asymptomatic) but who clearly are *at risk* for biological, psychological, or sociocultural reasons. For example, a selective prevention program seeking to reduce the incidence of comorbid disordered eating and substance misuse could focus on girls ages 10 through 14 who necessarily are dealing with pubertal development, who live in a society that defines women in terms of slenderness and passivity, *and* who have a parent or older sibling who suffers from one or more of the following: severe depression, substance abuse, anxiety disorder, and ED. The prototypical multi-lesson ED or substance misuse prevention *curriculum* for middle or high school girls would fall between universal and selective prevention on the mental health intervention continuum (Cox & Levine, in press).

Indicated/targeted prevention programs are designed for people who have been identified as being at high risk due to warning signs (e.g., mild symptoms) and/or clear precursors (e.g., high levels of weight/shape concern; experimentation with binge drinking). The "definitely at-risk" status, as determined by screening methods with well-documented sensitivity and specificity, "indicates" that an individual or group intervention tailored for them is warranted (Levine & Smolak, 2006).

10.3.3 Prevention and Developmental Contextualism

The theory of developmental contextualism, developed by Lerner and colleagues in the mid-1980s, acknowledges the interrelationship of correlated behaviors—both high risk/unhealthy and resilient/healthy—and addresses overlapping risk-resilience factors by "embed[ding] the study of children in the actual families, neighborhoods, and communities in which they live" (Lerner, Ostrom, & Freel, 1997, p. 504). The label "developmental contextual" emphasizes the dynamic, changing, malleable, and nonreductionist relationships between individuals and the multiple physical and social dimensions of their ecology (Lerner, Fisher, & Weinberg, 2000; Lerner et al., 1997; Schoon, 2012). Examples of influential contexts are family, school, neighborhood, religious community, mass media, and cultural values. These ecological contexts are "nested spheres of influence varying in proximity to the individual and ranging from the micro- to the macro-context" (Schoon, 2012, p. 146, based on Bronfenbrenner's classic developmental theory).

Over time these contexts not only shape, but they are shaped by, the "lived experience" of individuals (Piran, 2001), who, as they develop, tend to have a growing capacity for participation in self-environment co-regulation (Schoon, 2012).

There are three important implications of this perspective, along with the NSVS model. First, the developmental-ecological contexts of adolescents should be the focus of the planned changes—the policies (at the higher levels of organization) and programs and other interventions (at the lower levels)—that we call prevention (see Lesson 6 below). This emphasis magnifies a previous important point about universal and selective prevention: effective changes must not only be planned, they must be integrated and "*aimed at changing the developmental system in which people are embedded*, *rather than at changing individuals*" (Lerner et al., 1997, p. 507; italics in the original). Implementation and evaluation of prevention in these contexts is simultaneously outreach, organizing, and empowerment in order to identify, mobilize, and strengthen *community* assets via policies and programs for youth development. This leads to the second implication: because shared risk and lack of resilience are products of multiple stressors and vulnerabilities at multiple ecological levels, prevention is necessarily about political and social change in the direction of social justice (Albee, 1983).

Third, prevention needs to arrange for positive youth development in a variety of interlocking areas, such as resilience, prosocial and community involvement, and cognitive and behavioral competencies (Guerra & Bradshaw, 2008). Prevention specialists need to work with community stakeholders to encourage and guide the "6 *C*s" of positive youth development (Guerra & Bradshaw, 2008; Lerner et al., 2000): *C*ompetence (life skills), *C*onnection, *C*haracter, *C*onfidence and positive sense of self, *C*aring (compassion), and *C*ontribution to community and society. Prevention requires fostering and reinforcing the engagement of youth in a critical social perspective (CSP) (see Lesson 3 below) and in doing meaningful things for themselves and others, including joining or leading adults in working for beneficial social changes (Piran, 2001, 2010).

10.4 Lessons and Principles for the Prevention of Drug Use and Disordered Eating

It is difficult to extract convergent guidelines from one extremely large body of prevention literature (substance misuse) and another very different and rapidly expanding body of theory and studies (disordered eating). Therefore, I offer seven shared conclusions as lessons to guide further programming and research in the simultaneous prevention of substance misuse and disordered eating in adolescents. Lessons 3, 4, and 6 also describe ED prevention approaches worth consulting as models of that lesson.

10.4.1 Lesson 1: Prevention Can Work, But Effects Are Limited and Conclusions Complicated

10.4.1.1 Substance Misuse

Reviews (see, e.g., Botvin & Griffin, in press-a, in press-b; Hansen, 1992; Tobler et al., 2000) of studies (including randomized controlled trials) conducted between 1978 and 2010 demonstrate that, in general, multi-lesson school-based programs for young adolescents *can* prevent initiation of and increases in tobacco, alcohol, and other drug use. Many variables mitigate the impact of universal-selective prevention, so, with some notable exceptions (e.g., Botvin's Life Skills Training program; see Griffin & Botvin, 2010), effect sizes for psychosocial interventions tend to be small (+.10 to +.30; Tobler et al., 2000).

10.4.1.2 Disordered Eating

According to meta-analyses by Stice, Shaw, and Marti (2007) and by Fingeret, Warren, Cepeda-Benito, and Gleaves (2006), both universal-selective and indicated programs tend to have beneficial and statistically significant effects on measures of risk (including internalization of the thin ideal, body dissatisfaction, and negative affect) and of eating pathology. As is the case for prevention of substance misuse, effect sizes tend to be small (e.g., +.06 to +.09 for universal-selective prevention) to moderate (for indicated prevention, e.g., +.18 to +.22). The most effective prevention in terms of body dissatisfaction, dieting, and eating pathology is accomplished by multi-session, interactive (i.e., engaging) programs that promote body acceptance and challenge continued internalization of the slender beauty ideal by female participants ages 15 through 25 who already have body image issues and other weight concerns. Nevertheless, universal-selective programs and indicated programs tend to be equally effective in reducing thin-ideal internalization and negative affect (Stice et al., 2007). Finally, it appears that program implementation by prevention specialists, rather than school staff, increases the likelihood of reduction in risk factors (but not eating pathology).

10.4.2 Lesson 2: Information Dissemination and Other Nonbehavioral Approaches Have Limited and Riskier Effects

10.4.2.1 Substance Misuse

A variety of different approaches have some demonstrated success, and many failures are attributable to severe methodological shortcomings (Hansen, 1992; Tobler et al., 2000). Some programs seek to foster rational decision-making and/or foster fear of drugs by educating students about the negative consequences of drug use and the etiology of drug abuse. These information-based approaches are sometimes supplemented by activities that increase appreciation and respect for the self and others in areas of life antithetical to drug use.

Many people now consider it a truism that information dissemination and affective education are useless, antiquated, and potentially dangerous. Yet, while

it is the case that as a group their mean effect sizes approach zero (Tobler et al., 2000), nearly a third of the *behavioral* results from informational, affective, and values-based programs conducted during the 1980s were positive (Hansen, 1992). However, another 25–30 % of these programs had the undesirable effect of increasing either drug use or positive attitudes toward drug use. The perception that such programs *can* backfire (i.e., be iatrogenic) has validity (Hansen, 1992).

10.4.2.2 Disordered Eating

Provision of information, particularly in a didactic or moralizing fashion, is often ineffective in changing beliefs, attitudes, and behavior (Levine & Smolak, 2006; Stice et al., 2007). There is disagreement among experts as to whether such information is likely to be harmful (see, e.g., Stice et al., 2007, vs. O'Dea, 2002).

10.4.3 Lesson 3: Information Dissemination Is an Important Aspect of Developing a Critical Perspective for Resisting and Transforming Negative Social Influences

10.4.3.1 Substance Abuse

Although provision of information alone in the service of prevention tends to be ineffective or even risky, knowledge remains the foundation of a critical perspective for resisting negative social influences. Along with healthier peer norms, specific resistance skills, and improved life skills, this critical perspective is a key component of effective prevention of substance misuse by adolescents (Botvin & Griffin, in press-a; Griffin & Botvin, 2010).

10.4.3.2 Disordered Eating

A good deal of evidence supports Piran's contention that a CSP underlies many effective prevention programs (Levine & Smolak, 2006; Piran, 2001, 2010; Piran & Teall, 2012). A CSP emerges most readily from dialogue-based education, facilitated by a skilled adult, that encourages increased awareness of and critical thinking about dominant cultural values and practices (e.g., "fat talk," teasing, peer beauty norms, mass media) in relation to gender, power and privilege, eating, and body image. These dialogues promote personal and contextualized knowledge about the experience of embodiment versus disembodiment. Muehlenkamp (2012) defines embodiment in terms of body regard or "connection to, ownership of, and understanding of the body (e.g., body integrity)" (p. 332).

A number of effective prevention programs for older children and adolescents (reviewed in Levine & Smolak, 2006) use various teaching tools to educate students about the "clash" between physical development (e.g., weight and fat gain during puberty; the genetics of diversity in weight and shape) + psychosocial development (e.g., basic needs for self-expression, a sense of control, and connection to others) + political developments (e.g., increasing freedom and opportunities for girls and women) versus unhealthy sociocultural factors such as media glorification of slenderness and dieting, rigid gender roles, sexual objectification, and peer teasing

and intimidation about fat. These sociocultural factors often distort body regard into disembodiment by promoting self-objectification, body shame and appearance dissatisfaction, the tendency to act on impulse and thus act out, mistrust of hunger and satiety signals, calorie-restrictive dieting, and negative affect. And the more disembodied one feels, the more vulnerable one is to external messages about the body and to unhealthy uses of the body for expressing anger, helplessness, and resentment (Muehlenkamp, 2012; Piran & Teall, 2012). Thus, some successful ED prevention programs, like some successful drug prevention programs (Griffin & Botvin, 2010), help students understand and think critically about whether and how they might resist the seductive psychological dilemmas created by culture.

10.4.3.3 Model Program: Piran's Ballet School Study

Piran applied the CSP model in a multifaceted program for adolescents in an elite, highly competitive, and residential ballet school in Toronto. Given the participants and high-risk setting, this was a selective-indicated program. The design, implementation, and very positive outcomes of this intensive intervention have been documented in great detail (Piran, 1999, 2001, 2010). Ultimately, Piran was able to create opportunities for relational dialogues that generated a discourse and a subculture of analysis and resistance. This type of information and education enabled Piran and the students to transform the school environment (see Lesson 6), including peer norms (e.g., no teasing), school policies (e.g., defining and forbidding harassment), curriculum (e.g., safety training), staff training and hiring (e.g., selecting supportive staff), and the physical setting (e.g., changing rooms that allow more privacy). As predicted by developmental contextualism, in the process (i.e., in this context) adolescents reduced their levels of disordered eating and increased their experiences of embodiment, agency, and meaningful interconnection with others.

10.4.3.4 Model Program: The *Media Smart* Literacy Program

Unhealthy cultural messages are rampant, and thus easily detected, in mass media. "Media literacy" is a type of CSP in which people work together to become more aware of media influences in their lives; to understand, criticize, and appreciate media content and techniques more fully; and to use this knowledge to resist and protest negative media influences while supporting and creating more positive media messages (Levine & Smolak, 2006). Wilksch and Wade (2009) conducted a randomized controlled investigation of *Media Smart*, an 8-lesson media literacy program designed for Grade 8 (M age = ~13.5) Australian girls and boys. This universal-selective program is interactive and incorporates the key literacy concepts of awareness, analysis, activism, and advocacy (Levine & Smolak, 2006). Compared to the control condition, *Media Smart* resulted in fewer shape and weight concerns and less dieting at the 30-month follow-up for girls and at 6-month follow-up for boys.

10.4.4 Lesson 4: Resistance Skills Are an Important Part of Competence Enhancement

10.4.4.1 Substance Misuse

Resistance skills include *cognitive* competencies similar to those comprising a CSP, such as understanding the immediate negative consequences of drug misuse and recognizing, understanding, and critically appraising the content and persuasive techniques of prodrug messages from mass media and peers. Another particularly important cognitive skill is understanding that, with exception of alcohol consumption by older adolescents, most adolescents do *not* smoke cigarettes or use other drugs. The teaching of resistance skills also includes modeling, practicing, and reinforcing *behavioral* competencies for avoiding high-risk situations for drug use, particularly those involving peers, and for generating counterarguments that facilitate coping with unavoidable high-risk situations. These so-called social influence programs have a more robust (but overall small positive) effect on knowledge, attitudes, and behavior than do non-skill-based interventions (Hansen, 1992; Tobler et al., 2000).

10.4.4.2 Disordered Eating

Prevention programs produce stronger average effects when, instead of being didactic and psychoeducational, they are interactive and teach skills for resisting unhealthy sociocultural influences or for increasing body satisfaction (Stice et al., 2007).

10.4.4.3 Model Programs: Dissonance-Based Interventions

As a theory-based and sustained program of development, evaluation, refinement, and dissemination, Stice's cognitive dissonance-based intervention (DBI) has transformed the prevention field in significant ways. At present it is unquestionably the most rigorously evaluated, replicable, and powerful ED prevention program. Integrating a CSP, social psychological and motivational principles, and cognitive-behavioral therapy techniques, DBI is designed to reduce risk factors and milder ED symptoms by facilitating resistance skills in adolescent girls and young women who already have high levels of body image concerns or bulimic symptoms. Consequently, this program falls into the "indicated" range of the prevention spectrum. The development, nature, and very positive short- and long-term prevention outcomes of Stice's DBI (Stice, Rohde, & Shaw, 2013) have been extensively documented (see, e.g., Becker, 2012; Stice, Rohde, Shaw, & Gau, 2011; Stice, Shaw, Becker, & Rohde, 2009).

Stice's DBI has been adapted into a universal-selective body image program (BIP) for young undergraduate women living in sororities or participating in varsity intercollegiate sports (Becker, 2012; Becker, Stice, Shaw, & Woda, 2009). The BIP, which also produces moderate-to-large prevention effect sizes that are sustained over time, is much more ecological (see Lesson 6 below) in its approach than Stice's DBI. The BIP's emphasis on community relationship building, peer interactions, peer leadership in implementing the program, changes in peer norms,

advocacy, and activism (Becker, 2012; Becker et al., 2009; Marchand, Stice, Rohde, & Becker, 2011) is consistent with the CSP (see Lesson 3), the importance of specific resistance skills as well as general life skills (Lessons 4 and 5), an ecological perspective (see Lesson 6), and developmental contextualism.

10.4.5 Lesson 5: Multifaceted Competence Enhancement Is Important

10.4.5.1 Substance Abuse

In accordance with the NSVS model, there is evidence that some adolescents use tobacco, alcohol, and other drugs to manage the dysphoria attendant to low self-esteem, social anxiety, poor social skills, and the transactions between life stressors and ineffective coping skills (Botvin & Griffin, in press-b). Botvin's LifeSkills Training (LST) extends the resistance model (normative expectations, immediate negative physiological and social effects of drug use, resistance skills) by using brief lectures, guided group discussions, and cognitive-behavioral techniques to teach, practice, and reinforce personal and interpersonal "life skills" (e.g., problem-solving, stress management, active and empathic listening, assertion). Numerous long-term evaluations converge in demonstrating that LST produces fairly large and very durable prevention effects on tobacco, alcohol, and marijuana use by young adolescents (Griffin & Botvin, 2010; Botvin & Griffin, in press-a, in press-b). Other reviews agree that comprehensive competence enhancement produces more robust and durable prevention effects than does information-based programming or resistance skill training alone (Hansen, 1992; Tobler et al., 2000). Interestingly, both the cognitive lessons and the specific resistance skill training are crucial "life skills" as part of the LST program.

10.4.5.2 Disordered Eating

In eating disorders prevention, it does not appear that addition of life skills such as stress management or decision-making results in an improvement of resistance skills (Stice et al., 2007). However, the effectiveness of multifaceted media literacy programs such as *Media Smart*, of empowerment-relational applications of the CSP (see, e.g., Piran, 1999 and Levine & Smolak, 2006), and of Becker's BIP indicates that certain life skills are indeed relevant. More research is needed to test the complex hypothesis that raising awareness of and fostering resistance to specific ED risk factors appears to be facilitated by development of skills in listening to and working with others, being assertive and courageous despite anxious uncertainty, and expressing oneself alone and with others in noting injustice and taking steps to rectify it.

10.4.6 Lesson 6: Ecological Approaches Are Very Important

10.4.6.1 Substance Misuse

Many drug prevention programs over the years, including those featuring resistance skill training and competence enhancement, have demonstrated limited long-term effects, and even the most successful programs could be enhanced (Tobler et al., 2000; see also Karki et al., 2013). Consistent with the principles of the NSVS model, developmental contextualism, and the CSP, one successful response to limited effects has been integration of school-based programming with efforts to improve the broader ecology of adolescents. Ecological factors include the school environment; parenting skills and parent–child relationships; the attitudes and behaviors of teachers, coaches, and other influential adults; the community; and mass media.

Tobler's meta-analysis (2000) found a significant pattern of increased effectiveness as programs evolve from interactive resistance skills training alone → interactive resistance skills training + life skills training → system-based programs that feature comprehensive skills training while extending the focus of prevention to families, school policies, and community policies and practices. Ecological approaches to prevention work best if programs—and especially programs for minority groups—are developed (tailored), implemented, evaluated, and maintained in consultation with school personnel, with students, and with stakeholders in the broader community. This form of developmental contextualism motivates community members to participate in implementing, evaluating, and maintaining prevention programs in ways that are inherently localized and culturally sensitive.

10.4.6.2 Disordered Eating

With respect to universal-selective prevention, a pattern of small effect sizes and limited durability of effects is also apparent in this field (Levine & Smolak, 2006; Piran, 2010; Stice et al., 2007). This pattern, coupled with the fundamental definition of universal prevention (Committee on the Prevention of Mental Disorders, 2009) and with the success of Piran's participatory action research and Becker's BIP, also points to the desirability of an ecological, context-specific approach to prevention.

10.4.6.3 Model Program: *Planet Health*

This is a school-based, ecological, and interdisciplinary intervention originally intended to prevent obesity in early adolescence (Austin, Field, Wiecha, Peterson, & Gortmaker, 2005) by decreasing television viewing and consumption of high-fat foods while increasing (a) consumption of fruits and vegetables and (b) both moderate and vigorous physical activity. Middle schools (grades 6 through 8) participating in *Planet Health* received teacher-training workshops, lessons to be integrated in the state-mandated curricula for physical education and for a wide variety of subjects, wellness sessions, and fitness funds. Attempts were also made to

work with families to modify the home environment to support the school's programs.

The initial outcomes of a randomized controlled evaluation of *Planet Health* were fascinating (Austin et al., 2005). The program failed to lower the incidence of obesity, although obesity prevalence was reduced among female students. What *Planet Health* did do was very significantly reduce, over a 2-year period, initiation (i.e., the incidence) of two forms of disordered eating behavior in girls: purging and use of diet pills. This unexpected finding was replicated in a follow-up randomized controlled trial over 1,400 girls and boys in grades 6 and 7 in 16 Massachusetts middle schools (Austin et al., 2007).

10.4.6.4 Model Program: Healthy Schools-Healthy Kids

McVey, Tweed, and Blackmore (2007) in Toronto developed an 8-month intervention to mobilize various people to get involved in analyzing and changing the ecology of a middle school in order to reduce risk and increase resilience. *Healthy Schools-Healthy Kids* integrated, for example, student curricula for learning about and improving body image, training and curriculum guides for teachers, and coordinated workshops and newsletters for parents. Curricula which addressed specific resistance skills and life skills (including healthy eating and an active lifestyle) were coordinated with small and gender-segregated peer support groups, staff training, student-generated public service announcements (PSAs) within the school, and a school play.

The results of this comprehensive school-based approach were very promising. At 6-month follow-up, girls and boys in the 7th grade who participated in the program reported less body dissatisfaction than a comparison sample, while girls attending the intervention schools also reported less commitment to the slender beauty ideal and fewer skipped meals. These risk factor reductions and the collaborative processes by which *Healthy Schools-Healthy Kids* was developed have generated, under McVey's leadership, further systemic developments, such as a web-based educational tool (matched to curriculum expectations of the Ontario and Nova Scotia Ministries of Education) designed for teachers and public health professionals who work with youth ages 9–12 years (Levine & McVey, 2012).

10.4.7 Lesson 7: Programs Should Be Engaging and Interactive for All Concerned

10.4.7.1 Substance Abuse and Disordered Eating

It appears that the commitment of key personnel to the prevention process and to the intervention itself is very important. Whether those who "deliver" and "facilitate" the program are regular classroom teachers, same-age or older peers, graduate students, or health professionals, they need to be committed to prevention in general and to faithful implementation of the program. Program fidelity will be increased when leaders are enthusiastic, willing to serve as positive role models, and provided with teaching manuals, student workbooks, and effective training and supervision.

From the perspective of participants, *active*, *interactive*, and *engaging* lessons will be significantly more effective than didactic, noninteractive interventions relying on lectures and films (Stice et al., 2007; Tobler et al., 2000).

10.5 Model Project for Preventing Comorbidity at the Universal-Selective Level

Only one prevention project has been systematically designed and evaluated with the goal of preventing problems located in the intersection between negative body image, disordered eating, and substance misuse. ATLAS and ATHENA, companion programs developed by Linn Goldberg and Diane Elliot (see, e.g., Elliot & Goldberg, 2008) at the Oregon Health Sciences University, are gender-specific interventions that integrate features of psychoeducation and positive norm development (Lesson 3 above), media literacy (Lessons 3 and 4), drug resistance and life skills (Lessons 4 and 5), use of engaging lessons (Lesson 7), and other well-established practices in designing (Lessons 1, 2, and 7) and evaluating drug prevention programs. Another important aspect of ATLAS and ATHENA is that they are explicitly ecological (Lessons 6 and 7). The setting is the team context, and the program is implemented during team practices in classroom meetings and in the weight room. Moreover, the program emphasizes training of, leadership by, and constant support from coaches and captains, all of whom are on "on the same page" as far as program themes and goals.

10.5.1 ATLAS

Adolescents Training and Learning to Avoid Steroids is an award-winning selective intervention to prevent misuse of anabolic steroids and food supplements by high school football players (Goldberg et al., 2000). In addition to providing education about developmental physiology and nutrition, ATLAS uses various teaching methods to encourage male athletes to think critically about the impact of powerful masculine norms and mass media on body image, eating, exercising, and substance misuse. The athletes get hands-on instruction and practice in resisting pressures for use of steroids and food supplements. This is complemented by direct instruction in safe, effective techniques for developing strength and managing weight (e.g., "getting larger"). As part of media literacy training, small groups of athletes work together to generate simulated media (e.g., PSAs, posters, video, theatrical performances) that promote healthier models and forms of strength and fitness.

Goldberg et al. (2000) found that, at 1-year follow-up, boys participating in the ATLAS program increased their knowledge (e.g., about exercise, alcohol, and anabolic steroids) and were more skeptical both about the value of anabolic steroids for bulking up and about promotion of supplements and steroids via positive images and messages in strength and fitness magazines. Athletes receiving the ATLAS program also reported great self-efficacy in healthy strength training while

perceiving their coaches as more intolerant of steroid and supplement use. Most important, not only did program participants report less intent to use, at follow-up they were less likely to initiate use of anabolic steroids, "athletic" supplements, other performance-enhancing drugs, alcohol, or other drugs (Goldberg et al., 2000).

10.5.2 ATHENA

The success of ATLAS spurred development of a parallel selective program for high school girls called *Athletes Targeting Healthy Exercise and Nutrition Alternatives*. ATHENA's overarching goal is to prevent eating problems and *unhealthy* forms of weight/shape management and of performance enhancement, including use of diet pills, nicotine, cocaine, "nutritional" supplements, and anabolic-androgenic steroids. ATHENA does not teach about ED, body weight, and weight management by calorie counting, although these topics tend to emerge during team discussions or in the students' creation of PSAs.

"Athletes" are defined as girls participating in varsity sports, as well as cheerleaders, flag twirlers, and members of the dance/drill teams performing at athletic events (Elliot et al., 2006). ATHENA is a gender-specific adaptation of the ATLAS program in that most young female performers want to be leaner and lighter, not bigger, more muscular, and much stronger. ATHENA follows the structure of ATLAS, and there are eight 45-min classroom sessions and three 30-min weight room sessions.

A large-scale randomized controlled trial revealed that, at the conclusion of the program, girls ages 14–16 who had participated in ATHENA were significantly less likely to (a) initiate use of diet pills, amphetamines, anabolic steroids, and muscle-building supplement and (b) express intentions to diet for weight loss and to control their weight through self-induced vomiting and use of drugs. Girls in the ATHENA program also reported healthier eating, better mood, and fewer injuries. In addition, across three cohorts of adolescent female athletes, there were increases in the proposed mediating protective factors: knowledge of the negative effects of steroids and diet pills, media literacy, drug resistance skills, and self-efficacy for healthy eating and for controlling mood. As important, there were also increases in the perception that coaches are opposed to, and that few peers use, body-shaping drugs and unhealthy weight management practices. Of course, there is still room for substantial improvements in this type of program. ATHENA did not produce a between-group difference in body image. Moreover, as is often the case in prevention programs (see Lesson 6), many positive effects seen in the short-term dissipated over the 1–3-year follow-up period (Elliot et al., 2008).

10.6 Conclusions and Future Directions

There are clear empirical and conceptual reasons for understanding substance misuse and disordered eating in adolescent girls as related within a spectrum of disembodiment and body disregard. Similarly, comparison of prevention studies and reviews in the fields of substance misuse and ED strongly suggests there are also conceptual and practical reasons for pursuing simultaneous prevention. The overlapping lessons from prevention work in both fields, embodied in the very promising ATLAS and ATHENA programs, are important implications for further program development and research.

One important challenge illuminated by the seven lessons is the need to combine a more ecological approach for adolescents with the more specific elements of the CSP, the teaching of skills to analyze and resist the slender beauty ideal and the objectification of girls and women, and the development of life skills. Adaptation of Piran's participatory action approach in the ballet school and/or Becker's BIP for work with adolescent athletes, dancers, actors, scouts, etc., would be a good first step in this regard. Both of these approaches extend the application of cultural literacy, as seen in the ATLAS/ATHENA project and in other successful programs (e.g., Stice's DBI; Wilksch and Wade's *Media Smart* intervention) to transformative social action such as establishment of new peer norms, development of healthier media, and other forms of in vivo activism and advocacy. These actions, particularly when they are developed and undertaken in collaboration with various community stakeholders, increase the potential for more sustained and universal preventive effects.

This type of work, though necessary, will certainly not be easy. Apart from the promising ATLAS and Media Smart programs, very little is known about the prevention of body image, eating problems, and muscle dysmorphia in boys and young men (Levine & Smolak, 2006). And honest and open critical analysis of the nature of and risk factors for substance misuse and disordered eating will need to address emotionally charged issues such as gender, race/ethnicity, social class, personal and political power, fairness and justice, and commercial interests. Another challenge is the coordination of universal, selective, and indicated prevention (Levine & McVey, 2012). Readers are referred to a recent chapter by Cox and Levine (in press) which used the model of the United States Air Force's approach to multidimensional, integrated suicide prevention to propose a similar spectrum approach to simultaneous prevention of disordered eating and non-suicidal self-injury.

A fourth and no less significant challenge is that, although schools are a logical and evidence-based site for effective prevention, "the schools" (i.e., board members, administrators, teachers, and staff) are under considerable pressure to do many important things, ranging from teaching basic subjects to creating the attitudinal and behavioral components of citizenship and leadership. Thus, future prevention programming will likely be under pressure to incorporate topics that are of as much if not more concern than disordered eating and substance abuse. Notable among these are violence and obesity. Two important findings that need to be

addressed in the simultaneous prevention of substance use and disordered eating are (1) the many shared risk factors for obesity and disordered eating patterns (Haines & Neumark-Sztainer, 2006), and (2) obesity/overweight as a risk factor for substance use in adolescent girls only and for whether older adolescent boys carry a weapon (Farhat, Iannotti, & Simons-Morton, 2010). These findings return us to the web of interrelationships, emphasized by the NSVS model and by developmental contextualism, between various risk factors and between various unhealthy, negative outcomes. In the present cultural contexts, obesity and overweight are risk factors for many negative outcomes and practices, just as being a victim of violence in many forms is a risk factor for ED, SUD, obesity, depression, and so forth.

Meeting each of these challenges is a daunting task. Nevertheless, the theory of developmental contextualism (Lerner et al., 2000) provides an excellent framework for envisioning social transformation and for judging the potential value and outcomes of more omnibus prevention programming. We need to keep asking: does the process of program development, implementation, evaluation, and dissemination enable adolescents to form meaningful, caring relationships with other adolescents and with adults in order to develop the confidence and new skills necessary to do meaningful things and be a meaningful, positive presence in their community? An affirmative answer means there is an excellent chance this process will contribute to prevention of various problem behaviors. Moreover, in many countries there are now many people with compassion, resources, and power who have a stake in working together to promote, at the population level, (1) a positive body image; (2) more active, less sedentary lifestyles; (3) better eating habits (e.g., more fruits and vegetables, less saturated fats, fewer soft drinks); (4) reduced misuse of drugs; (5) greater safety and respect for its citizens, regardless of weight, shape, race, etc.; and (6) more involvement of adolescents and other people of all ages in improving society.

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Part II

Clinical Perspectives

Introduction to Eating Disorders for Substance Abuse Specialists

11

Amy Baker Dennis and Tamara Pryor

Abstract

Eating disorders and substance use disorders co-occur frequently; however, at the present time there are no evidence-based treatments to guide the practitioner faced with this comorbid condition. Given the high rates of co-occurrence and the complex nature of these disorders, it is surprising to note that most substance abuse clinicians and treatment programs have not incorporated eating disorder protocols into their practices. Likewise, most eating disorder specialists are not adequately trained in the treatment of substance use disorders, and very few eating disorder treatment programs provide comprehensive, integrated services for these dually diagnosed patients. Consequently, clinicians presented with these patients tend to focus on their area of specialty without addressing directly the other comorbid condition. Inadvertently, this can prolong the patient's suffering as they vacillate between their substance use disorder and their eating disorder. Additionally, outpatient clinicians needing to refer patients to a higher level of care or parents seeking treatment for their loved one with both disorders find locating providers or programs that effectively treat both disorders complicated and confusing. Should they seek a reputable substance abuse program or an eating disorder program? This chapter is designed for substance abuse specialists that have limited knowledge or expertise in the diagnosis, assessment, and treatment of individuals with ED. Cross-training between fields is the foundation to developing comprehensive and integrated programs for this population.

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Eating disorders (ED) and substance use disorders (SUD) co-occur frequently; however, at the present time there are no evidence-based treatments (EBT) to guide the practitioner faced with this comorbid condition. Given the high rates of co-occurrence and the complex nature of these disorders, it is surprising to note that most substance abuse clinicians and treatment programs have not incorporated eating disorder protocols into their practices. Likewise, most eating disorder specialists are not adequately trained in the treatment of SUD and very few eating disorder treatment programs provide comprehensive, integrated services for these dually diagnosed patients. Consequently, clinicians presented with these patients tend to focus on their area of specialty without addressing directly the other comorbid condition. Inadvertently, this can prolong the patient's suffering as they vacillate between their SUD and their ED (Dennis & Helfman, 2010). Additionally, outpatient clinicians needing to refer patients to a higher level of care or parents seeking treatment for their loved one with both disorders find locating providers or programs that effectively treat both disorders complicated and confusing. The intention of this chapter is to provide a basic overview of ED for students, clinicians, and researchers in the health and mental health fields. It is specifically designed to inform substance abuse specialists that have limited knowledge or expertise in the diagnosis, assessment, and treatment of individuals with ED. Chapter 12 provides a similar overview of SUD for the ED treatment provider who does not have a comprehensive understanding of the diagnosis, assessment, and treatment of SUD. These chapters provide a basic foundation for the reader who is interested in working with individuals that struggle with both ED and SUD. One of the primary goals of this volume is to move the mental health field toward an integrated approach to treatment that can be adopted by both the ED and SUD treatment communities. However, a considerable amount of cross-training between disciplines and specialists needs to be initiated before an integrated approach to treatment can take place (Helfman & Dennis, 2010). Research suggests that when comorbid diagnoses are treated concurrently and integrated on-site, treatment retention and outcome improve (Weisner, Mertens, Tam, & Moore, 2001).

11.1 Eating Disorders

There are three primary ED: *anorexia nervosa* (AN, with the following subtypes: restricting anorexia nervosa [ANR] and binge/purge anorexia nervosa [ANBP]), *bulimia nervosa* (BN), and *binge eating disorder* (BED). A fourth, residual category entitled *Other Specified Feeding or Eating Disorder* (OSFED) will also be

reviewed. This category includes disorders of eating that do not meet the full diagnostic criteria for a specific eating disorder, but significantly interfere with psychological, occupational, or social functioning. Please note that this residual category was formerly entitled Eating Disorder Not Otherwise Specified (EDNOS), in DSM-IV-TR (American Psychiatric Association, 1994), and included the diagnostic criteria for BED.

This chapter begins with a general overview of diagnosis, clinical characteristics, common medical complications, assessment, and EBT for each specific ED. It will be followed by a brief discussion of common risk factors associated with the development of ED and patterns of comorbidity with other psychiatric disorders.

11.2 Anorexia Nervosa

11.2.1 Diagnosis

The DSM-5 (American Psychiatric Association, 2013) diagnostic criteria for AN have three principle features. First, there is evidence of restricted energy intake leading to significantly low body weight that is less than minimally normal or less than expected for children and adolescents in the context of age, sex, developmental trajectory, and physical health. Second, there is an intense fear of gaining weight or "becoming fat" or persistent behaviors that interfere with weight gain even when significantly under minimally expected weight. Finally, there is a disturbance in the way in which one's body weight or shape is experienced (i.e., persistent lack of recognition of the seriousness of low body weight). Although the DSM-5 removes the amenorrhea criteria to meet the diagnosis of AN, females who endorse primary or secondary amenorrhea have poorer bone health than those that fail to meet this criterion.

As noted earlier, there are two subtypes of anorexia nervosa (e.g., restricting subtype, ANR, and binge/purge subtype, ANBP). Although there is considerable crossover between these subtypes, DSM-5 requires subtyping be specified for the last 3 months (e.g., the current episode of the illness). Individuals with ANR do not engage in recurrent episodes of binge eating or purging, whereas individuals with ANBP regularly binge eat and employ compensatory behaviors to counteract the effects of the consumption of calories (i.e., self-induced vomiting, misuse of laxatives, diuretics, enemas, excessive exercise).

11.2.2 Clinical Characteristics

Although dramatic weight loss is the most profound clinical feature of AN, it is a complex psychiatric illness that impacts psychological, social, and physical functioning. This disorder typically develops during early to mid-adolescence but has become increasingly common in older individuals. Lifetime prevalence estimates

Table 11.1 Lifetime prevalence of eating disorders by gender

Eating disorder	Adult females (%)	Adult males (%)	Adolescent females[a] (%)	Adolescent males[a] (%)
Anorexia nervosa	0.9	0.3	0.3	0.3
Bulimia nervosa	1.5	0.5	1.3	0.5
Binge eating disorder	3.5	2.0	2.3	0.8

Adapted from Hudson et al. (2007) ($n = 2{,}980$)
[a]Adapted from Swanson et al. (2011) ($n = 10{,}123$)

are .9 % among women and .3 % among men with a mean age of onset 18.9 years (Hudson, Hiripi, Pope, & Kessler, 2007; Swanson, Crow, Le Grange, Swendsen, & Merikangas, 2011) (see Table 11.1). A 2009 study that examined 1,666 AN and 793 BN entering an outpatient program from 1985 to 2008 found the age of onset is decreasing in younger generations (Favaro, Caregaro, Tenconi, Bosello, & Santonastaso, 2009). Some researchers suggest that current statistics may underestimate the prevalence of AN in males, as they are less likely to seek treatment, and physicians and mental health providers may fail to detect or diagnose an ED in males (Strother, Lemberg, Stanford, & Turberville, 2012) (see Chap. 20).

From a psychological perspective, AN is a disorder of both control and denial. Underlying psychological problems are frequently displaced onto food, weight, and body issues. Regulating food intake, body size, and weight brings a personal sense of mastery and control into a world that is perceived as unmanageable. For many individuals, engaging in ED behaviors seems to answer a question, solve a problem, or alter the environment in a positive way. Rigid control of food intake and weight is often believed to either cause or prevent some life event or change a mood state (e.g., prevent parental divorce, enhance athletic prowess, attract a romantic partner, delay attendance at college, deter sexual abuse, reduce anxiety or negative affect) (Dennis & Sansone, 1997). Body image disturbance (i.e., the misperception or overestimation of body size and shape) contributes to the active denial of the seriousness of low weight. Pervasive denial (anosognosia) may require parents or significant others to intervene to insure adequate medical, nutritional, and psychological treatments. Unfortunately, the disorder tends to worsen familial and interpersonal relationships, increases anxiety and depressive symptoms, diminishes concentration, and impairs judgment and cognitive capacity.

From a neurobiological perspective, individuals with AN often exhibit significant cognitive rigidity (i.e., reduced cognitive flexibility or impairment in set-shifting) and weak central coherence (attending to the details of a situation with an inability to see the "bigger picture") (Nunn, Frampton, Fuglset, Torzsok-Sonnevend, & Lask, 2011). These deficits may manifest in an inability to switch from one task to another task in a flexible and efficient manner or adapt to changing goals, rules, or environmental experiences. Consequently, once dieting and other compensatory behaviors (e.g., exercising) have commenced, rigid rules and rituals are meticulously followed to insure weight loss.

Unusual food-related behaviors are common in AN including categorizing foods into "good" (low calorie) or "bad" (high calorie or high fat) foods and the strict avoidance of, or purging of, unacceptable items such as meat, carbohydrates, sweets, and fats. "Safe foods" (fruits and vegetables) are preferred to higher caloric foods, resulting in low daily caloric intake and significant weight loss. Although research has suggested that individuals with AN often have an impaired sense of taste (Kaye, Fudge, & Paulus, 2009; Nunn et al., 2011), food consumption is often governed by rigid internal rules (e.g., eating only one meal per day, no food combining, the elimination of whole food groups, specific placement of food on a plate, exact number of chews per bite, time and location of acceptable eating, avoidance of eating in front of others). When the rules are challenged or broken, it is often accompanied by tremendous anxiety and guilt. The pleasure and enjoyment of eating is lost. Yet, paradoxically, although individuals with AN avoid eating, there appears to be a need to vicariously enjoy food as long as it is not ingested. They may enjoy preparing foods for others, hoarding or storing large amounts of food, collecting recipes, watching gourmet cooking shows, and working in the food service industry or experience pleasure watching others eat.

As AN progresses, dieting and the accompanying compensatory behaviors become an increasingly important focus in life. This is often at the expense of interpersonal relationships and leisure activities. Profound changes in mood (e.g., depression, anxiety, irritability, or lability) can impact on family dynamics and result in social withdrawal and avoidance of peer relationships. The maintenance of weight loss becomes a measure of personal success, self-esteem, control, and security. Attempts to confront, alter, or normalize eating patterns or eliminate self-destructive compensatory behaviors are often met with anger and resistance.

11.2.3 Medical Complications

A large number of medical complications can result from the severe weight loss and malnutrition inherent in AN (American Psychiatric Association, 2006). Typical physical complaints include fatigue, hypotension, abdominal discomfort or constipation, cold intolerance, lethargy or hyperactivity, hair loss, and dry skin. Among the more common medical complications are amenorrhea, dehydration and electrolyte disturbance, gastrointestinal disturbance, kidney dysfunction, and neurological abnormalities (Academy for Eating Disorders, 2012; Weiderman & Pryor, 1996). There is a regression of the hypothalamic-pituitary-gonadal axis in those with AN so that endocrine functioning often resembles that of prepubertal or pubertal individuals. There may be some reduction in antidiuretic hormones resulting in partial diabetes insipidus (Birmingham & Treasure, 2010). Osteoporosis is a potential long-term consequence of low calcium intake and absorption, reduced estrogen secretion, and increased cortisol secretion.

A common medical finding in individuals with AN is a slowed heart rate (bradycardia). Although normal with long-term aerobic training, in AN it is likely due to malnutrition (downregulation of the sympathetic nervous system secondary

to a catabolic state) or hypothyroidism (Birmingham & Treasure, 2010). Gastrointestinal disturbance is common and may include constipation, physical trauma from vomiting, and loss of bowel activity due to chronic abuse of laxatives. Additionally, delayed gastric emptying (gastroparesis), a by-product of starvation, often leads to uncomfortable feelings of fullness. Thus, many individuals with AN avoid eating not only from fear of weight gain but also to avoid the discomfort and "bloating" following ingestion of even a small amount of food.

Self-induced vomiting and/or diuretic or laxative abuse may cause a variety of metabolic abnormalities. The most common of these are elevated serum bicarbonate (metabolic alkalosis), hypochloremia (low chloride), hypokalemia (low potassium), hyponatremia (decreased sodium), hypomagnesemia (low magnesium), and hypophosphatemia (low phosphates) (Mehler & Anderson, 2010). The clinical expression of electrolyte abnormalities may appear as muscle weakness, fatigue, dysphoria, constipation, dizziness, and heart palpitations, while at its worst, cardiac arrhythmias and sudden death. Individuals with AN who purge may vacillate between dehydration and excessive fluid retention. Subtle and sometimes profound changes in cognitive functioning, often complicated by electrolyte and micronutrient deficits, impair an already unresponsive cognitive and psychological state of awareness and ability to participate in recovery. A variety of neurological complications can arise in AN, including shrinkage of the brain, headaches, confusion and seizures (as a result of hypoglycemia), and tingling sensations in the extremities (peripheral paresthesia) (Birmingham & Treasure, 2010; Weiderman & Pryor, 1996). (For further information, see Chap. 15.)

11.2.4 Assessment

Assessment of AN is a process that is conducted over several sessions by a multidisciplinary team of health and mental health professionals. A thorough assessment should include a comprehensive physical exam and lab tests to identify any medical complications or electrolyte abnormalities that may need immediate attention (see Chap. 15). In addition to the DSM-5 diagnostic criteria, there are numerous self-report and structured interview–screening devices that can be employed to measure the severity of eating-related symptomatology, ED attitudes, and behaviors (ED screening instruments are detailed elsewhere, see Chap. 14). Due to the high prevalence of comorbid psychopathology in AN, screening for mood disorders, anxiety disorders, SUD, impulse control disorders, and personality disorders should be conducted during the assessment to insure the development of a comprehensive treatment plan (Dennis & Sansone, in press). The assessment process should culminate in a comprehensive team treatment plan (e.g., physician, therapist, dietitian, and other specialists if necessary) and assignment to the appropriate level of care.

A patient's level of care is determined by several factors including severity of illness, medical complications, psychiatric comorbidity, lethality/dangerousness of behaviors, and availability of services. The American Psychiatric Association

Table 11.2 Lifetime prevalence of psychiatric disorders among eating disorders

Eating disorders	Mood disorders (%)	Anxiety disorders (%)	Impulse control disorders (%)	Substance use disorders (%)	Personality disorders[a]
Anorexia nervosa	42.1	47.9	30.8	27.0	AV, BPD, OCPD
Bulimia nervosa	70.7	80.6	63.8	36.8	BPD, DEP, AV
Binge eating disorder	46.4	65.1	43.3	23.3	OCPD, AV BPD

Adapted from Hudson et al. (2007) ($n = 2{,}980$)
AV avoidant personality disorder, *BPD* borderline personality disorder, *OCPD* obsessive–compulsive personality disorder, *DEP* dependent personality disorder
[a]Adapted from Sansone, Levitt, and Sansone (2006)

practice guidelines (2006) outline five basic levels of care for patients with AN. Outpatient care (usually less than 9 h per week) is indicated for medically stable patients whose weight is greater than 85 % of ideal, without suicidal ideation and fair to good motivation. Outpatients need to be self-sufficient, able to manage their behaviors, and have adequate social support. Intensive outpatient programs ([IOP] 9–20 h per week where the patient resides at home) are utilized for patients with fair motivation and body weight within 80 % of ideal. Patients are provided with structured meal support and mild external structure that can produce significant behavioral change. Day treatment/partial hospitalization programs ([PHP] at least 20 h per week with the patient residing at home or in supervised housing) are designed for patients whose body weight is higher than 80 % of ideal but have low levels of motivation, are preoccupied with intrusive thoughts, and need higher external structure and significant meal support. Residential treatment (24-h "live in" supervised care) is indicated when a patient is below 85 % of ideal weight, needs supervision for all meals, and requires a full-time structured environment to reduce ED symptoms and compensatory behaviors as well as improve medical stability. Finally, inpatient hospitalization, the highest level of care, is reserved for patients requiring medical stabilization, who have low levels of motivation and/or comorbid psychiatric conditions that require full-time supervision (see Table 11.2).

Determining the appropriate level of care at intake can be a challenge. Unfortunately, some American insurance carriers require a "failed" outpatient experience before they will approve payment for a higher level of care. In other cases, insurance providers may limit the consumer's choices by only providing payment for certain levels of care or paying for only "preapproved" treatment providers or programs. ED are protracted illnesses and instead of "treating to outcome" (100 % of expected body weight), some insurances will only pay for a predetermined number of days, or to 85 % of expected body weight regardless of the patient's mental health or recovery status. Unfortunately, this policy can lead to high relapse rates and multiple readmissions.

11.2.5 Psychological Treatments

To date, there are no psychotherapy EBT for adult anorexia nervosa (Keel & McCormick, 2010). However, one study compared a "novel" approach Maudsley Model of Anorexia Nervosa Treatment for Adults (MANTRA) to Specialist Supportive Clinical Management (SSCM) in a randomized controlled trial (Schmidt et al., 2012). Although recovery rates for both conditions were low, patients assigned to SSCM condition were less likely than the MANTRA group to require additional inpatient or day care treatment following the 20-week treatment study.

Another promising approach is outpatient Cognitive Behavioral Therapy-Enhanced (CBT-E) which was adapted for the treatment of adult AN. In a recent study, Fairburn, Cooper, Doll, O'Connor, Palmer, & Dalle Grave (2013) report that after 40 weekly sessions of CBT-E, 64 % of patients completed the treatment. One third of the study participants were withdrawn from the study and were referred for more intensive treatment. Sixty-two percent of the completers achieved a BMI $\geq$ 18.5, and 87 % showed a significant reduction in ED psychopathology and general psychiatric features by the end of treatment. At the completion of the 60-week follow-up period, 55 % of the completers had maintained a BMI $\geq$ 18.5 and 78 % had minimal residual ED psychopathology (Fairburn et al., 2013).

Finally, in another recently published multicenter randomized controlled efficacy trial in adults with AN (Zipfel et al., 2014), researchers compared two manualized outpatient treatments (CBT-E and focal psychodynamic therapy) to optimise treatment as usual. At the conclusion of 10 months of treatment, body mass index increased in all groups but there were no significant differences between treatments, including at 12 month follow-up.

Due to the dearth of RCT in adult AN, little information is available to guide the clinician in the treatment of this population. In practice, most outpatient, intermediate (i.e., PHP or IOP), and residential/hospital programs use an eclectic treatment approach to weight restoration, the elimination of compensatory behaviors, and ED psychopathology. Integrated approaches, which combine cognitive and behavioral strategies and interpersonal, dialectical, psychodynamic, and pharmacological interventions, are often employed. Individual psychotherapy, family therapy, group, and nutritional counseling all play an important role in the recovery process.

Fortunately, there is emerging data to suggest that there are two effective interventions for the treatment of adolescent AN (Lock et al., 2010; Robin et al., 1999). Both studies, a decade apart, compared manualized family-based treatment to a manualized individual therapy, specifically designed for adolescent ED. These approaches to weight restoration and resolution of underlying ED psychopathology are strikingly different. In family therapy, the adolescent anorexic is seen with the entire family for each session. In phase one of treatment, parents are absolved of causing the ED and encouraged to take control of their adolescent's eating behaviors and manage the refeeding process. During phase two, the parents transition eating and weight control back to the adolescent. Finally, in phase three, the therapist helps the adolescent and family readjust to a normal life by focusing on family communication styles, social issues, and any other problems that may have

contributed to the development of the ED or may present a risk for relapse. In contrast, patients in individual therapy are seen alone by a therapist who periodically meets in collateral sessions with the parents. Treatment focuses primarily on the amelioration of ED symptoms by examining pertinent developmental themes including coping skills deficits; individuation from the nuclear family; identity confusion; physical, social, and emotional growth; and the relationship of these issues to eating, weight expectations, and body image. The therapist strongly encourages the adolescent to refrain from dieting by setting firm goals for weight gain. But, the adolescent is in charge of eating and weight gain. The therapist helps the patient distinguish emotional states from bodily needs, encourages separation and individuation, and increases their capacity to tolerate negative affect.

Robin and colleagues (1999) found that family therapy (Behavioral Family Systems Therapy, or BFST) produced greater weight gain and resumption of menses at the end of 1-year treatment than individual therapy (Ego-Oriented Individual Therapy, or EOIT) but that there were no significant differences between groups at the end of 1-year follow-up. There were significant improvements on ego functioning, eating attitudes, family relations, and depression at the end of treatment and 1-year follow-up in both groups.

In the replication study done by Lock and colleagues (2010), comparing family-based treatment (FBT) and individual therapy (EOIT was renamed Adolescent Focused Therapy or AFT), similar results were found. There were no differences in full remission between conditions at the end of treatment. Family therapy was superior for partial remission at the end of treatment but there were no differences between groups at 1-year follow-up. Both conditions produced similar results at 6- and 12-month follow-up for weight gain and reduction in ED pathology.

An important step in fully understanding the clinical implications of these studies is to determine what adolescents fair best in which treatment condition. A recent study has been published looking at moderators and mediators of remission in FBT and AFT for AN (LeGrange et al., 2012). These authors cautiously recommend that for adolescents with ANBP that have high ED psychopathology (measured by the EDE-Global; Cooper, Cooper, & Fairburn, 1989) and high levels of obsessive–compulsive features of eating symptoms and behaviors (measured by YBC-ED; Masure, Halmi, Sunday, Romano, & Einhorn, 1994), FBT appears to produce better results by the end of treatment. Both FBT and AFT appear useful psychotherapies for adolescents with lower levels of psychopathology (LeGrange et al., 2012) (see Chap. 26).

11.2.6 Pharmacological Treatments

Clinically relevant RCT in the treatment of AN have used atypical antipsychotics, selective serotonin reuptake inhibitors (SSRIs), and zinc supplementation (Flament, Bissada, & Spettigue, 2012). Earlier studies suggested that the SSRI fluoxetine can help prevent relapse in weight-restored AN patients (Kaye et al., 2001; Kaye, Weltzin, Hsu, & Bulik, 1991), although two other studies showed no significant

differences between patients who received fluoxetine and those that did not (Strober, Freeman, DeAntonio, Lampert, & Diamond, 1997; Walsh et al., 2006). Due to fluoxetine being a potentially activating SSRI, caution is advised when prescribing it to highly anxious patients (Stahl, 2005).

Although medications are often prescribed, there continues to be controversy in the field with some researchers arguing that no medication has been proven to have a decisive and conclusive impact on the primary psychological features or upon weight restoration in AN (Bulik, Berkman, Brownley, Sedway, & Lohr, 2007; Guarda, 2008) and that there is only limited evidence for the use of medications to treat chronic AN (Yager et al., 2012).

In a comprehensive review of pharmacological treatments for AN between 1977 and 2010, olanzapine was found to increase weight gain and improve depression, anxiety, aggression, and obsessive–compulsiveness in the treatment of AN (specifically ANBP subtype) (Aigner, Treasure, Kaye, Kasper, & The WFSBP Task Force on Eating Disorders, 2011). Brewerton (2012) suggests that olanzapine may currently be the best psychotropic medication for the treatment of AN, particularly if patients do not have access to structured, intensive treatments (e.g., inpatient, residential, or IOP). Although olanzapine and other antipsychotic medications have demonstrated some efficacy in the treatment of AN, the potential risks remain a concern (e.g., off-label use, metabolic disturbances, extrapyramidal side effects). Clinically speaking, some suggest that weight status should be mastered psychologically, not pharmacologically, when possible (Dennis & Sansone, in press).

11.3 Bulimia Nervosa

11.3.1 Diagnosis

The DSM-5 (American Psychiatric Association, 2013) criterion for BN defines binge episodes as the consumption of large amounts of food in a discrete period of time where the individual experiences a sense of "loss of control" over eating. Binge episodes are followed by recurrent inappropriate compensatory behaviors designed to prevent weight gain, such as self-induced vomiting, misuse of laxatives, diuretics or other medications, fasting, or excessive exercising. The frequency criterion for these binge/purge episodes requires that they occur at least once per week for 3 months. Finally, self-evaluation is unduly influenced by body shape and weight.

11.3.2 Clinical Characteristics

The lifetime prevalence of BN in adult women based upon 3 population-based surveys has been estimated as 1.1–2.8 % and 0.5 % in males with the mean age of onset 19.7 years (Bushnell et al., 1990; Garfinkel et al., 1995; Hudson et al., 2007)

(see Table 11.1). Approximately 25 % of patients with BN have had AN in the past (Agras, Walsh, Fairburn, Wilson, & Kraemer, 2000).

There are many similarities between AN (particularly the ANBP subtype) and BN. The core psychopathology for both disorders, which initiates and maintains the ED, is a dysfunctional system for evaluating self-worth (Fairburn, Cooper, & Shafran, 2003). Instead of positive self-appraisal being based on the sum of personal qualities and achievements in multiple arenas (i.e., talents, academic aptitude, athletic prowess, work productivity, values, morals, quality of relationships with family, friends, coworkers), individuals with BN consistently judge themselves based on their weight, shape, and appearance. They believe that taking control of their eating, weight, and shape will lead to increased self-worth. Thus, restricting food intake and attempting to adhere to extreme dietary rules leads to binge eating which is further managed by engaging in compensatory behaviors (i.e., self-induced vomiting, laxative and/or diuretic use, excessive exercise). This cycle is often exacerbated by negative mood states that may predate the onset of BN or be the consequence of dietary restraint and compensatory behaviors.

Dieting behaviors usually precede the onset of BN, but in a significant subset of cases (37 %), binge eating comes first (Brewerton, Dansky, Kilpatrick, & O'Neil, 2000). Episodes of prolonged and severe calorie restriction promote binge eating (Keys, Brozek, Henschel, Mickelsen, & Longstreet, 1950). Self-induced vomiting, diuretic or laxative abuse, and extreme exercising evolve as management tactics designed to rid the body of excessive calories consumed during a binge. For most individuals, this behavior escalates into repetitive and severe cycles of binge eating and purging varying in frequency from once per week to several times per day.

Binge eating initially evolves either in response to a physiological drive to replace calories or as a way to manage negative affect but subsequently becomes a tool for self-soothing. The binge/purge cycle often functions as a coping strategy designed to help the individual distance from or manage negative affect states (e.g., loneliness, boredom, anger, fear, sadness, or frustration). There is some evidence that cycles of binge eating and purging may actually be biochemically reinforced through the release of neurochemicals, such as endorphins (Hawkes, 1992), and monoamine neurotransmitters, such as serotonin and dopamine (Brewerton, Lydiard, Laraia, Shook, & Ballenger, 1992; Jimerson, Lesem, Kaye, & Brewerton, 1992).

BN is a secretive and isolating disorder. Unlike AN, bulimic individuals recognize their abnormal behaviors. There is frequently a worsening of symptoms during times of stress. As the disorder progresses, a complex lifestyle can develop which is designed to protect the ED and is often accompanied by lying, feelings of shame, self-deprecating thoughts, labile and/or depressed mood, and low self-esteem. It is not uncommon for interpersonal relationships to become strained or abandoned.

11.3.3 Medical Complications

The medical complications of BN range from fairly benign transient symptoms, such as fatigue, bloating, and constipation, to chronic and/or life-threatening conditions, including hypokalemia (low potassium), cathartic colon, aspiration, impaired renal function, and cardiac arrest (Sansone & Sansone, 1994).

Binge eating alone (beyond the secondary effects of rapid, excessive caloric intake and obesity) rarely causes significant medical complications. Gastric rupture, the most serious complication, is uncommon (Sansone & Sansone, 1994). More often, patients describe nausea, abdominal pain and distension, prolonged digestion, and various rates of significant weight gain despite purging or extremes of exercise. Self-induced vomiting, the most common means of purging, is reported in more than 75 % of patients with BN (Fairburn, 1995). Self-induced vomiting can lead to a number of medical complications including dental erosion, enlarged salivary glands, and esophageal and pharyngeal complications because of repeated contact with gastric acids. Blood in the vomitus is an indication of upper gastrointestinal tears or erosion, which are serious complications of purging. Perforation of the upper digestive tract, esophagus, or stomach is an extremely rare but potentially lethal complication (McGilley & Pryor, 1998). Serious depletion of hydrogen chloride, potassium, sodium, and magnesium can occur because of the excessive loss of fluids during vomiting and laxative or diuretic abuse. Patients who complain of fatigue, muscle spasms, or heart palpitations may be experiencing transient episodes of electrolyte disturbance. Severe dehydration and electrolyte abnormalities can become life threatening. Paresthesias, tetany, seizures, or cardiac arrhythmias are potential metabolic complications that require acute care (Birmingham & Treasure, 2010) (see Chap. 15).

11.3.4 Assessment

The assessment phase of treatment for BN is quite similar to that described above for AN (specific BN screening tools are detailed elsewhere in this volume, see Chap. 14). In addition to a comprehensive clinical interview, patients should have a complete medical evaluation and lab tests to rule out electrolyte abnormalities, dehydration, or complications associated with purging or other compensatory behaviors.

There are high rates of psychiatric comorbidity in BN with 80 % of patients struggling with anxiety disorders, 70 % suffering from a mood disorder, 64 % with impulse control disorders, and 37 % with SUD (Hudson et al., 2007) (see Table 11.2). Additionally, personality disorders, especially avoidant, dependent, and borderline personality disorder (BPD), are evident in approximately 25 % of BN patients (Sansone et al., 2006). As a result, patients should be methodically screened for these co-occurring disorders and treatment plans should address both the ED and all other comorbid conditions (Dennis & Sansone, in press).

Treatment teams for bulimic individuals vary depending on the specific needs of the patient but may include a pediatrician, general practice or family medicine physician, psychiatrist, therapist, dietitian, skills group therapist, endocrinologist (for diabetic patients), and a gynecologist (for high-risk pregnant patients).

For most BN patients, outpatient treatment provides sufficient structure and support to recover from their ED. However, higher levels of care, including IOP, PHP, or residential, may be necessary if intractable binge eating and purging does not respond to aggressive treatment in a less restrictive environment (Yager et al., 2012). Inpatient hospitalization may be required if there are severe physiological or cardiac disturbances, psychiatric decompensation (e.g., suicidal gestures or persistent self-harm behaviors), or if other serious comorbid disorders are present that interfere with the treatment of BN [e.g., SUD, major depressive disorder (MDD)].

11.3.5 Psychological Treatments

There are currently three EBT for BN including cognitive behavioral therapy for bulimia nervosa (CBT-BN & CBT-Enhanced), interpersonal psychotherapy (IPT), and dialectical behavior therapy (DBT). Additionally, preliminary findings suggest that FBT for adolescents, and Integrative Cognitive–Affective Therapy (ICAT) for adults, may also be effective treatments for BN.

CBT for BN is a structured, symptom-focused intervention which targets the core psychopathology that initiates and maintains the ED. Treatment focuses on normalizing eating patterns (elimination of dietary restraint and increasing caloric intake); confronting dysfunctional thoughts around the *meaning* of weight, shape, and appearance; and the development of coping strategies, problem-solving skills, mood management techniques, and relapse prevention plans.

CBT for depression was modified for the treatment of BN in 1981 (Fairburn, 1981). Since then, CBT-BN has been extensively studied and numerous RCT have compared its effectiveness to medications and other psychotherapies. Although CBT-BN is considered "first line" treatment (Agras et al., 2000), up to 50 % of patients do not respond to this intervention (Fairburn et al., 2003). This prompted researchers to explore how CBT-BN could be broadened and made more effective for patients that were not helped with earlier versions. CBT-E (Enhanced) was specifically designed to address other common psychopathological processes that often co-occur with BN including low self-esteem, clinical perfectionism, mood intolerance, and interpersonal difficulties (Fairburn et al., 2003). Tentative results from a multisite study comparing CBT-BN and CBT-E suggest that the enhanced version of CBT may be more effective in treating BN patients that have marked additional psychopathology (Cooper & Fairburn, 2011) (see Chap. 24).

The second EBT for BN is interpersonal psychotherapy (IPT), a structured, non-interpretive, dynamic intervention, first designed for the treatment of depression (Klerman, Weissman, Rounseville, & Chevron, 1984) and then adapted for the treatment of ED (Fairburn, 1997). This approach suggests that individuals develop psychological symptoms in response to difficulties in their interpersonal

relationships, and the resolution of those difficulties will result in symptom amelioration. Treatment focuses on interpersonal interactions in four primary areas (1) unresolved grief and loss (the individual does not feel emotions, but may experience other symptoms), (2) role disputes (significant people in the person's life have different expectations about their relationship), (3) role transitions (difficulty coping with life transitions and role changes), and (4) interpersonal deficits (the individual lacks the skills to form and maintain good quality relationships). In RCT, IPT has been found to be as effective as CBT in the treatment of patients with BN and BED (Murphy, Straebler, Basdem, Cooper, & Fairburn, 2012).

Another cognitive-based intervention that has been found effective in the treatment of BN is DBT. This approach was originally developed to treat patients with BPD and has been successfully adapted for the treatment of BN and BED (Chen & Safer, 2010; Telch, Agras, & Linehan, 2000). DBT combines cognitive behavioral change strategies with mindfulness interventions to promote interpersonal effectiveness, distress tolerance, global self-regulation, and self-acceptance. This approach appears to have promise for the treatment of multidiagnostic (i.e., other comorbid psychiatric and personality disorders) ED patients (Federici, Wisniewski, & Ben-Porath, 2012) as well as patients with comorbid SUD (Courbasson, Nishikawa, & Dixon, 2012) (see Chap. 25).

FBT for AN has been adapted for the treatment of adolescent patients with BN (Le Grange, Crosby, Rathouz, & Leventhal, 2007). In a RCT comparing individual supportive therapy (SPT) for BN to FBT-BN, researchers found that by the end of treatment, those in the FBT group had higher remission and partial remission rates and demonstrated larger reductions in self-induced vomiting and other compensatory behaviors. However, subjects in the SPT group "caught up" to those in the FBT group and there were no significant differences between groups at 6-month follow-up. It should be noted that remission rates for both adolescent interventions were between 30 and 40 %, which is somewhat lower than the remission rates seen in CBT-BN. Further research should include a comparison of FBT-BN to CBT-BN in an adolescent population (see Chap. 26).

Finally, ICAT (Wonderlich et al., 2010) is an approach that utilizes components of CBT, IPT, and DBT for the treatment of bulimia nervosa. Treatment focuses not only on ED symptoms and behavioral change, but also on personality and temperament. ICAT specifically targets self-discrepancy (e.g., discrepancy between perceived self and ideal self), interpersonal insecurity, low self-esteem, negative affectivity, and ineffective interpersonal relationship styles. In a recent RCT comparing ICAT to CBT-E, study participants in both groups had significant improvements on all outcome measures and there were no statistically significant differences found between the two conditions at end of treatment or at 4-month follow-up (Wonderlich et al., 2013). Although further studies are necessary, ICAT appears to be a potentially effective intervention for individuals with bulimia nervosa.

11.3.6 Pharmacological Treatments

Over the past three decades, numerous studies have been conducted to determine the efficacy of medications (primarily antidepressants) in the treatment of BN. Results from a systematic review of 36 RCT found that both tricyclic antidepressants and the SSRI fluoxetine were effective in reducing bulimic episodes in a subset of BN patients (Aigner et al., 2011). In another recent meta-analysis, 9 of 11 BN pharmacotherapy studies yielded a statistically significant reduction in binge/purge frequency (Flament et al., 2012).

Fluoxetine, at a dose of 60 mg/day, is currently the only medication approved by the FDA for the treatment of BN and considered the "gold standard." However, end-of-treatment remission rates are relatively lower (between 20 and 35 %) than seen in psychotherapy trials for BN. Overall, CBT is more effective than medication alone in reducing binge eating and purging, but there is a slight benefit from the combination of both treatments for patients with comorbid major depression (Walsh et al., 1997).

11.4 Binge Eating Disorder

11.4.1 Diagnosis

With the publication of the DSM-5 (American Psychiatric Association, 2013), BED is officially an independent ED diagnosis. The criterion for BED includes recurrent episodes of binge eating that occur, on average, at least once a week for 3 months without the use of compensatory weight control methods. There is a reported sense of lack of control, which includes eating until uncomfortably full, eating more rapidly than normal, eating when not physically hungry, and eating in isolation. Binges are frequently accompanied by feelings of disgust, shame, or guilt (Wilson, 2011).

11.4.2 Clinical Characteristics

BED is the most common ED with lifetime prevalence rates of 3.5 % in females and 2.0 % in males in the USA with the mean age of onset 25.4 years (Hudson et al., 2007) (see Table 11.1). These rates are almost double those for both AN and BN combined. Retrospective data from adult samples and prospective studies in young children suggest that the risk for BED may be present well before the manifestation of the disorder (Tanofsky-Kraff et al., 2013).

There are several similarities between individuals with BED and those with BN. Both groups overvalue body shape and weight, engage in episode of binge eating, experience a sense of "loss of control" over eating, and have considerable comorbid psychiatric illnesses and personality disorders. However, there are distinct differences between these two disorders. Dieting usually predates binge eating

in BN, but in BED binge eating often precedes dieting behaviors (Grillo & Mashev, 2000). Although individuals with BED periodically engage in fasting, dieting, purging, or bouts of overexercising, they do not meet the frequency criteria associated with BN. In BN, binge eating is considered a consequence of severe dietary restraint (i.e., dieting, skipping meals, fasting). Individuals with BED report very little dietary restraint during binge episodes or between episodes.

There are also significant differences between obese binge eaters and obese individuals that do not have BED. First, obese binge eaters tend to ingest more calories than obese individuals that do not binge eat. Additionally, those with BED reported greater functional impairment and lower quality of life in the domains of physical health, emotional well-being, work, sexual life, and self-esteem and have greater psychiatric comorbidity than obese individuals without BED (Wonderlich, Gordon, Mitchell, Crosby, & Engel, 2009).

11.4.3 Medical Complications

BED is a psychiatric disorder and should not be confused with obesity, which is a medical condition. Support for the idea that BED predicts weight gain or negative medical complications and outcomes is both limited and mixed. However, there is good evidence to suggest that BED appears to be associated with increased health-care utilization (Wonderlich et al., 2009). An obese individual with BED may experience medical disorders such as hyperlipidemia (elevated lipids in the blood) and type 2 diabetes. This combination of obesity and BED may also confer a risk of metabolic syndrome (i.e., cluster of related risk factors for atherosclerotic cardiovascular disease, including abdominal obesity, dyslipidemia, hypertension, and abnormal glucose metabolism) above the risk attributable to obesity alone (Bulik, Sullivan, & Kendler, 2002; Hudson et al., 2010; Roehrig, Masheb, White, & Grilo, 2009) (see Chap. 15).

11.4.4 Assessment

Similar to AN and BN, the assessment of BED is best conducted by a multidisciplinary team. A thorough assessment should include a comprehensive physical exam and laboratory tests to identify any medical complications that may need immediate or ongoing attention. Both self-report questionnaires and clinical interviews conducted by trained personnel are used to identify individuals with BED (BED screening instruments are detailed elsewhere, see Chap. 14). Due to the significant prevalence of co-occurring disorders in BED, instruments to assess mood disorders, anxiety disorders, SUD, and personality disorders should also be administered to determine level of care and aid in the development of a comprehensive treatment plan (see Table 11.2).

11.4.5 Psychological Treatments

To date, treatments for BED include DBT, IPT, and CBT. CBT and IPT remain the treatments of choice for the full range of BED patients (Fairburn, 2008; Streigel-Moore et al., 2010). They produce the greatest degree of remission from binge eating as well as improvement in associated psychopathology such as depression (Wilson, 2011). A study by Telch and colleagues (2001) found that modified DBT for BED was more effective than a wait list control in eliminating binge eating.

Unfortunately, the cost of these specialized treatments, along with the limited availability of therapists who are trained to perform them, prompted researchers to investigate a mode of self-help based on CBT (CBT guided self-help or CBTgsh). They found that CBTgsh was equally as effective as IPT in eliminating binge eating (Wilson, Wilfley, Agras, & Bryson, 2010) (see Chap. 27). Numerous studies have evaluated the effects of differing combinations of CBT, CBTgsh, IPT, and behavioral weight loss (BWL). In a controlled treatment study of 205 BED patients, Wilson and colleagues (2010) compared CBTgsh with BWL and IPT. Posttreatment analyses revealed no differences among the three treatments on remission from binge eating. However, at 2-year follow-up, both CBTgsh and IPT were significantly superior to BWL in producing remission from binge eating. The BWL approach produced short-term weight loss, but long-term weight loss has yet to be demonstrated (Wilson et al., 2010). Research suggests that obese individuals with BED lose less weight than obese patients without BED in BWL treatments (Blaine & Rodman, 2007). In summary, the behavioral therapies currently offered are all associated with reductions in binge behavior. However, to date, these therapies do not result in more than minimal weight loss. The value and necessity of weight loss for BED sufferers who are obese remains of critical concern for many patients, as well as a source of controversy within the field of ED practitioners.

11.4.6 Pharmacological Treatment

There is growing evidence suggesting that pharmacotherapy may be beneficial for some patients with BED (McElroy, Guerdjikova, Mori, & O'Melia, 2012). For BED, RCT have been conducted using SSRI and serotonin norepinephrine reuptake inhibitors (SNRI), mood stabilizers, and antiobesity medications. Studies conducted using fluvoxamine (Hudson et al., 1998), sertraline (McElroy et al., 2000), fluoxetine (Arnold et al., 2002), and citalopram (McElroy et al., 2003) have all resulted in significant reductions in the frequency of binge eating episodes, modest decreases in BMI, and overall clinical improvement. An RCT using the anticonvulsant topiramate significantly reduced frequency of binge eating episodes, weight, and BMI (McElroy et al., 2007). However, it should be noted that topiramate has been associated with neurocognitive side effects and has not received approval from the FDA as a weight loss agent. In summary, a review by Bodell and Devlin (2010) serves to remind us that the long-term effectiveness of

pharmacotherapy with BED patients is still unknown. No placebo-controlled maintenance trials of patients in remission from BED have been published (McElroy et al., 2012). Studies to date have shown that pharmacotherapy for BED is still significantly less effective than CBT.

11.5 Other Specified Feeding and Eating Disorders

11.5.1 Diagnosis

The DSM-5 (American Psychiatric Association, 2013) ED work group proposed changes to reduce the reliance on EDNOS in clinical settings. Specifically, DSM-5 expanded the diagnostic criteria for AN and BN and moved BED out of the EDNOS category. BED is now formally recognized as the third diagnosable ED. Additionally, the residual category of ED that are clinically significant but do not meet the full criteria for AN, BN, or BED has been renamed OSFED. Individuals that display the following symptoms meet the diagnostic criteria for OSFED:

1. Atypical AN in which all criteria for AN are met except that the individuals' weight is within or above the normal range for height and age.
2. Subthreshold BN in which all criteria are met except the binge eating and compensatory behaviors occur on average less than once a week and/or for less than 3 months.
3. Subthreshold BED in which all criteria for BED are met except with lower frequency and/or limited duration.
4. Purging Disorder (PD) in which individuals purge (e.g., vomit, misuse laxatives, or other medications) without engaging in binge eating.
5. Night Eating Syndrome (NED) in which the individual eats excessive amounts of food after the evening meal or after awakening from sleep. The individual is aware and recalls the nighttime eating behavior.
6. Other Feeding or Eating Condition Not Elsewhere Classified is a category for cases that are clinically significant but do not meet criteria for formal ED diagnoses.

11.5.2 Clinical Characteristics

Historically, EDNOS has been the most prevalent ED diagnosis. A comprehensive literature search identified 125 studies completed from 1987 to 2007 (Thomas, Vartanian, & Brownell, 2009). Random effects analyses indicated that whereas EDNOS did not differ significantly from AN and BED on eating pathology or general psychopathology, BN exhibited greater eating and general psychopathology than EDNOS. Moderator analyses indicated that EDNOS groups who met all diagnostic criteria for AN except for amenorrhea did not differ from full syndrome cases. EDNOS groups who met all criteria for BN or BED except for binge

frequency did not differ significantly from full syndrome cases. Results suggest that EDNOS and now OSFED represent a set of disorders associated with significant impairment, often experiencing similar problems and levels of distress as those with AN or BN (Stice, Killen, Hayward, & Taylor, 1998).

In a study to track service consumptions in adult referrals to an ED service over a 3-year period, results found EDNOS to be the most common diagnostic group (42.8 %). There was no relationship between diagnosis and service needs, concluding that AN and BN were no more labor-intensive overall than EDNOS patients (Button, Benson, Nollett, & Palmer, 2005). A study comparing DSM-IV and DSM-5 ED criteria in a community sample demonstrated a significant reduction in the use of the EDNOS diagnosis as a result of removing BED (Keel, Brown, Holm-Denoma, & Bodell, 2011). It remains to be seen whether service consumption will be the same between OSFED and other ED diagnoses in the future.

11.5.3 Medical Complications

In all meaningful ways, individuals with EDNOS/OSFED are very similar to those with fully diagnosable ED and are at risk for the same medical complications individuals who restrict and/or binge and purge may experience. Electrolyte imbalance may cause cardiac complications and, occasionally, sudden death. Restrictors may experience low blood pressure, slower heart rate, and disruption of hormones. A study by Crow and colleagues (2009) found that EDNOS mortality rates are similar to rates for AN, indicating the potential severity of this disorder.

11.5.4 Assessment and Treatment

Many people with a diagnosable eating disturbance fall through the cracks because health-care professionals are less accustomed to looking for the "subclinical" or "atypical" signs that would indicate OSFED. This is especially true when someone is of a normal weight, male, or presenting for treatment during or beyond midlife. Unfortunately, this prevents patients from receiving an adequate diagnosis and treatment. It is critical to assess OSFED with the same recommended ED-specific assessment tools as those utilized for AN, BN, and BED. It is also imperative to apply the same psychological and pharmacological treatment strategies outlined above as these patients often present with similar symptom severity and comorbidity.

11.6 Risk Factors and Comorbidity in Eating Disorders

The biopsychosocial model of ED suggests that there are biological, psychological, interpersonal, familial, and sociocultural factors that place individuals at risk for the development of ED (Johnson & Connors, 1987). Contrary to early theories of

causality, eating disorders are not "disorders of choice." Decades of research suggest that ED are serious life-threatening psychiatric illnesses that are biogenetically mediated (Kaye, 2009). Twin studies in AN and BN, designed to distinguish genetic from environmental effects, report a 50–80 % *genetic* contribution to liability (Bulik et al., 2006; Kendler, MacLean, Neale, Kessler, & Eaves, 1991; Klump, McGue, & Iacono, 2001). Similar heritability estimates have been found in bipolar disorder and schizophrenia, which are both considered genetically influenced disorders. Additionally, AN and BN are more prevalent in families where other relatives have an ED. In other words, AN, BN, and subthreshold forms of ED are cross-transmitted in families (Lilenfeld et al., 1998; Strober, Freeman, Lampert, Diamond, & Kaye, 2000).

Over the past several decades, dozens of potential risk factors have been proposed, but very few have been subjected to rigorous longitudinal and cross-sectional study. However, eight salient factors have been identified using the Kraemer et al. (1997) risk model, including gender, race and ethnicity, higher BMI, childhood eating problems, sexual abuse, psychiatric comorbidity, low self-esteem, and dieting and weight concerns (Jacobi, Hayward, de Zwaan, Kraemer, & Agras, 2004). Females are 1.75–3.0 times more likely than males to develop AN, BN, and BED (Hudson et al., 2007). As far as race and ethnicity are concerned, Native Americans have the highest rates of ED, followed by equal rates for whites and Hispanics with lower rates found in Asians and blacks (Striegel-Moore & Smolak, 1996).

Childhood obesity (higher BMI) was found to be a variable risk factor in the development of BN (Fairburn, Welsh, Doll, Davies, & O'Connor, 1997). Gastrointestinal, digestive problems, picky eating, and struggles around meals in childhood have been identified as a variable risk factor in the development of AN, while eating too little in childhood is predictive of future BN (Kotler, Cohen, Davis, Pine, & Walsh, 2001).

Several studies have found elevated rates of sexual and/or physical abuse or neglect in individuals with bulimic symptoms including ANBP, BN (Dansky, Brewerton, & Kilpatrick, 2000), and BED (Johnson, Cohen, Kasen, & Brook, 2002). However, premorbidly high rates of abuse and neglect have been found in other psychiatric disorders which suggests that it is a nonspecific, variable risk factor in the development of ANBP, BN, and BED. The link between trauma and ED has not only been established in adult women but also in children, adolescents, boys, and men (Brewerton, 2007) (see Chap. 17). Further research is necessary to determine the role of abuse and/or neglect in the development of AN.

Research on psychiatric comorbidity has identified numerous correlates but few risk factors. Obsessive–compulsive disorder (OCD) has been found as a unique predictor of subsequent AN (Buckner, Silgado, & Lewinsohn, 2010). In BN, premorbid anxiety and negative affectivity are considered nonspecific, variable risk factors, as they are not unique to the development of ED but are also present prior to the onset of other psychiatric disorders including SUD and affective disorders (Ingram & Price, 2000). Both full and partial syndrome posttraumatic

stress disorder (PTSD) are associated with the emergence of BN and bulimic symptoms and may also be a nonspecific risk factor (Brewerton, 2007).

Similarly, low self-esteem, negative self-evaluation, and ineffectiveness in BN (Button, Sonuga-Barke, Davies, & Thompson, 1996) and low self-esteem, high body concerns, escape-avoidance coping strategies, and low perceived social support in BED (Ghadri & Scott, 2001) have been found to predate the ED and are classified as nonspecific, variable risk factors. Unfortunately, there are no longitudinal studies to date that suggest these factors precede the onset of AN.

Finally, dieting has long been considered a "gateway" behavior in the development of ED. There is a strong relationship between premorbid dieting and ANBP and BN, but less evidence to support this notion in AN or BED. In fact there is a substantial subgroup of BED patients (e.g., 35 %) that report engaging in binge eating prior to the onset of dieting behaviors (Grillo & Mashev, 2000). Although there is preliminary data to suggest that weight concerns, negative body image, and dieting may be risk factors in the development of AN, further research is needed to clarify the relationship between these variables (Jacobi, Morris, & de Zwaan, 2007).

Family history of psychopathology is not considered a formal risk factor for the development of ED; however, it is important to note the results of a large body of cross-sectional research examining the role of psychiatric disorders in first-degree relatives of individuals with ED. Family studies in AN reveal elevated rates of ED (AN and BN), mood disorders, anxiety disorders, and obsessive–compulsive personality disorder (OCPD). Similarly, there were substantial rates of ED, mood disorders, anxiety disorders, SUD, and BPD found among relatives of BN patients. Elevated rates of ED, mood disorders, and anxiety disorder have also been reported in first-degree relatives of individuals with BED (Lilenfeld, Ringham, Kalarchian, & Marcus, 2008) (see Chap. 5).

11.7 Psychiatric Comorbidity

Two large national comorbidity replication studies have been completed in the last decade that have identified prevalence rates, clinical correlates, and patterns of comorbidity with other mental disorders in an adult ED sample ($n = 2{,}980$; age 18 and older; Hudson et al., 2007) and in an adolescent sample ($n = 10{,}123$ adolescents, age 13–18; Swanson et al., 2011) (see Table 11.1). In the adult sample, over 56 % of respondents with AN, 95 % with BN, and 79 % with BED met the criteria for at least one additional psychiatric condition including mood, anxiety, SUD, or impulse control disorders. Similar results were found in the adolescent sample with 55 % of AN, 88 % of BN, and 84 % of BED respondents meeting the DSM-IV criteria for mood, anxiety, SUD, or impulse control disorders.

The most common anxiety disorders found in both the adult and adolescent sample of AN, BN, and BED respondents were specific phobia, social phobia, and PTSD. The prevalence of separation anxiety disorder in adolescents was also significant in all three major ED categories. High rates of MDD were found in

both samples across all ED, with bipolar disorders being more common in BN respondents.

In the adult comorbidity study (Hudson et al., 2007), attention-deficit/hyperactivity disorder (ADHD) and oppositional-defiant disorder (ODD) were prevalent across each of the ED diagnostic groups. Conduct disorder (CD) was more prevalent in BN and BED. A different pattern of comorbidity emerged in the adolescent sample (Swanson et al., 2011) with ODD the most prevalent impulse control disorder found in AN and BED and CD the most common in BN (see Table 11.2).

There are high rates of SUD in ED. Approximately 50 % of individuals with ED are abusing alcohol, and/or illicit, prescription, or over-the-counter drugs, or Internet supplements (The National Center on Addiction and Substance Abuse (CASA), 2003). The highest rates of alcohol use disorder (AUD) have been reported in BN (34 %); however, 25 % of those with AN and 21 % with BED have abused or are dependent on alcohol (Hudson et al., 2007). Similar results were found with illicit drug abuse and dependence (26 % BN, 19 % BED, and 18 % AN). Importantly, researchers have found that rates of SUD vary greatly across AN subtypes with the ANR group reporting the lowest rates of AUD (14 %) and drug abuse (6 %) and the ANBP group reporting rates similar to individuals with BN (AUD 36 %, drug abuse 32 %) (Root et al., 2010).

Sansone et al. (2006) conducted a comprehensive review of empirical studies on the prevalence of personality disorders in subjects with ED. Although this area of study is plagued by small sample sizes and biased samples (e.g., inpatient vs. outpatient samples), important patterns of association emerged. In subjects with ANR, cluster C personality disorders were the most common including OCPD and avoidant personality disorder. BPD was the most common personality disorder found in both ANBP and BN, but no specific pattern of personality pathology emerged for BED. However, one large multisite study found that approximately 30 % of all ED patients met the diagnostic criteria for OCPD and there was no significant difference in comorbidity frequencies between ED subtypes. These researchers conclude that OCPD may be a core behavioral feature that increases one's vulnerability to the development of an ED (Halmi et al., 2005).

Conclusions

In this chapter, we have provided the reader with a basic overview of diagnosis, clinical characteristics, medical complications, assessment, and EBT approaches for individuals with AN, BN, and BED. It was specifically aimed at providing the substance abuse treatment provider with a more comprehensive understanding of the dynamics and clinical presentation of individuals with ED. The following companion chapter, entitled *Introduction to Substance Use Disorders for the Eating Disorder Specialist*, will acquaint the eating disorder specialist with the diagnosis, assessment, and treatment of SUD. Clinical characteristics, medical complications, and EBT for each psychoactive substance class will be described with special attention to over-the-counter, Internet, and prescription medications frequently abused by individuals with ED. Together, these chapters

are designed to begin the cross-training process and prepare the reader to more fully assimilate and apply the content of the remaining chapters in this textbook.

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Introduction to Substance Use Disorders for the Eating Disorder Specialist

12

Amy Baker Dennis and Tamara Pryor

Abstract

Substance use disorders and eating disorders co-occur frequently; however, currently, there are no evidence-based treatments to guide the practitioner faced with this comorbid condition. Very few eating disorder programs have incorporated substance abuse protocols into their programs, and likewise, few substance abuse programs can effectively treat the patient with a serious eating disorder. Consequently, clinicians presented with these patients tend to focus on their area of specialty without addressing directly the other comorbid condition. Inadvertently, this can prolong the patient's suffering as they vacillate between their substance use disorder and their eating disorder. The intention of this chapter is to provide a brief overview of substance use disorders for students, clinicians, and researchers in the health and mental health field. It was specifically designed for the eating disorder specialist that has limited knowledge of the psychoactive properties of drugs of abuse, the clinical characteristics of individuals with alcohol and drug abuse problems, the philosophy and/or vernacular of abstinence-based models (e.g., Alcoholics Anonymous [AA], Narcotics Anonymous [NA], or Cocaine Anonymous [CA]), and other evidence-based models/approaches for the treatment of substance use disorders.

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12.1 Introduction

Substance use disorders (SUD) and eating disorders (ED) co-occur frequently; however, currently, there are no evidence-based treatments to guide the practitioner faced with this comorbid condition. Given the high rates of co-occurrence and the complex nature of these disorders, it is surprising to note that most ED clinicians and treatment programs have not incorporated SUD protocols into their practices. Likewise, most substance abuse specialists are not adequately trained in the diagnosis, assessment, and treatment of ED, and very few SUD treatment programs provide comprehensive, integrated services for these dually diagnosed patients. Consequently, clinicians presented with these patients tend to focus on their area of specialty without addressing directly the other comorbid condition. Inadvertently, this can prolong the patient's suffering as they vacillate between their SUD and their ED. Additionally, outpatient clinicians needing to refer patients to a higher level of care or parents seeking treatment for their loved one with both disorders find locating providers or programs that effectively treat both disorders complicated and confusing. The intention of this chapter is to provide a brief overview of SUD for students, clinicians, and researchers in the health and mental health field. It was specifically designed for the ED specialist that has limited knowledge of the psychoactive properties of drugs of abuse, the clinical characteristics of individuals with SUD, the philosophy and/or vernacular of abstinence-based models, and other evidence-based models/approaches for the treatment of SUD. Chapter 11 provides a similar review of ED for the substance abuse specialist. Together, these two chapters are designed to provide a foundation for the reader who is interested in working with individuals who struggle with both ED and SUD. Information on developing a comprehensive, integrated program for these two disorders is discussed in Chap. 21. It is vital that treatment providers in both fields acquire knowledge of the other and that research findings and empirically supported interventions from both specialties be incorporated to create an integrated approach to treatment and recovery (Dennis & Helfman, 2010).

This chapter begins with a discussion of the DSM-5 (American Psychiatric Association, 2013) diagnostic criteria for SUD and a brief discussion of prevalence, clinical characteristics, and medical complications for the following SUD: (1) alcohol; (2) cannabis; (3) sedative, hypnotic, and anxiolytics; (4) stimulants; (5) hallucinogens; (6) opioids; and (7) substances that are frequently used and/or abused by individuals with ED. This will be followed by a definition of recovery, assessment strategies, a review of patient placement criteria (i.e., levels of care),

and psychological and pharmacological interventions for SUD. The chapter will conclude with an examination of the risk factors associated with the development of SUD and a review of the most common psychiatric comorbidities found in individuals with alcohol and/or drug problems.

12.2 Substance Use Disorders

Substance use disorders (SUD) are characterized by a series of cognitive, behavioral, and physiological symptoms that indicate an individual is continuing to use substances without regard to the consequences of repeated use. SUD include ten basic categories: alcohol, caffeine, cannabis, hallucinogens, inhalants, opioids, sedative-hypnotics or anxiolytics, stimulants, and tobacco.

With the publication of DSM-5 (American Psychiatric Association, 2013), a few significant changes have occurred that impact how substance abuse problems are diagnosed. First, the categories of substance abuse and dependence have been collapsed. Second, each diagnosis requires an index of severity (i.e., mild, moderate, or severe). Third, criterion has been grouped into four categories: (1) impaired control, (2) social impairment, (3) risky behavior, and (4) pharmacological criteria. Impaired control implies that the individual is using larger amounts of the substance over longer periods of time than intended; has a desire to reduce or control use but has been unsuccessful; spends a considerable amount of time procuring, using, and recovering from the effects of the substance; and has significant urges to use (craving). Social impairment includes an inability to fulfill role obligations at work, home, or school; significant social or interpersonal problems; and withdrawal from previous relationships, activities, or hobbies in order to use the substance. Risky behaviors include use of the substance in physically hazardous situations and continued use even when faced with persistent physical or psychological problems. Pharmacological criteria include tolerance (requiring increased amounts of the substance to achieve the desired effect) and withdrawal (a significantly unpleasant group of symptoms that occur upon abrupt discontinuation of the substance). Neither of the pharmacological criteria (tolerance or withdrawal) is necessary to make a diagnosis of SUD. Finally, to receive a diagnosis of "mild" SUD, the number of endorsed symptoms has been increased from one to two, and symptoms must lead to clinically significant impairment or distress.

12.3 Alcohol Use Disorders

12.3.1 Prevalence

In 2011, according to the National Survey on Drug Use and Health (SAMHSA, 2012), approximately 52 % of Americans (133.4 million) age 12 and older reported being current drinkers (see Table 12.1). Among that group, a majority was male (57 %). However, the rates of alcohol *use* among youth aged 12–17 were similar for

Table 12.1 2011 Prevalence of substance use and dependence among Americans aged 12 and older

Substance	Use (past month)	Abuse/dependence
Alcohol	133.4	16.7
Illicit drugs	22.5	6.5
Marijuana	18.1	4.2
Psychotherapeutics	6.1	1.8
Cocaine	1.4	0.8
Hallucinogens	1.0	0.3
Inhalants	0.6	0.1
Heroin	0.3	0.4

Adapted from SAMHSA (2012)
Note: Statistics reported in *millions*

both males and females (13 %). The highest rates of alcohol consumption were found in young adults aged 18–25 with approximately 58 % of females and 63 % of males reporting current drinking.

Approximately 16.7 million people (6.5 % of the population age 12 or older) have an alcohol use disorder (AUD) (SAMHSA, 2012). It is estimated that 5 % of adolescents 12–17 years of age and 9 % of adults (18 and older) have AUD. Adult men have higher rates of AUD (12 %) than adult women (5 %), with the highest rates seen in individuals between the ages of 17 and 29 (16 %) and the lowest rates seen in individuals 65 and older (2 %) (American Psychiatric Association, 2013).

12.3.2 Clinical Characteristics

Individuals with AUD have complex and diverse clinical presentations. However, for many individuals, alcohol use and intoxication begins in adolescence. The usual age of first drinking is 15, with the heaviest drinking occurring between age 18 and 22 (Schuckit, 2006). This age pattern is similar between the general population and those who later develop AUD. However, one study found that early-onset drinking (prior to age 15) increased the risk of developing AUD by two to three times compared to individuals that started drinking after age 19 (DeWit, Adlaf, Offord, & Ogbourne, 2000). Early-onset AUD is associated with a family history of alcohol dependence and found in adolescents that have preexisting personality traits of behavioral disinhibition (i.e., an inability to inhibit behavioral impulses, sensation seeking, unconventionality, and rebelliousness) and negative emotionality (i.e., extreme sadness, fear, worry, or anger leading to feelings of isolation, suspiciousness, and interpersonal hostility) (Hicks, Durbin, Blonigen, Iacono, & McGue, 2012). By age 18, more than 60 % of adolescents have experienced drunkenness, and approximately 19 % of individuals between the age of 17 and 20 reported driving under the influence (SAMHSA, 2012).

Alcohol *abuse* often begins in the early to mid-20s with significant signs of AUD apparent in a majority of individuals by the late 30s. In the early stages, daily drinking or frequent binge drinking episodes are common. Drinkers often report a sense of euphoria (“buzz”) after the first few drinks. With continued heavy

drinking, tolerance develops and the individual must increase their consumption to achieve the desired effect. Craving (a strong desire to use the substance) develops and is often triggered by environmental cues (i.e., time of day, bar scenes, parties, celebrations) and/or a need to "self-medicate" or alter negative internal mood states (i.e., life stress, interpersonal conflicts, fatigue, boredom, anxiety, depression). Frequent intoxication can lead to periods of amnesia (blackouts) and debilitating hangovers. Withdrawal symptoms (i.e., tachycardia, psychomotor agitation, insomnia, nausea, diaphoresis, anxiety) often appear between 4 and 12 h after a reduction in alcohol ingestion and often compel the individual to continue consumption to avoid or relieve these unpleasant side effects.

As the illness progresses, occupational, legal, social, psychological, interpersonal, and medical problems become evident. Once a pattern of chronic use develops, the individual becomes increasingly preoccupied with procuring and consuming. Work performance and/or productivity may suffer from drinking on the job or as the result of missed work due to recovering from the effects of alcohol. Legal problems including traffic citations for driving while impaired, arrests due to public intoxication, and domestic violence are common. Daily life maintenance tasks, personal hygiene, and childcare responsibilities may be neglected. Social engagements that do not include alcohol may be avoided, and previously enjoyable activities or hobbies are often abandoned. Profound changes in mood (e.g., depression, anxiety, irritability, aggression, hostility, lability) frequently impact interpersonal functioning and can lead to social withdrawal, violent outbursts and abuse, or suicidal ideations or attempts. Chronic alcohol abuse is associated with significant physical problems (e.g., cirrhosis, ulcers, pancreatitis, cognitive impairment) and may lead to emergency room visits or hospitalizations. Finally, it is not uncommon for individuals with AUD to have other comorbid SUD. Sometimes, alcohol may be used to reduce or eliminate the negative side effects of other drugs (e.g., anxiety, psychomotor agitation, or insomnia from stimulants) or as a substitute when the drug of choice is not readily available.

AUD is a chronic relapsing disorder. Some individuals may have periods of remission (become abstinent) after a crisis (e.g., threat of divorce, loss of a job, arrest, car accident) or a medical emergency (e.g., bleeding ulcers, pancreatitis). However, frequently, these periods of abstinence are followed by controlled or non-problematic drinking but eventually result in the resumption of alcohol abuse and the reemergence of all of the associated consequences.

12.3.3 Medical Complications

Alcohol is considered a central nervous system depressant, and heavy use can have an impact on every organ system in the body. Cardiovascular problems include high blood pressure and an increased risk of an enlarged heart, arrhythmias, heart failure, and stroke. Chronic drinking can also increase LDL cholesterol and increase the risk of cardiomyopathy. Circulatory problems such as anemia (abnormally low number of oxygen carrying red blood cells) can trigger symptoms of fatigue,

light-headedness, and shortness of breath. Numerous gastrointestinal problems can result from alcohol abuse including digestive problems (gastritis, stomach, and esophageal ulcers), pancreatitis, alcohol hepatitis, and cirrhosis. The nutritional status of the individual with moderate to severe AUD may be significantly compromised. Chronic heavy drinking is associated with malnutrition, vitamin deficiencies, and impaired absorption, metabolism, and utilization (See Chap. 23). The neuroskeletal system can also be disrupted, as excessive alcohol use can interfere with the production of new bone and can lead to a reduction in bone density (osteopenia or osteoporosis) and an increased risk for fractures. AUD can affect the nervous system causing peripheral neuropathy (numbness and pain in the hands and feet), cognitive impairment (problems with short-term memory, learning, abstraction, and problem solving), and mild anterograde amnesia (blackouts) or severe anterograde amnesia (Wernicke–Korsakoff syndrome) (See Chap. 15).

12.4 Cannabis Use Disorder

Cannabis is the scientific term for the psychoactive substance derived from the plant Cannabis sativa. The chemical compound it contains that produces the subjective "high" is delta-9-tetrahydrocannabinol (THC) (Budney, Vandrey, & Fearer, 2011). Cannabis is the most frequently used illegal substance worldwide, with approximately 4 % of the adult world population (162 million people) using cannabis annually and 0.6 % (22.5 million) using cannabis daily (United Nations Office on Drugs and Crime, 2006). During the last several decades, over 200 slang terms have been used to describe cannabis including marijuana, Mary Jane, weed, pot, grass, dope, and ganja.

Epidemiological, laboratory, and clinical studies have demonstrated the existence, increasing prevalence, and clinical significance of cannabis use disorders. In 1999, the Institute of Medicine released a report describing the potential negative effects of cannabis including addiction, but also provided a clear statement regarding its potential medical benefits. Currently, in the USA, cannabis remains classified as a *Schedule 1 Substance* (i.e., a substance with high abuse potential that has no accepted medical uses or accepted standards for safe use under medical supervision). However, numerous states have passed laws "legalizing" the medical use of cannabis. In 2012, 66 % of voters approved the legalization of cannabis in Colorado where there are currently more dispensaries for marijuana-infused products (MIP) than liquor stores, Starbucks, or public schools (Osher, 2010).

Controversy regarding its legal status, addictive potential, health consequences, and medical use has pervaded the lay and scientific communities for decades. Ambivalence and debate will likely continue into the foreseeable future.

12.4.1 Prevalence

In 2011, approximately 18.1 million Americans aged 12 and older were current users of cannabis, and it is used by 80.5 % of all illicit drug users. Additionally, 2.6 million persons aged 12 or older used cannabis for the first time within the past year (approximately 7,200 new users every day) (SAMHSA, 2012) (see Table 12.1). The average age of first use among persons aged 12–49 was 17.5 years. Also in 2011, the number of cannabis users in the USA increased from 17.4 million to 18.1 million, and it has the highest abuse rates of any psychoactive substance.

12.4.2 Clinical Characteristics

Cannabis is sold and administered in preparations that vary in potency. The most common preparation is the dried plant form and ranges from 1 to 15 % THC concentration. Hashish refers to the *resin* of the plant which typically contains 10–20 % THC, but may reach as high as 60 % THC. In most instances, cannabis is burned and the smoke inhaled. Cannabis is also dissolved and ingested in the form of candy and baked goods. The cannabis "high" typically occurs within 1 min, reaches a peak in 15–30 min, and persists for up to 4 h (Budney et al., 2011). Subjectively, the user feels an initial euphoric effect characterized by a sense of relaxation. Perception is altered such that time seems to slow and many users report an increased ability to hear, visualize, and appreciate sights and sounds such as artwork, movies, and music. Feelings of anxiety, fear, and paranoia have also been reported in novice users or following the inhalation or ingestion of a higher than usual dose.

Cannabis use is less likely to lead to physical dependence than most other common illicit drugs. In the USA, it is estimated that 9 % of those who try cannabis become dependent compared to 17 % who try cocaine and 23 % who try heroin (Anthony, 2006). Risk of dependence is greater for those who use cannabis more frequently, for those who start using at an earlier age, and for those who have a family history of SUD (Copeland & Swift, 2009). Studies of chronic cannabis users suggest that sustained use may impair attention, memory, and complex cognitive abilities such as problem solving and mental flexibility (Solowij et al., 2002). Abrupt cessation of daily or near-daily cannabis use results in the onset of a cannabis withdrawal syndrome. Common symptoms include anger, anxiety, irritability, decreased appetite or weight loss, restlessness, sleep difficulty, and depressed mood. Withdrawal symptoms generally occur within the first 24 h and last approximately 1–2 weeks (Budney et al., 2011).

In an effort to more definitively understand the physiological and psychological appeal of cannabis, a number of neurobiological studies have been conducted which focus on the effects of THC in brain areas and systems associated with reward, reinforcement, and addiction. THC appears to enhance dopamine (DA) neuronal firing and synaptic DA levels in the reward pathway of the brain. Congruent with these findings, abrupt cessation of chronic THC exposure decreases DA. This effect

has been linked to the dysphoric effects associated with withdrawal from drugs such as alcohol, opiates, and cocaine (Gardner, 2005).

12.4.3 Medical Complications

It is uncommon to require medical care for acute toxicity although potential side effects include drowsiness, dizziness, tachycardia, dysphoria, and on rare occasions, visual hallucinations and drug-induced psychosis. Although rare, case reports have been published suggesting that the acute cardiovascular effects of cannabis can contribute to cardiac-related fatalities (Budney et al., 2011).

12.5 Sedative, Hypnotic, or Other Anxiolytic Use Disorder

This classification of drugs includes benzodiazepines, nonbenzodiazepines, and barbiturates. Like alcohol, these agents are central nervous system depressants. At lower doses, these drugs reduce anxiety and promote sedation; however at high doses, they can produce stupor, amnesia, coma, and death. These drugs can be obtained by prescription or illegally. Both individuals who are prescribed these medications and those who use them recreationally can develop a SUD.

Benzodiazepines. The most common sedative-hypnotic drugs in this category include diazepam (Valium), oxazepam (Serax), clonazepam (Klonopin), lorazepam (Ativan), alprazolam (Xanax), and temazepam (Restoril). These medications are primarily used as muscle relaxants, anticonvulsants, short-term antianxiety agents, sleep aides, and in alcohol withdrawal.

Nonbenzodiazepine ("Z-drugs"). The pharmacodynamics of this class of sedative-hypnotic drugs is nearly the same as benzodiazepine and therefore has similar benefits, side effect profiles, and risks. They have entirely different chemical structures than benzodiazepines and have demonstrated efficacy in treating sleep disorders. There are three primary groups of Z-drugs: (1) imidazopyridines, zolpidem (Ambien, Intermezzo, Stilnox); (2) cyclopyrrolones, zopiclone (Imovane, Zimovane, Imrest) and eszopiclone (Lunesta); and (3) pyrazolopyrimidines, zaleplon (Sonata, Starnoc). As compared to benzodiazepines or barbiturates, Z-drugs promote sleep at doses that produce lower levels of residual sedation and impairment in psychomotor or cognitive functioning, have less pronounced amnesic and antianxiety effects, and are generally considered safer, especially in over dosage. Additionally, they are less likely to produce physical dependence and addiction, although these issues can still occur (Touitou, 2007).

Barbiturates. Prior to the introduction of benzodiazepines, barbiturates were used as anxiolytics, sedative-hypnotic agents, and anticonvulsants. Ultrashort-acting barbiturates such as thiopental sodium (Pentothal) and thiamylal (Surital) are used intravenously to produce unconsciousness in surgical patients. Short-acting barbiturates (3–6 h) such as pentobarbital (Nembutal) and secobarbital (Seconal) are used as sleeping aids. Barbiturates with intermediate duration (6–12 h) include amobarbital (Amytal) and butabarbital sodium (Butisol) and are prescribed to relieve insomnia. The longer-lasting barbiturate phenobarbital (Solfoton) is used primarily to treat and prevent seizures. Slang terms for barbiturates include barbs, dolls, downers, goofballs, and sleepers (Hamid, El-Maliakh, & Vandeveir, 2005).

12.5.1 Prevalence

The 12-month prevalence of sedative, hypnotic, or anxiolytic use disorder is estimated to be 0.3 % among 12- to 17-year-olds and 0.2 % among adults age 18 years and older. The 12-month prevalence rate decreases as a function of age and is greatest among 18- to 29-year-olds (0.5 %). There are differences in the prevalence of sedative, hypnotic, or anxiolytic use disorder across racial/ethnic subgroups in the US population. For adolescents, the rates are greatest among whites (0.3 %). Among adults, the prevalence is greatest among Native Americans and Alaska Natives (0.8 %) (American Psychiatric Association, 2013).

12.5.2 Clinical Characteristics

Drugs in this classification have a potential for misuse if the drug has strong euphoric or mood-altering effects. Among the drugs in this classification, barbiturates produce the greatest pleasant mood alterations (Ciraulo & Knapp, 2011). Recreational users report that barbiturates promote relaxation, contentment, and euphoria. Sedative, hypnotics, and anxiolytics enhance the effects of the more sedating amino acid brain neurotransmitter, gamma-aminobutyric acid (GABA) (Breier & Paul, 1990).

These medications are primarily used in the treatment of insomnia, seizure disorders, and anxiety disorders. They are also prescribed to patients going through alcohol withdrawal and early in the course of treatment of an ED in an attempt to relieve extreme anxiety reported during efforts to return to normal eating behavior. However, even when carefully prescribed and monitored, they can often confound clinical presentations of mood and anxiety disorders at usual dosage ranges. Tolerance often develops rapidly, particularly to the sedating effects of these drugs. As a result, some patients increase their dosage levels (with or without prescriber direction)—even those patients without clinical or family histories of SUD.

12.5.3 Medical Complications

Compared with the other types of sedative-hypnotics, benzodiazepines are relatively safe in overdose but can be particularly dangerous when mixed with alcohol, barbiturates, opioids, or tricyclic antidepressants. The most common symptoms of benzodiazepine overdose include central nervous system (CNS) depression, ataxia, slurred speech, impaired balance, and respiratory depression. Toxic reactions of long-term abuse are characterized by decreased CNS, cardiac, and respiratory functioning; sedation; confusion; disorientation; and impaired memory. The clinical picture resembles severe alcohol intoxication. Although toxic reactions should not be taken lightly, deaths are relatively rare (<1 %) (Schuckit, Smith, Kramer, Danko, & Volpe, 2002).

Z-drugs are notable for producing side effects such as pronounced amnesia and more rarely hallucinations, especially when used in large doses. On rare occasions, these drugs can produce a fugue state where the patient reports sleepwalking or performing acts such as cooking a meal or driving a car while effectively unconscious and with no memory of the event upon waking (Stone, Zorick, & Tsuang, 2007). Daytime-related anxiety can also occur from chronic nightly usage (Fontaine, Beaudry, Le Morvan, Beauclair, & Chouinard, 1990).

The barbiturates produce depression of central nervous system activity that can range from mild sedation to coma. They are not selective in their actions, and their antianxiety effects are not separable from their other depressant effects. Respiratory depression may be a major contributing factor to death in cases of barbiturate overdose (Ciraulo & Knapp, 2011) (See Chap. 15).

12.6 Stimulant Use Disorders

Stimulants are a class of drugs that are prescribed for the treatment of narcolepsy, attention deficit hyperactivity disorder (ADD/ADHD), treatment-resistant depression, HIV-related neuropsychiatric symptoms, and obesity (Hill & Weiss, 2011). Central nervous stimulants activate the psychological reward system in the brain and increase the release of dopamine (DA) resulting in pleasurable effects. Prescription stimulants, including amphetamines (e.g., Dexedrine and Adderall), methamphetamine (e.g., Desoxyn) and methylphenidate (e.g., Ritalin and Concerta), lisdexamfetamine dimesylate (e.g., Vyvanse), and their illegal derivatives including cocaine (also known as coke, coca, snow, flake, or blow), crack cocaine (also known as rock, iron, base, work, cavvy), powder or crystal methamphetamine (also known as meth, crystal, ice, glass, crank, tina), and synthetic cathinones/mephedrone (also known as bath salts, ivory wave, purple wave, bliss, vanilla sky, bloom, red dove, Scarface), are frequently used and abused recreationally by individuals that are not being treated for the above conditions.

Historically, there have been four high-risk groups that have used stimulants to increase energy, improve performance, or lose weight: the military, students, athletes (Hill & Weiss, 2011), and dieters. Since World War II, military forces

have been prescribed stimulants to increase performance and reduce combat fatigue. Nonmedical use of stimulants by students to increase energy levels, decrease the need for sleep, and assist with studying for prolonged periods of time is common. Stimulants and over-the-counter amphetamine "look-alikes" (e.g., caffeine, ephedra, bitter orange, modafinil and adrafinil) are routinely used by collegiate and professional athletes to increase performance, concentration, and reduce weight (McDuff & Baron, 2005). However, individuals in any profession that requires prolonged periods of concentration (e.g., pilots, air traffic controllers, long haul truck drivers) are at risk for stimulant abuse. Finally, both prescription (i.e., ADHD medications and antiobesity drugs) and illicit stimulants (i.e., cocaine, methamphetamine) are used to promote weight loss in dieters. Phentermine, a noradrenergic sympathetic amine, approved by the FDA, is the most widely prescribed medication for the short-term treatment of obesity in the USA. However, there is a high potential for abuse with these drugs (Cochrane, Malcolm, & Brewerton, 1998; Hendricks & Greenway, 2011; Jeffers, Benotsch, & Koester, 2013).

12.6.1 Prevalence

According to the National Survey on Drug Use and Health (SAMHSA, 2012), the prevalence rates for stimulant use for persons aged 12 and older are as follows: nonmedical users of stimulants, 0.4 % or 970,000; cocaine, 0.5 % or 1.4 million; and methamphetamine, 0.2 % or 439,000. Males are more likely than females to use stimulants, and persons between the ages of 18 and 25 have the highest use rates (SAMHSA, 2012). Illicit use of prescription stimulants has become a serious issue on college campuses. It appears that the primary motivation for nonmedical use of stimulants is a belief that these medications will improve academic performance (Teter, McCabe, Cranford, Boyd, & Guthrie, 2005). One study found that 8.1 % of undergraduate students attending a large public Midwestern university reported lifetime illicit use of prescription stimulants, which exceeded the number of students on campus who reported medical use of stimulants for ADHD (McCabe, Teter, & Boyd, 2006). They also identified several risk factors for illicit use of prescription stimulants which included being white, male, a member of a fraternity or sorority, Jewish, having a lower grade point average, and perfectionist personality traits (McCabe et al., 2006). Likewise, Tuttle, Scheurichn and Ranseen (2010) found that 10.1 % of medical students in a public medical college reported using nonmedical prescription stimulants to enhance their performance.

12.6.2 Clinical Characteristics

The clinical effects of stimulant use vary depending on the frequency of use patterns (episodic vs. chronic use), potency of the drug, dosage, and route of administration. In low doses, stimulants increase the libido, alertness,

concentration, and energy and reduce appetite and fatigue. In higher doses, they can cause euphoria, mania, insomnia, anxiety, aggressive behaviors, or stimulant psychosis (e.g., hallucinations, delusions, and thought disorders). Chronic use of stimulants often follows a binge-abstinence pattern (taking the drug repeatedly within a relatively short period of time in escalating doses). For example, cocaine binges can last up to 12 h with methamphetamine binges lasting several days (Ciccarone, 2011). Following the binge, the user often experiences psychological and behavioral withdrawal symptoms (referred to as a "crash") including hypersomnia, depression, lethargy, and powerful cravings.

Prescription stimulants are primarily orally administered; however, some non-medical abusers crush the tablets and inhale (snort) them or dissolve them in water and inject them into the blood stream. Likewise, cocaine is inhaled or injected. Crack is a freebase form of cocaine that is smoked. There are several common routes of administration with methamphetamine including intravenous injection, smoking (i.e., vaporizing it to inhale the fumes, not burning it to inhale the smoke), snorting, and the insertion of suppositories in the anal or vaginal track. "Bath salts" can be swallowed, snorted, smoked, or injected. Heavy use is clearly related to dependency, but smoking and/or injecting stimulants (as opposed to ingesting or snorting) are associated with an increased likelihood of escalating tolerance and addiction (Ciccarone, 2011).

Stimulant abuse is also associated with the abuse of other psychoactive substances including heroin (e.g., cocaine combined with heroin and injected, "speedballs"), tobacco, alcohol, and marijuana. Additionally, it is not uncommon for stimulant abusers to use benzodiazepines or other sedative-hypnotics to manage bouts of anxiety, restlessness, or insomnia. Finally, more than 1/3 of individuals with stimulant use disorder have a lifetime mood or anxiety disorder, and at least 13 % report one or more symptoms of psychosis (Sara et al., 2012). Stimulant use disorder may also be associated with other mental disorders including posttraumatic stress disorder, antisocial personality disorder, and gambling disorder (American Psychiatric Association, 2013).

12.6.3 Medical Complications

Cocaine, amphetamine, and methamphetamine abuse can all be marked by toxic cardiovascular complications including, tachycardia, hypertension, and myocardial infarction. Though longitudinal data linking stimulant abuse to cardiovascular disease is lacking, a study by Yeo et al. (2007) found that chronic stimulant dependence heightens the risk of artherosclerotic heart disease, cardiomyopathies, and sudden death from arrhythmias. Neurological complications of abuse include seizures and a risk for hemorrhagic strokes. Neurobehavioral complications include memory/learning impairment, anxiety, paranoia, psychosis, and anorexia.

Cardiac complications specific to cocaine abuse include an increased myocardial oxygen demand via increased heart rate, coronary artery vasoconstriction, and cocaine-associated chest pain. A medical complication of the respiratory tract

includes a nasal septal perforation. The most acute systemic complication from cocaine abuse is muscle breakdown and renal failure (rhabdomyolysis).

Although abusers of "bath salts" experience many of the same symptoms as other stimulant abusers (e.g., headaches, heart palpitations, nausea, hallucinations, panic attacks, breakdown in skeletal muscle tissue, and paranoia), violent behaviors, heart attacks, kidney failure, liver failure, suicide, and an increased tolerance for pain have been reported (National Institute on Drug Abuse, 2012) (See Chap. 15).

12.7 Hallucinogen Use Disorder

Drugs considered hallucinogens are a diverse group of compounds that includes the phencyclidines (or phencyclidine-like substances) of PCP, ketamine, cyclohexamide, and dizocilpine, as well as the phenylalkylamines (e.g., mescaline): the indoleamines (e.g., psilocybin) and the ergolines, such as LSD (American Psychiatric Association, 2013). MDMA (also called "ecstasy") is technically neither a stimulant nor a hallucinogen. It is a member of the class of drugs called entactogens. However, it is listed under the category of "other hallucinogen intoxication" for diagnostic descriptive purpose in the DSM-5 (American Psychiatric Association, 2013). Recreational users of hallucinogens commonly refer to them as "club drugs." Club drugs are licit and illicit drugs from different classes used in bars, clubs, and concerts to enhance the sensory experience. Recently, the potential therapeutic effects of MDMA for PTSD have been studied in a RCT. Mithoefer and colleagues (Mithoefer et al., 2013; Mithoefer, Wagner, Mithoefer, Jerome, & Doblin, 2011) found that the majority of subjects with severe treatment-refractory PTSD had significant and long-lasting symptomatic relief provided by MDMA-assisted psychotherapy. No subjects reported harm from participation in the study.

12.7.1 Prevalence

Hallucinogen use disorder is the least prevalent of all SUD. In the USA, the 12-month prevalence is estimated to be 0.5 % among 12- to 17-year-olds and 0.1 % among adults. Native American and Alaska Native adolescents have the highest rates (1.2 %) of hallucinogen use disorder; among adults, the prevalence is the same for Native Americans, Alaska Natives, whites, and Hispanics (0.2 %) (American Psychiatric Association, 2013).

12.7.2 Clinical Characteristics

Hallucinogens comprise a group of substances having diverse chemical structures and involve different molecular mechanisms that produce an altered state of consciousness. The phencyclidines or phencyclidine-like substances (e.g., PCP,

ketamine) were first developed as dissociative anesthetics in the 1950s and became street drugs in the 1960s. PCP is known colloquially as angel dust, KJ (Kristal joint), illy, or wet. PCP comes in both powder and liquid form ("embalming fluid"), but typically, it is sprayed onto leafy material such as cannabis, mint, or oregano leaves and then smoked.

The term hallucinogen means "producer of hallucinations." A hallucination causes disturbances in judgment, orientation, intellect, memory, emotion, and level of consciousness (Pechnick & Cunningham, 2011). They produce feelings of separation from mind and body. Hallucinogens are most often taken orally, although some forms can also be smoked, snorted, or injected (e.g., ecstasy). Duration of effects varies across types of hallucinogen: LSD and ecstasy have a long duration such that users spend anywhere from hours to days using and recovering. The hallucinogenic effects in vulnerable individuals may last for weeks and precipitate a persistent psychotic episode resembling schizophrenia (McCann, 2011).

It was in the early 1960s when a psychology instructor from Harvard, Timothy Leary, began experimenting with hallucinogens, particularly LSD. He claimed LSD provided happiness, enhanced creativity, increased self-awareness, and might be useful as an adjunct to psychotherapy (Leary, 1997). By the mid-1960s, more than 1,000 articles on LSD appeared in the medical literature. Sandoz Laboratories stopped distributing the drug in 1966 because of the reported adverse reactions and resulting public fear. Today, LSD and the other hallucinogens (PCP, MDMA [ecstasy]) are classified as Schedule I drugs. Ketamine is used therapeutically as an anesthetic and is currently undergoing research for its antidepressant effects (Pechnick & Cunningham, 2011). A meta-analysis of randomized controlled trials (Krebs & Johansen, 2012) found evidence for the beneficial effects of a single dose of LSD, in decreasing alcohol misuse. There is also research supporting the medical use of psilocybin and LSD to terminate cluster headaches (Sewell, Halpern, & Pope, 2006) as well as psilocybin being safely used with acute reductions in core OCD symptoms (Moreno, Wiegand, Tatano, & Delgado, 2006). Clearly, further research examining accepted medical use of hallucinogens is warranted.

12.7.3 Medical Complications

Behavioral effects of PCP can vary by dosage. Low doses produce numbness in the extremities and intoxication, characterized by staggering and slurred speech. In moderate doses, analgesia and anesthesia are observed. Large doses may produce convulsions.

Hallucinogen toxicity from the so-called "bad trip" may include confusion, anxiety, depression, paranoia, drug-induced psychosis, and respiratory depression (especially with concurrent alcohol use). Acute anxiety or panic reactions usually wear off within 24 h. Depression with suicidal ideation can occur several days after LSD use. Psychosis can develop and persist after hallucinogen use, but it remains

unclear whether hallucinogen use can "cause" long-term psychosis or if it has a role in precipitating the onset of the illness (Pechnick & Cunningham, 2011).

12.8 Opioid Use Disorders

Opioid use disorder, as defined by the DSM-5 (American Psychiatric Association, 2013), includes signs and symptoms indicating a compulsive, prolonged self-administration of opioid substances that are used for no legitimate medical purpose or, if used to treat a medical condition, are used in doses in excess of the amount prescribed for that medical condition.

The term opiates refers to morphine and codeine that occur in opium along with many similarly psychoactive derivatives such as heroin, dilaudid, fentanyl, hydrocodone, and oxycodone (Jaffe & Martin, 1991). Heroin is only available illegally in the USA. Opiates such as codeine, hydrocodone, oxycodone, fentanyl, and morphine are commonly used for pain control.

The use of opium for intoxicant purposes and for pain relief appears to date back to at least ancient Greece where routes of administration were oral ingestion or inhalation of opium vapors. Use of opium via smoking appears to have first become popular in China in the 1600s after a ban on tobacco (Brownstein, 1993). In 1898, the Friedrich Bayer Company first synthesized aspirin and two weeks later, heroin. The pharmacologist at Bayer was most interested in finding a cough suppressant with a more benign side effect profile than morphine. Although this was later determined to be a false conclusion, it was believed that heroin stimulated and strengthened the lungs. Heroin and other opiates were sold as medicines for gynecological, respiratory, and nearly every other conceivable indication (Sneader, 1998). Gradually, reports that the drug was habit forming and used as a means of entertainment resulted in the 1914 passage of the Harrison Narcotic Act, and access to heroin was restricted. By 1924, heroin could no longer legally be prescribed.

12.8.1 Prevalence

Among adults age 18 and over, the 12-month prevalence rate of opioid use disorder is approximately 0.37 % (American Psychiatric Association, 2013). Rates are higher in males than in females (0.49 % vs. 0.26 %), with the male-to-female ratio of 1.5:1 for prescription opioids and 3:1 for heroin. Opioid use disorder is lowest among African Americans (0.18 %), average among whites (0.38 %), Asians or Pacific Islanders (0.35 %), and Hispanics (0.39 %) with the highest rates found in Native Americans (1.25 %) (American Psychiatric Association, 2013). In 2011, among persons 12–49 years, the average age at first use was 21.8 years for pain relievers and 22.1 years for heroin (SAMHSA, 2012). There were approximately 178,000 persons aged 12 or older who initiated heroin use and approximately 1.9 million new nonmedical users of pain relievers in 2011. It is important to note that

over five times as many individuals are dependent on prescription opioid pain relievers than on heroin (SAMHSA, 2012).

12.8.2 Clinical Characteristics

Most recreational users of opiates either snort or inject them intravenously, which produces flushing and an intensely pleasurable, diffuse bodily sensation that resembles orgasm. This initial "rush" is followed by a sense of well-being. With chronic use, these positive effects become unreliable (Epstein, Phillips, & Preston, 2011). Eventually, the effects of heroin and morphine become emotionally numbing and deadening (Marlowe, 1994). Signs of being "high" or intoxicated occur immediately and include psychomotor retardation, drowsiness, inactivity, impaired concentration, and constriction of the pupils, respiratory depression, and slurred speech. Nausea, vomiting, and constipation are common after repeated opiate use. Opiate abusers quickly build tolerance to their drug of choice, requiring increasing doses to achieve the desired effect, which can lead to unintentional overdosing. Withdrawal symptoms usually begin 8–10 h after the last dose and can be highly uncomfortable with a flu-like syndrome that is not life threatening. The most common symptoms are yawning, watery eyes, runny nose, chills, muscle aches, nausea, diarrhea, and cravings. Eating and sleeping are disrupted, and a fever may persist for as long as 2 weeks (Himmelsbach, 1942).

12.8.3 Medical Complications

Opioid use is associated with the slowing of gastrointestinal activity and a decrease in gut motility that produces severe constipation. Persons who sniff or "snort" heroin or other opioids may develop irritation of the nasal mucosa, sometimes resulting in perforation of the nasal septum. Difficulties in sexual functioning are commonly reported with males experiencing erectile dysfunction and females having irregular menses. In those who inject opioids, veins can become severely sclerosed resulting in peripheral edema. When veins become unusable, individuals often inject directly into their subcutaneous tissue with the result of an increased risk of infections such as cellulitis and tetanus. The use of contaminated needles by opioid injectors puts the user at risk for infections that include bacterial endocarditis, hepatitis, HIV infection, and tuberculosis. Infections are less common in opioid use disorder with prescription opioids. The variability in the development of opioid tolerance, as well as the fluctuating levels of purity of illicit opioids, may help explain why even opioid users with some degree of tolerance may experience severe opioid overdoses. The tendency to combine other drugs such as alcohol or sedatives with opioids may also contribute to overdosage (Jaffe, 1992). Opioid overdose is a medical emergency. Signs of acute opioid toxicity include up to complete and unresponsive coma, severe respiratory depression, and pinpoint pupils. There is pulmonary edema associated with the severe respiratory depression

(Jaffe, 1992). Opioid use disorder is associated with a mortality risk as high as 1.5–2 % per year. Death most often results from overdose, accidents, injuries, AIDs, or other general medical complications (American Psychiatric Association, 2013). Opiate overdose can be reversed by opiate antagonists (e.g., naltrexone, nalmefene, and naloxone) (See Chap. 15).

12.9 Substances Frequently Abused by Individuals with Eating Disorders

In addition to alcohol, cannabis, stimulants, sedative-hypnotics, opiates, hallucinogens, and club drugs, individuals with ED frequently abuse over-the-counter drugs, prescription medications, and performance enhancers.

12.9.1 Over-the-Counter Medications

A brief reminder to the reader is indicated in this discussion of over-the-counter medications. Below will be a brief overview of commonly used and abused substances (laxatives, diuretics, and diet pills) by individuals with ED. The term "over the counter" is used to describe substances and preparations that can be purchased in a pharmacy without a prescription, but also include products that can be bought in grocery stores, health food stores, or over the Internet. Unfortunately, these "dietary supplements," "herbal remedies," "energy boosters," "fat busters," and "colonics" are not under the jurisdiction or regulation of the US Food and Drug Administration (FDA) and therefore are not required to demonstrate safety or efficacy. There are serious and potentially life-threatening complications from the chronic use of some of these products (Consumer Reports, 2012; Mascolo, Chu, & Mehler, 2011; Steffen, Mitchell, & Roerig, 2007). Treatment providers should routinely inquire about the use of these medications as patients frequently do not spontaneously disclose the use of over-the-counter products or herbal supplements.

12.9.1.1 Laxatives

Laxatives are the most commonly abused over-the-counter medications used by individuals with ED (Mitchell, Specker, & Edmonson, 1997). In the last decade, prevalence rates for laxative use in ED outpatients have ranged from 26 % (Bryant-Waugh, Turner, East, Gamble, & Mehta, 2006) to 67 % (Steffen et al., 2007).

Despite their ineffectiveness as a weight reduction strategy, individuals with ED report using laxatives to manage anxiety, combat feelings of fullness or bloating, and promote weight loss. It is a common misperception among individuals with ED that laxative use will prevent caloric absorption; however, laxatives do not act on the small intestine where most of the absorption takes place. Any appreciable weight reduction is primarily due to significant fluid loss.

Laxatives can be divided into categories based on their mode of action—(1) stimulants, (2) osmotic agents, (3) lubricants, (4) bulk forming, and (5) softening

(Colton & Woodside, 1999)—and are sold over the counter and increasingly marketed on the Internet as "herbal remedies." Unfortunately, tolerance builds with long-term laxative use, requiring larger and more frequent dosages to obtain the desired results. Laxative abuse can lead to chronic constipation, severe dehydration, edema, bleeding, cathartic colon, electrolyte abnormalities, impaired bowel function, and rectal prolapse.

Most treatment protocols recommend immediate discontinuation of laxative use with no weaning or tapering. During the initial phase of withdrawal, patients often experience fluid retention and rebound edema. Bulk-forming agents such as Metamucil and Prodiem with at least 250 ml of water with each dose are recommended (Colton & Woodside, 1999). Additionally, patients may need increased contact with treatment providers and continued reassurance that a return to normal bowel functions may take up to two weeks.

Clinically speaking, ED patients that abuse laxatives may represent a more pathologically complex subgroup. Relative to non-abusers, ED patients that abuse laxatives are more likely to have symptoms of borderline personality disorder (Johnson, Tobin, & Enright, 1989; Tobin, Johnson, & Dennis, 1992), multi-impulsive behaviors including greater self-injury, and poly-substance abuse (Favaro & Santtonastaso, 1998; Wiederman & Pryor, 1996), a history of sexual abuse (Pryor, Wiederman, & McGilley, 1996), higher rates of depression requiring hospitalization, and past suicide attempts and are significantly more likely to also use diuretics, enemas, and diet pills (Mitchell, Boutacoff, Hatsukami, Pyle, & Eckert, 1986). Waller, Newton, Hardy, and Svetlik (1990) found that laxative abuse was correlated with higher body dissatisfaction and drive for thinness in patients with bulimia nervosa (BN). Finally, two studies have reported that laxative use is a predictor of poor treatment response in patients with BN (Blouin et al., 1994; Maddocks & Kaplan, 1991).

12.9.1.2 Diuretics

Individuals with ED also abuse diuretics. Approximately 31 % of BN patients report diuretic use for weight control purposes (Roerig, Mitchell, & de Zwaan, 2003). Diuretic use is often initiated to manage premenstrual fluid retention. Individuals with ED most commonly abuse over-the-counter diuretics; however, there is a subgroup of ED patients that abuse highly potent prescription diuretics (Pomeroy, Mitchell, Seim, & Seppala, 1998).

Similar to laxatives, diuretics are ineffective in preventing weight gain. Chronic diuretic use can cause nausea, abdominal pain and constipation, polyuria, heart palpitations, fluid and electrolyte imbalances, and kidney damage. Discontinuation of diuretics can cause rebound edema and weight gain. In a majority of cases, patients can be tapered off diuretics over the course of several days. Patients are encouraged to restrict their sodium intake and elevate their legs. However, if there is evidence of significant electrolyte abnormalities (e.g., hypokalemia), treatments need to be individualized and hospitalization may be required (Mitchell, Pomeroy, & Huber, 1988).

12.9.1.3 Appetite Suppressants

Other substances frequently used and abused by individuals with ED are various appetite suppressants including diet pills, caffeine, nicotine, and artificial sweeteners.

One study found that rates of diet pill use among ED patients rose 12 % between 1985 and 2003 (e.g., from 52 to 64 %, respectively) (Roerig et al., 2003). The most common ingredients found in over-the-counter diet aids include ephedrine (Ma Huang), caffeine, chromium, bulk forming, and stimulant laxatives among others. These substances can cause serious medical complications including elevated blood pressure, insomnia, tachycardia, depression, renal failure, neurological problems, stroke, seizures, and cerebrovascular hemorrhage.

In 1999, prescription strength orlistat (Xenical at 120 mg) was approved by the FDA as a weight loss medication to treat obesity. In 2007, the FDA approved the first nonprescription weight loss medication, orlistat (Alli at 60 mg) for over-the-counter use. A large multisite study found that 6 % of ED patients use Alli with approximately 23 % admitting that they had exceeded the maximum recommended dosage (Steffen et al., 2010). They also reported that a majority of Alli users were bulimic or binge eaters (as opposed to restricting anorexics) and were more likely to also use laxatives, diuretics, other diet pills, syrup of ipecac, and herbal fat burners as weight loss aids (Steffen et al., 2010).

Alli is an intestinal lipase inhibitor that works by disrupting the absorption of fat in the small intestines and reduces the absorption of fat-soluble vitamins. Potential side effects associated with the use of orlistat include abdominal pain, flatulence, soft stools, steatorrhea (excessive fat in the stool causing an oily appearance), and fecal urgency/incontinence.

In addition to the use of diet pills, individuals with eating disorders also use nicotine and caffeine to suppress appetite. In a large study of 1,206 monozygotic and 877 dizygotic adult female twins, researchers reported that regular smoking and caffeine disorder were the most prevalent SUD found in women with ED (Baker, Mitchell, Neale, & Kendler, 2010). Approximately 26 % of AN subjects and 23 % of BN subjects met the criteria for caffeine disorder, and 52 % of AN subjects and 45 % of BN subjects were regular smokers. These researchers found that rates of caffeine disorder and regular smoking were more prevalent in women with AN compared to BN (Baker et al., 2010).

Finally, individuals with ED frequently use large quantities of artificial sweeteners with the belief that these products will facilitate weight loss and/or prevent weight gain. In a study by Ohlrich, Aughey, and Dixon (1989), investigators found that 18 out of 21 consecutive ED patients reported the daily use of sorbitol (i.e., sugar-free gum). A more recent study by Klein, Boudreau, Devlin, and Walsh (2006) assessed weekly use of chewing gum; artificially sweetened, low calorie beverages; and packets of artificial sweetener in female patients with ANR, ANBP, and BN. On average, weekly pieces of gum chewed were ANR = 31, ANBP = 27, and BN-31; weekly consumption of 12-oz servings of diet beverages were ANR = 16, ANBP = 40, and BN = 25; and weekly number of packets of artificial sweetener used were ANR = 350, ANBP = 101, and BN = 39.

Taken together, it appears that women with ANBP and BN (the two groups that endorse purging) tend to report a higher use of gum and diet beverages than women with ANR. However, women with ANR reported a much higher use of artificial sweetener packets than either of the two purging groups (Klein et al., 2006).

12.9.2 Prescription Medications

12.9.2.1 Insulin

A meta-analysis of the existing studies on ED individuals with type 1 diabetes found that females with type I diabetes are three times more likely to have BN and two times more likely to have BED compared to their nondiabetic peers (Nielsen, 2002). Approximately 0–11 % of females with type 1 diabetes meet the criteria for a full syndrome ED, most commonly BN or BED, with another 7–35 % reporting symptoms of subthreshold ED (Colton, Rodin, Bergenstal, & Parkin, 2009). Adolescent girls and women with type 1 diabetes tend to have a higher BMI than nondiabetic peers, which may lead to body dissatisfaction, desire to lose weight, and an increased risk of ED (DCCT Research Group, 2001).

Intentional omission of insulin (also known as insulin purging) is a common phenomenon among individuals with ED and type 1 diabetes. Approximately 6 % of type 1 and 2.2 % of type 2 diabetics reported deliberate omission of insulin to control or lose weight (Herpertz, Albus, & Kielman, 2001). Complications of poor glycemic control can include ketoacidosis, vision problems, neuropathy, hearing loss, hypertension, kidney disease, and stroke.

It is important to note that several aspects of the standard protocol for the treatment of diabetes conflict with customary ED interventions. Diabetes treatment requires constant checking of glucose levels before each meal and snack to determine the amount of insulin that should be administered in order to prevent hypo- or hyperglycemia. Patients are educated to focus on food choices, reduce/avoid certain foods, and make lifestyle changes that often include reduced caloric intake and weight loss and encouraged to engage in regular physical activity (American Diabetes Association, 2008).

The co-occurrence of these disorders can negatively affect the course and treatment of an ED. If insulin abuse is suspected, clinicians are advised to work closely with the patient's endocrinologist, who should be considered for inclusion on the patient's treatment team.

12.9.2.2 ADHD Medications

Attention deficit hyperactivity disorder (ADHD) frequently co-occurs in individuals with ED (Ptacek, Kuzelova, & Stepankova, 2010) and in those with SUD (Brook, Brook, Zhang, & Koppel, 2010). Methylphenidate (i.e., Ritalin), amphetamine (i.e., Dexedrine, Adderall), and lisdexamfetamine dimesylate (i.e., Vyvanse) are frequently prescribed for the treatment of ADHD and used to improve concentration and reduce restlessness and impulsivity. However, one of the major side effects of these ADHD medications is reduced appetite and weight loss which

can lead to abuse by individuals with ED. Approximately 33 % of adults with ADHD have histories of AUD, and 20 % have histories of drug use disorders (Waid, LaRowe, Anton, & Johnson, 2004). Another study found that adolescent girls with ADHD were 5.6 times more likely to develop BN and 2.7 times more likely to develop AN than their non-ADHD peers (Biederman et al., 2007).

12.9.2.3 Levothyroxine

Levothyroxine (i.e., Synthroid, Levothroid, Levoxyl, Tirosint, Unithroid) is a medication prescribed to replace a hormone normally produced by the thyroid gland to regulate metabolism and the body's energy. Little is known about the rates of abuse of thyroid medication for weight loss in ED patients; however, there are several anecdotal reports in the literature (Crow, Mitchell, & Kendall, 1997; Fornari, Edleman, & Katz, 1990; Schmidt & O'Donoghue, 1992). An underactive thyroid often produces weight gain, and excessive self-administration of thyroid medication is a weight control strategy used by some patients with ED. Clinicians should routinely screen for levothyroxine use in their ED patients and be aware of the signs and symptoms of hyperthyroidism which include increased basal metabolic rate, polyphagia, weight loss, heat intolerance and sweating, tachycardia and arrhythmias, fatigue, muscle weakness, and simple tremors to severe myopathy (Crow et al., 1997).

12.9.3 Performance Enhancers

12.9.3.1 Steroids

ED or disordered eating is common in athletes and most prevalent in athletes that participate in sports where aesthetics are critical (i.e., dancing, figure skating, diving, gymnastics, body building), "making weight" is necessary to compete (i.e., wrestling, jockeys, rowing), and low body fat is perceived to improve performance (i.e., track and field, cross-country, swimming) (Baum, 2000). Performance-enhancing drugs (i.e., steroids, growth hormones, stimulants, and sports supplements) are often used by athletes to increase strength, decrease fatigue, and build muscle. Many of these substances have been banned by sports-regulating authorities (e.g., International Olympics Committee, Tour de France) and are illegal; however, some are available by prescription or can be purchased over the Internet.

Approximately 13.5 % of female athletes have a diagnosable ED (Sundgot-Borgen & Torstveit, 2004), and roughly 3 % use anabolic steroids to enhance their performance (Johnson, Powers, & Dick, 1999). Muscle dysmorphia is a subtype of body dysmorphic disorder which has been linked to both ED and steroid abuse in men (Strother, Lemberg, Stanford, & Turberville, 2012) (See Chap. 20).

The long-term effects of steroid abuse include adverse cardiovascular effects including hypertension, cardiomyopathy, and arrhythmias; neuroendocrine effects including suppression of the hypothalamic-pituitary-testicular axis, infertility, and prostatic hypertrophy; and neuropsychiatric effects including hypomanic or manic

symptoms, aggressive and/or violent behavior, and depression (Kanayama, Hudson, & Pope, 2008). Withdrawal from steroids should be medically monitored and can cause severe mood swings, depression, suicidal ideations, fatigue, restlessness, loss of appetite, insomnia, reduced libido, and "cravings" for the drug.

12.10 Definition of Recovery

Successful treatment and resultant recovery from a SUD can be defined in a multitude of ways. In the substance abuse field, historical definitions of recovery revolve around complete and sustained abstinence. A broader definition offered by O'Brien and McKay (2007) conceptualizes successful treatment as one that leads to significant reduction in substance use and ultimately to an improvement in the patient's ability to function in society. This definition takes into account the chronicity and remitting nature of SUD and builds on the view of addiction as a disease, much like diabetes. In this model, the aim of treatment is to manage as opposed to cure the disease. For example, individuals that *do not meet* the criteria for AUD but engage in episodes of problem drinking may not be willing to accept abstinence as the goal for treatment.

12.11 Levels of Care

SUD are complex psychiatric disorders that are heterogeneous in etiology and clinical presentation. As a result, this population varies widely, and no single treatment approach or level of care can effectively or efficiently accommodate the diverse clinical needs of these patients. Unfortunately, even today, the general public and many health and mental health-care professionals believe that there is a universally accepted, standardized approach to the treatment of alcohol and drug abuse problems (i.e., 28-day residential treatment). However, the "one size fits all" approach to treatment is neither cost-effective nor consistently successful in producing positive treatment outcome. As a result, the American Society of Addiction created patient placement criteria (ASAM PPC) for the treatment of substance-related disorders, which has undergone two revisions (ASAM PPC-2R) and currently includes criteria for people with co-occurring mental and SUD (Mee-Lee, Shulman, Fishman, Gastfriend, & Griffith, 2001). The purpose of the patient placement process is to match the patient to a specific setting and intensity of treatment by conducting a comprehensive assessment of the severity of the patient's illness and level of functioning. This assessment is conducted upon admission, during the continued stay review, and at discharge (Gastfriend & Mee-Lee, 2011). The current version of the PPC-2R lists five levels of care:

Level 0.5: Early Intervention. This is considered a pretreatment level of care for individuals that have risk factors or problems associated with substance use. Interventions are designed to help the individual recognize the negative

consequences of their substance use and gain skills and strategies to avoid future problems with drug and/or alcohol use.

Level I: Outpatient Treatment. This level of care usually consists of one or two weekly sessions of individual and/or group sessions for individuals with less severe symptoms of substance abuse or for individuals who need continued support for ongoing recovery. The focus of treatment is lifestyle, attitudinal, and behavioral changes that are necessary to reduce the negative consequences associated with substance abuse or issues that could promote relapse.

Level II: Intensive Outpatient/Partial Hospitalization. Intensive outpatient treatment (IOP) or partial hospitalization (PHP) programs provide extended mental health services (9–70 h per week) during the day, after work, in the evenings, and on weekends; however, patients reside at home. This level of care is designed to provide a higher intensity of programming, increased contact with clinical staff, and more support for individuals with SUD than traditional outpatient treatments. The basic services provided by this level of care include medical, psychiatric, and psychopharmacological consultation, medication management, and 24-h crisis services. Upon the completion of residential or inpatient treatment, individuals with SUD are often referred to this level of care to continue the recovery process. However, if medically stable, a patient may be able to start treatment in a "robust" (i.e., 70 h per week with supportive housing) PHP level of care.

Level III: Residential/Inpatient Services. This level of care is provided to individuals that require 24-h, supervised "live-in" care to prevent imminent danger or the negative consequences of continued substance use. Residential services include medication management, counseling and psychoeducation, and skills building to help individuals safely transition to less restrictive levels of care.

Level IV: Medically Managed Intensive Inpatient Services. This level of care is reserved for individuals that have severe mental health and substance-related problems that require 24-h medical management and/or require medically supervised detoxification services. Staffing consists of addiction medicine physicians, skilled nursing staff, and other mental health clinicians who provide specialized biomedical, psychiatric, and nursing services.

This continuum of care is fluid, and patients can move back and forth through the levels depending on their specific needs. Additionally, length of stay in each level of care is not static and depends on severity of illness and progress/response to treatment.

12.12 Assessment

A thorough assessment for SUD should include (1) a systematic and detailed evaluation of current and past alcohol and other substance use and abuse, (2) a comprehensive psychiatric evaluation to determine the presence and extent of other comorbid psychiatric conditions, (3) a physical examination with laboratory tests and drug screens, (4) a review of the individual's environment to determine the

strength of their social support network and determine barriers to treatment, (5) determination of stage of change for each problem, and (6) a discussion of the individual's response to previous psychological and/or pharmacological treatments. A systematic assessment should culminate in a comprehensive treatment plan and assignment to the appropriate level of care.

In addition to the diagnostic criteria set forth in the DSM-5 (American Psychiatric Association, 2013), there are several screening tools that are available to determine the frequency and intensity of alcohol/drug use, symptoms of dependence, and substance-related consequences (screening instruments are detailed elsewhere; See Chap. 14).

A comprehensive psychiatric evaluation should include attention to the most common comorbid conditions found in SUD including mood disorders, anxiety disorders, ED, trauma- and stress-related disorders (particularly posttraumatic stress disorders [PTSD]), impulse control disorders, and personality disorders (i.e., antisocial and borderline personality disorder [BPD]). The identification of all comorbid conditions will assist the clinician in the selection of both psychological and psychopharmacological interventions (Dennis & Sansone, in press). It is not uncommon for individuals (particularly women) with SUD to have multiple comorbid psychiatric conditions (SAMHSA, 2005). Approximately 30 % of alcoholic men (Helzer & Pryzbeck, 1988) and 60–70 % of alcoholic women (Swendsen et al., 2010) have preexisting psychiatric disorders; however, comorbid conditions can occur before, during, or after the onset of an SUD. Additionally, SUD can mask underlying mental illness, which may fully emerge with abstinence from drugs/alcohol.

A complete physical exam should include a review of each organ system to determine the presence and extent of medical complications due to substance abuse. Additionally, laboratory testing at the onset of treatment can provide valuable information to the patient that denies or minimizes their substance use. Periodic testing throughout treatment can also alert the clinician to signs of relapse (See Chap. 15).

Understanding the individual's current social support system and what they perceive as barriers to treatment can assist the clinician in treatment planning, selection of treatment modality, and relapse prevention (Longabaugh, Wirtz, Beattie, Noel, & Stout, 1995). For example, does the individual have emotional, financial, or physical support (e.g., childcare, transportation) from family, friends, partners, employers, the legal system, the medical community, religious, or other local organizations to promote and encourage the treatment and recovery process? Often, the barriers that exist before treatment are the same obstacles that interfere with successful recovery.

Upon intake, determining the "stage of change" can assist the treatment provider in developing a comprehensive treatment plan. A stage of change assessment should be conducted for *each* abused substance and *each* additional comorbid condition, as desire and commitment to modify attitudes and behaviors may vary depending on the issue. For example, the patient may be willing to take medication

to manage their bipolar disorder or stop using alcohol and cocaine, but unwilling to address weight gain which would be necessary for recovery from their ED.

Finally, understanding what previous treatments and/or pharmacological interventions worked or did not work in the past can inform and guide the current treatment team. It is important to create a treatment plan and utilize interventions based on extending or supporting what has worked well in previous interventions.

12.13 Psychological Treatments

In this section, we will briefly review the 12-step approach to the treatment of SUD and several evidence-based (EBT) psychological treatments including motivational interventions (MI), contingency management (CM), and cognitive behavioral therapy (CBT). For a more comprehensive understanding of these and other approaches, please refer to individual chapters in the treatment section of this volume, the American Psychiatric Association (2006), and Connery and Kleber (2007).

12.13.1 12-Step Approach to the Treatment

Historically, the foundation for SUD treatment has been a 12-step, psychoeducational, abstinence model approach, delivered in a group format in intensive outpatient and residential settings. This model views AUD/SUD as a physical, emotional, and spiritual disease that can be arrested, but not cured. Recovery is viewed as a lifelong process that involves working the 12-steps of Alcoholics Anonymous (AA), Narcotics Anonymous (NA), or Cocaine Anonymous (CA) and abstaining from the use of psychoactive substances. In the USA, this is the most common intervention for SUD/AUD and utilized by most all accredited residential and intensive treatment programs (Ries, Galanter, Tonigan, & Ziegler, 2011). Additionally, as of 2013, AA estimates there are over 115,000 self-help groups and 2 million members worldwide (http://www.aa.org, 2013).

Briefly, the 12-Step approach utilizes a set of guiding principles that outline a course of action for recovery from SUD/AUD and other behavioral addictions. According to this approach, the first step toward recovery is acceptance and surrender. The individual must accept that they suffer from a chronic, relapsing, progressive illness (alcoholism/drug addiction) that has made their life unmanageable. Since there is no effective cure for alcoholism or drug addiction, the only viable solution is complete abstinence. In other words, the first step requires the individual to move away from denial and recognize that their prior efforts to reduce or eliminate the negative consequences of their substance use have not been successful.

Steps 2, 3, 6, 7, and 11 focus on developing hope, faith, trust, and a relationship with a power greater than themselves. Although 12-Step programs are based on spiritual principles and utilize the concept of a "higher power," they distinguish

themselves from formal religious practices. Spirituality is defined as "that which gives people meaning and purpose in life" (Puchalski, Dorff, & Hendi, 2004, p. 689), and a "higher power" can be defined as anything that is "greater" than the individual (e.g., God, nature, science, consciousness, existential freedom, or even the *collective* wisdom and experience of an AA group).

Steps 4, 5, 8, 9 and 10 require the individual to engage in a fact-finding and fact-facing process known as a "moral inventory." This process requires the individual to take a fearless journey into their past and compile a list of traits, dysfunctional behaviors, and self-defeating patterns that have compromised personal integrity and important interpersonal relationships. The inventory should also be counterbalanced by the individual's strengths and competencies. Once the inventory is complete, the individual is asked to share their "personal story" with others which promotes humility, fearlessness, and honesty. In Steps 8 and 9, participants are asked to make a list of people they have harmed and make amends where possible. These steps provide the individual with an opportunity to apologize for past harm; remove guilt, shame, and remorse; and promote positive future relationships (Nelson, 1990).

Step 12 asks participants to incorporate these guiding principles into their daily lives and help others who are suffering from similar addictions. The rationale is that helping and supporting others deepens the individual's commitment to the program and lifelong recovery.

12.13.1.1 Twelve-Step Facilitation

Twelve-step facilitation (TSF) is a brief (12–15 sessions), structured, evidence-based (Moos & Timko, 2008), manual-driven approach to facilitating early recovery from AUD and SUD (Nowinski, Baker, & Carroll, 1995). This approach rests on the assumption that peer support is an essential component of the recovery process. The primary goals of this approach are (1) to promote abstinence from substance use and (2) facilitate active participation in a 12-step program (Ries et al., 2011).

TSF can be utilized in both inpatient and outpatient settings and has been successfully used with patients that have other comorbid psychiatric disorders (Ries et al., 2011). Although this approach was originally designed to be applied in individual treatment, several studies have explored its use in a group format. A recently completed large multisite study compared a TSF individual plus group model (i.e., Stimulant Abuser Groups to Engage in 12-Step [STAGE-12]) to treatment as usual (TAU) in intensive outpatient treatment for stimulant abusers. Compared to the TAU group, individuals in STAGE-12 did have higher rates of attendance at 12-step meetings during active TSF treatment and at 6-month follow-up (Donovan et al., 2013).

12.13.2 Motivational Interventions

Alcoholism and drug addiction are chronic, relapsing illnesses. The reinforcing properties of these substances and the psychological and physiological dependence they engender often prevent individuals from acknowledging the frequency and intensity of their substance use problems, which can result in avoidance of treatment. Unfortunately, even today, families as well as many health and mental health professionals view this avoidance as "denial" and believe that individuals must "hit bottom" or experience significant negative consequences from their substance abuse before they will self-refer for treatment. A widely supported belief in the mental health field is that unmotivated patients fair poorly in treatment. As a result, in the past, individuals with low motivation were often turned away from formal treatment programs and referred to self-help programs (e.g., AA) in hopes that peer support and testimony would increase their desire to change (DiClemente, Kofeldt, & Gemmell 2011). However, there have been substantial changes in the field of SUD treatment in the past few decades, and more frequently, treatment professionals are focusing on utilizing evidence-based strategies to *motivate* patients not just educate or medicate.

Motivational interviewing (MI) is an individual, client-centered, humanistic approach infused with change focused strategies borrowed from traditional behavior therapy. MI focuses on helping people explore and resolve their ambivalence about behavioral change. Clinicians are encouraged to avoid giving advice or guiding patients toward specific solutions. Rather, the therapist's role is to be nonjudgmental and empathetic, actively listen, clarify the patient's thoughts and experiences, roll with resistance, elicit change talk, and collaboratively help patients explore the implications of behavioral change (DiClemente, Van Orden, & Wright 2011). MI has demonstrated efficacy as brief intervention and an adjunct to other interventions in the treatment of SUD (Miller & Rose, 2009).

Originally developed by William Miller (1983) as an intervention for problem drinking, MI has evolved into a manual-guided, brief treatment known as motivational enhancement therapy (MET) and has been added to other active individual and group treatments for an array of target problems including cardiovascular rehabilitation, diabetes management, dietary change, hypertension, illicit drug use, infection risk reduction, management of chronic mental disorders, gambling, smoking, and ED (Miller & Rose, 2009) (See Chap. 22).

12.13.3 Contingency Management

Contingency management (CM) is an intervention that involves the systematic application of the operant conditioning principles of reinforcement and punishment. CM was initially implemented in the substance abuse field in the 1960s in methadone clinics to increase counseling attendance and abstinence from opiates. However, over the past two decades, there has been a significant increase in the number of published studies using CM for the treatment of AUD, cocaine, cannabis,

stimulants, polysubstance use disorders, and smoking cessation (Higgins, Sigmon, & Heil, 2011).

CM interventions are carefully designed and implemented to retain patients in treatment, increase therapy attendance, reduce/eliminate drug use, and promote adherence to medications and other therapeutic regimens. The most thoroughly researched form of CM for SUD is voucher-based CM (Higgins, Silverman, & Heil, 2008). In this approach, patients earn vouchers that can be exchanged for retail items, prizes, cash, or privileges. One fundamental principle underlying CM is that behavior that is followed by reinforcing consequences will increase the likelihood of that behavior in the future (i.e., operant conditioning). Providing substitute reinforcement for treatment compliance can help bridge the gap between the elimination of substance abuse and the delayed naturalistic rewards of abstinence which include improved health, interpersonal relationships, and increased productivity. A recent meta-analysis of voucher-based CM found that out of 72 controlled studies published between 2005 and 2009, 88 % reported statistically significant treatment effects (Higgins et al., 2011).

12.13.4 Cognitive Behavioral Therapy

Cognitive behavioral therapy (CBT) refers to a group of therapeutic strategies based on both classical and operant conditioning principles and the work of Beck (1963) and Ellis (1962). The basic model posits that mental disorders and psychological distress are maintained by maladaptive cognitions. The core premise of this approach is that an individual's conscious thoughts (cognitions) are based on beliefs and assumptions (schema) derived from previous life experiences and that these thoughts determine how the person structures and reacts to their environment. The therapist and patient work collaboratively to identify the faulty or maladaptive cognitions that maintain their symptoms and self-defeating behaviors. Through Socratic questioning, hypothesis testing, problem solving, and homework, the patient challenges the validity of their faulty assumptions (i.e., cognitive restructuring) and modifies maladaptive behavioral patterns.

CBT is an active, structured, symptom focused, goal-directed approach that has been found effective in the treatment of mood disorders, anxiety disorders, PTSD, eating disorders, sleep disorders, personality disorders, psychotic disorders, and SUD (Hofmann et al., 2012). Because a significant majority of SUD patients have co-occurring psychiatric disorders, CBT can address multiple disorders in a comprehensive and integrated manner. The skills learned in CBT are designed to be generalizable and will benefit the individual long after they leave treatment (Carroll, 2011).

Over the past three decades, there have been numerous RCT of CBT for a wide range of SUD. CBT has been found effective in the treatment of AUD (Miller & Wilbourne, 2002; Morgenstern & Longabaugh, 2000), cocaine use disorders (Carroll, Rounsaville, Nich, & Gordon, 1994; Monti, Rohsenow, Michalec, Martin, & Abrams, 1997), cannabis use disorder (Copeland, Swift, Roffman, & Stephans, 2001;

MTP Research Group, 2004), opiate use disorder (Linehan et al., 2002), and polysubstance abuse (Linehan et al., 1999; Pollack et al., 2002; Schmitz, Averill, Sayre, McCleary, & Swann, 2002). CBT has also been found effective when combined with CM in the treatment of cocaine use disorders (Higgins et al., 1992) and opiate use disorders (Bickel, Amass, Higgins, Badger, & Esch, 1997) (See Chap. 25).

12.14 Pharmacological Treatments

Pharmacological interventions are commonly used in the withdrawal phase of treatment for AUD and opioid use disorders and post-withdrawal to manage craving and to prevent relapse. A comprehensive discussion of medications used for substance withdrawal is beyond the scope of this chapter; however, it can be found in Chap. 15. In this section, we will briefly review the pharmacological treatments that have an FDA approval for the treatment of SUD.

12.14.1 Alcohol Use Disorders

There are currently four FDA-approved, clinically useful medications for the treatment of AUD. Disulfiram (Antabuse) is an oral medication designed to reduce the likelihood of alcohol consumption due to its adverse effects. This medication produces highly unpleasant symptoms when combined with alcohol, including nausea, vomiting, headache, flushing, hypotension, vertigo, tachycardia, and dysphoria. In several RCT, disulfiram was found to reduce drinking days; however, there is no evidence that it improved relapse rates compared with placebo (Garbutt, West, Carey, Lohr, & Crews, 1999). Naltrexone (Revia [orally administered] and Vivitrol [injectable]) is an opioid receptor antagonist that decreases the reinforcing effects of alcohol ingestion, including reduced feelings of intoxication and fewer cravings. Naltrexone has been found to have short-term benefits in reducing relapse to heavy drinking; however, the evidence for longer-term use is less compelling (Williams, 2005). Acamprosate (Campral) is the most recently FDA-approved medication for AUD. This medication has been found effective in reducing relapse rates in alcohol-dependent individuals when combined with psychosocial treatments (Rosenthal, 2011).

Several other psychopharmacological agents have been studied (off-label investigations) in the treatment of AUD including anticonvulsants (topiramate [Topamax]), antispasmodics (baclofen [Lioresal and Kemstro]), serotonin reuptake inhibitors (fluoxetine [Prozac] and sertraline [Zoloft and Lustral]), and antiemetics (ondansetron [Zofran]). Each of these medications has demonstrated some efficacy for particular AUD subgroups (see Rosenthal, 2011 for a complete review of these agents).

12.14.2 Opioid Use Disorders

The FDA has approved five medications for the treatment of opioid use disorders. Methadone was first introduced in the early 1970s as a treatment for opiate dependence. It is a synthetic opioid and agonist (mimics the action of an opiate) that mitigates withdrawal symptoms, blocks the euphoric and sedating effects of opiates, and relieves craving. Methadone can only be dispensed at outpatient opioid treatment programs (OTP) that are certified by the Substance Abuse and Mental Health Services Administration (SAMHSA) and registered with the Drug Enforcement Administration (DEA). Daily oral administration of methadone, psychosocial interventions, and HIV education are all important components of the comprehensive treatment provided in these programs. Studies suggest that there is strong evidence that methadone maintenance keeps opiate abusers in treatment and reduces opioid use better than treatments without medication (Saxon & Miotto, 2011).

Buprenorphine (Subutex) and buprenorphine/naloxone (Suboxone) were approved by the FDA in 2002, and a new combination of buprenorphine and naloxone (Zubsolv) was approved in 2013. Buprenorphine is an opioid partial agonist, and naloxone is an opioid antagonist used to counter the effects of opiate overdose. Taken sublingually (placed under the tongue and allowed to dissolve), at low doses, buprenorphine produces sufficient agonist effects to prevent withdrawal symptoms and at moderate doses, has a "ceiling" for euphoric effects. Buprenorphine can only be prescribed and dispensed by certified OTP or through an approved program for physicians and pharmacists. Studies have shown that buprenorphine is more effective than placebo and equally as effective as moderate doses of methadone in opioid maintenance therapy. Also, it is known to cause a milder withdrawal syndrome compared to methadone (Stotts, Dodrill, & Kosten, 2009).

Naltrexone (Vivitrol) is a nonaddictive, nonnarcotic opioid antagonist. This medication does not mimic the effects of opioids (like methadone and buprenorphine), rather it blocks the receptor cites in the brain, thus preventing the euphoric effects of the drug. Naltrexone is administered in an injectable, long-acting form once per month to prevent relapse. Although some reviews suggest that retention and relapse rates for opioid use disorders are low, other studies suggest that naltrexone may be the treatment of choice for highly motivated patients (e.g., health-care professionals) that need to remain opiate-free (Veilleux, Colvin, Anderson, York, & Heinz, 2010).

A new form of buprenorphine (Probuphine) for the treatment of opiate addiction is currently under review by the FDA. This long-acting version is implanted under the skin in the upper arm and remains in place for 6 months. Approval was denied in April 2013 pending further information on the effects of higher doses and the development of a comprehensive plan for training physicians that would be inserting and removing the implant (Titan, 2013).

12.15 Risk Factors and Comorbidity in Substance Use Disorders

SUD are the result of the interplay between genes, environment, and developmental risk factors that influence one's susceptibility to psychoactive substance abuse. Although there is robust evidence indicating that genes play a significant role in the development of SUD, important psychological and social factors need to be considered in both the development and maintenance of these disorders.

Results from the 2011 national survey on drug usage found that 20.6 million individuals (8 % of the general population) are abusing or dependent on drugs and/or alcohol, with 14 million alcohol dependent, 4 million abusing or dependent on illicit drugs, and almost 3 million abusing or dependent on both alcohol and other drugs (SAMHSA, 2012) (see Table 12.1).

Gender, age, marital status, ethnicity, and employment status all appear to be risk factors associated with the development of SUD. Sociodemographic data suggest that males initiate substance use earlier than females, and males are more likely than females to abuse psychoactive drugs (approximately 10 vs. 6 %) or be heavy alcohol drinkers (6 vs. 3 %) (SAMHSA, 2012). Among individuals aged 18 or older, age of first use of cannabis (14 or younger) was associated with higher rates of SUD than adults who first used cannabis after age 18. Similarly, individuals that used alcohol at age 14 or younger were 7 times more likely to develop AUD than adults who consumed their first drink at age 21 or older (SAMHSA, 2012). Unmarried individuals have higher rates of substance *use* and approximately 11 % of divorced or separated women, and 16 % of women who have never married have a diagnosable SUD, as compared to only 4 % of married women (SAMHSA, 2004). In 2011, rates of SUD were lowest among Asians (3 %) and blacks (7 %). The highest rates of SUD were found in American Indians and Alaska Natives (17 %), followed by Native Hawaiians or other Pacific Islanders (11 %), Hispanics (9 %), and whites (8 %). Unemployed adults are more likely than employed adults to have SUD (15 vs. 8 %) (SAMHSA, 2012).

There have been several compelling family, twin, and adoption studies that suggest SUD is genetically influenced and that heritability factors place certain individuals at risk for the development of these disorders. Alcohol use disorders (AUD) run in families. McGue (1999) reported that 50–60 % of the phenotypic variance found in alcohol dependence in both men and women is due to genetic factors. In other words, children of alcoholics are five to six times more likely than the general population to develop AUD. Similarly, researchers have found that adolescents of parents that abuse illicit drugs are 45–79 % more likely than the general population to abuse drugs (Agrawal & Lynskey, 2006; Tsuang, Bar, Harley, & Lyon, 2001). In a large twin study done by Kendler, Jacobson, Prescott, and Neale (2003), genetic factors accounted for 73 % of the variance in cannabis abuse, 63 % in cocaine abuse, 63 % in hallucinogens, 51 % in sedative abuse, and 57 % in stimulant abuse. The only psychoactive substance in this study that showed a different phenotypic pattern was opiate abuse, where only 23 % of the variance could be attributed to genetic factors and 77 % to environmental factors. These researchers also found that an increased risk for the development of SUD was

nonspecific (i.e., genetic liability put one at risk to abuse *any* psychoactive drug) (Kendler et al., 2003). Additionally, genes that influence the development of SUD are also relevant to the development of other externalizing psychopathologies including conduct disorders, antisocial personality disorder, and ADHD (Kendler, Prescott, Myers, & Neale, 2003).

One of the strongest predictors of current substance abuse is past substance abuse. Previous epidemiological research on the "gateway pattern" found a predictable sequence of drug use initiation that begins with alcohol and nicotine use followed by cannabis then other illicit drugs (Kandel, Yamaguchi, & Chen, 1992). However, a recent epidemiological survey was conducted in 17 countries, and the results suggest that the pattern found in the USA is not consistently found in other countries. Other factors influence the ordering and progression of drug use around the world including availability of particular substances and cultural attitudes (Degenhardt et al., 2010).

Several environmental factors have been suggested as important in the initiation and maintenance of SUD. First, SUD tend to aggregate in families. In part, this is due to genetic influences, but studies have suggested that parental substance abuse can influence the development of SUD in children (Jennison & Johnson, 2001). In a large national study, looking at the relationship between childhood adversities and adult psychiatric disorders, researchers found that the maladaptive family functioning cluster (i.e., parental mental illness, parental SUD, criminality, family violence, physical abuse, sexual abuse, and neglect) predicted the development of and persistence of SUD (Green et al., 2010; McLaughlin et al., 2010).

There are also strong associations between mental disorders and SUD. Several longitudinal and cross-sectional surveys have confirmed that mental disorders can be conceptualized as risk factors for SUD (Compton, Conway, Stinson, Colliver, & Grant, 2005) because they precede the onset of SUD and can divide the population into high- and low-risk groups (Kraemer et al., 1997). The presence of premorbid mood disorders (particularly bipolar disorder), anxiety disorders (including PTSD), and externalizing disorders (i.e., ADHD, oppositional defiant disorder, conduct disorder, intermittent explosive disorder) predicted the likelihood of future SUD (Glantz et al., 2009). Similarly, Compton, Thomas, Stinson, and Grant (2007) found that individuals who met the criteria for *any* personality disorder were 1.8 times more likely to be substance abusers and 3.3 times more likely to be drug dependent than those who did not have a personality disorder. Finally, in a large national study of DSM-IV comorbidity in borderline personality disorder (BPD), researchers found that half of the respondents (51 %) had a SUD, 51 % had a mood disorder, and 60 % had a comorbid anxiety disorder (Grant et al., 2008).

Conclusions

This chapter was compiled for the ED treatment provider that is not well acquainted with the diagnosis, assessment, and treatment of patients with SUD. In the companion chapter, entitled *Introduction to Eating Disorders for the Substance Abuse Specialist* (See Chap. 11), we provided a basic overview of

diagnosis, clinical characteristics, medical complications, assessment, and evidence-based approaches for individuals with anorexia nervosa, bulimia nervosa, and binge eating disorder. Together, these chapters are designed to begin the cross-training process and prepare the reader to more fully assimilate and apply the content of the remaining chapters in this textbook. See Chap. 21 for a discussion of integrated treatment for patients with both ED and SUD.

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Are Eating Disorders Addictions?

13

Timothy D. Brewerton

Abstract

Eating disorders overlap with substance use disorders and addictions in many important ways, including clinical phenomenology, comorbidity, pathophysiology, neurobiology, and treatment approaches. Evidence is reviewed for and against the contention that the eating disordered behaviors of dieting, binge eating, purging, and exercising are potentially addictive behaviors. In addition, abuse of and dependence upon substances meant to inhibit appetite (stimulants), reduce caloric absorption (laxatives, lipase inhibitors), decrease water weight (diuretics), or induce vomiting (ipecac) are characteristic features of eating disorders with bulimic features. The phenomenology of the eating disorders is viewed in light of the DSM-5 criteria for an addictive disorder and the new description of an addiction recently published by the American Society of Addiction Medicine. Both sets of criteria support the conclusion that eating disordered behaviors can be phenotypically conceptualized as addictive. On the other hand, eating disorders exhibit clinical characteristics not seen in classical substance use disorders or addictions, including distortions in body size and shape as well as intense fear of gaining weight. The therapeutic implications of treating eating disorders as addictive disorders are discussed. Taken together, eating disorders may be conceptualized as addictive disorders in at least a subset of individuals, but further research is required to determine if tolerance and withdrawal occur in humans.

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13.1 Introduction

Research evidence supports the contention that eating disorders (ED), substance use disorders (SUD), and addictions are complex disorders that are caused by multiple factors. There is simply no one cause; rather, ED and SUD result from an interaction of an array of genetic and environmental factors. These factors can be understood in light of a biopsychosocial, spiritual, and developmental continuum in which both nature and nurture interact over time. In the last century, most research studies focused on either some aspect of nurture or some aspect of nature and rarely were they truly integrated. It has not been until the dawn of this millennium that investigators have been studying them together and showing quite readily how much they interact and influence each other. A good way of thinking about it is that "genetics loads the gun, and environment pulls the trigger."

Latent vulnerability theory helps to explain why only four or five out of 100 girls who go on a serious diet will develop anorexia nervosa (AN), bulimia nervosa (BN), binge eating disorder (BED), or an other specified ED. An important point to emphasize for the purposes of this discussion is that all of the problematic behaviors associated with ED—dieting, binge eating, purging, and exercising—are known to alter brain neurochemistry, sometimes in very profound ways. This is particularly true for the prolonged starvation that results from chronic dieting and weight loss. Over time, these four basic behaviors proceed to expose the genetically mediated latent or hidden vulnerability in any given individual that in turn leads to the overt manifestations of an ED. Importantly, all of these four ED behaviors have been found to have addictive features, which are the focus of this chapter. Does the science support this perspective?

This chapter will first outline the new criteria for substance-related disorders and addictions set forth by the publication of the DSM-5 (American Psychiatric Association, 2013) and the Public Policy Statement on addictions published by the American Society of Addiction Medicine (ASAM, 2011) and discuss their relationship to ED (particularly bulimic-spectrum disorders). This will be followed by a summary of the similarities and differences between addictions and ED. Both the pros and cons of conceptualizing binge eating as a "food addiction" will be explored. Finally, the chapter will conclude with a discussion of treatment implications and directions for further research.

Table 13.1 DSM-5 criteria for unspecified substance use disorder as applied to food. There is a problematic pattern of use leading to clinically significant impairment or distress that is manifested by two of the following (American Psychiatric Association, 2013)

YES	1. The substance is often taken in larger amounts or over a longer period than was intended
YES	2. There is a persistent desire or unsuccessful efforts to cut down or control use of the substance
YES	3. A great deal of time is spent in activities necessary to obtain the substance, use the substance, or recover from its effects
YES	4. Craving, or a strong desire or urge to use the substance
YES	5. Recurrent use of the substance despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of its use
YES	6. Continued use of the substance despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of its use
YES	7. Important social, occupational, or recreational activities are given up or reduced because of use of the substance
YES	8. Recurrent use of the substance in situations in which it is physically hazardous
PROBABLY	9. Tolerance, as defined by either of the following: (a) A need for markedly increased amounts of the substance to achieve intoxication or desired effect (b) A markedly diminished effect with continued use of the same amount of the substance
NO[a]	10. Withdrawal, as manifested by either of the following: (a) The characteristic withdrawal syndrome for other substance (b) The substance (or a closely related substance) is taken to relieve or avoid withdrawal symptoms

[a]YES in animal studies for sugar

13.2 DSM-5 Criteria for Substance-Related Disorders and Addictions

When the DSM-5 (American Psychiatric Associations, 2013) criteria for substance dependence are examined in light of ED symptoms, such as binge eating, many behavioral similarities are apparent (see Table 13.1). For many years, substance dependence was defined by the presence of tolerance and withdrawal, but now they are not necessary for a diagnosis of SUD to be made. When it comes to the behaviors of dieting, binge eating, purging, and exercising, it is not yet clear whether there are true tolerance and withdrawal to these behaviors in humans.

The DSM-5 criteria for an "other (or unknown) substance-related disorder" will be reviewed here as it applies to food, which is the primary substance abused by patients with bulimic-spectrum disorders (see Table 13.1). The "A" criteria indicate "*a problematic pattern of use of an intoxicating substance not able to be classified with the alcohol*; *caffeine*; *cannabis*; *hallucinogen* (*phencyclidine and others*); *inhalant*; *opioid*; *sedative*, *hypnotic*, *or anxiolytic*; *stimulant*; *or tobacco categories*. . .)." The online Merriam-Webster dictionary definition of the word

"intoxicating" is as follows: "a) producing in a person a state ranging from euphoria to stupor, usually accompanied by loss of inhibitions and control; inebriating; b) stimulating, exciting, or producing great elation." It can be argued that certain foods are indeed "intoxicating" to some people, particularly those with bulimic-spectrum disorders. It has been shown that such individuals experience food in a different way than individuals without bulimic tendencies. In particular, highly palatable foods induce a high degree of "hedonic reward," and the saliency for food is enhanced. In addition, recent ecological momentary assessment (EMA) findings indicate that binge eating and purging induce positive affect and are therefore experienced as particularly pleasurable (see Chap. 16).

The DSM-5 criteria continue: "*and leading to clinically significant impairment or distress, as manifested by at least two of the following, occurring within a 12-month period.*"

1. "*The substance is often taken in larger amounts or over a longer period than was intended.*" This clearly occurs with binge eating and subsequent purging, which tends to be chronic once commenced. Fichter, Quadflieg, and Hedlund (2008) reported that 36 % of BED and 28 % of BN patients still received an ED diagnosis at 12-year follow-up.
2. "*There is a persistent desire or unsuccessful efforts to cut down or control use of the substance.*" By definition, a binge entails not only eating a large amount of food but also the subjective sense of loss of control over eating. Furthermore, binge eating is typically experienced as ego-dystonic, a behavior that, although pleasurable, is undesired and often shame based. Even with the best treatments for BN or BED, many patients do not obtain nor do they maintain full abstinence.
3. "*A great deal of time is spent in activities necessary to obtain the substance, use the substance, or recover from its effects.*" This is also present in bulimic-spectrum disorders, particularly when there are both binge eating and purging. Binge and/or purge episodes take up inordinate amounts of time and also take a toll mentally and physically, often leading to marked fatigue as well as other related medical effects (see Chap. 15). Individuals who binge and purge devote a great deal of time and money to acquiring food and getting rid of it, often in secret and with great distress, guilt, and shame.
4. "*Craving, or a strong desire or urge to use the substance.*" Craving has been described as a feature in bulimic ED (Van den Eynde et al., 2012), which is only aggravated by dietary restriction (Moreno-Dominguez, Rodriguez-Ruiz, Fernandez-Santaella, Ortega-Rolden, & Cepeda-Benito, 2012).
5. "*Recurrent use of the substance despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of its use.*" By definition, bulimic disorders are recurrent and persistent, and as a result, a host of adverse consequences may ensue, including social, interpersonal, academic, economic, and/or medical. Negative reactions of family and friends to bingeing and other ED behaviors are common. Those who are most chronic are also the ones who are the most self-destructive (Fichter et al., 2008).

6. "*Continued use of the substance despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of its use*." Impairment in the social life of individuals with BN, BED, subthreshold BED, or any recurrent binge eating was commonly reported in the National Comorbidity Survey Replication (Hudson, Hiripi, Pope, & Kessler, 2007).
7. "*Important social, occupational, or recreational activities are given up or reduced because of use of the substance*." In addition to impairment in social life, there is also impairment in home, work, and the personal life of individuals with bulimic-spectrum disorders (Hudson et al., 2007). Withdrawal from others and overt social phobia is commonly associated with bulimic disorders and can be exacerbated by the shame and secretiveness associated with this behavior (Brewerton, Lydiard, Ballenger, & Herzog, 1993; Hudson et al., 2007).
8. "*Recurrent use of the substance in situations in which it is physically hazardous*." Bulimic disorders and binge eating occur while driving a motor vehicle, and this is a recognized cause of accidents (Petridou & Moustaki, 2000). The author has personally known of many patients who engage in bulimic behaviors in their automobiles while driving, including one patient who was killed in a head-on collision while actively bingeing and purging.
9. "*Use of the substance is continued despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by the substance*." Such persistence in the face of negative outcomes, both medical and psychiatric, is also a common characteristic of ED (see Chap. 11). Examples include fatigue, fluid and electrolyte abnormalities leading to muscle weakness and spasms, cardiac arrhythmias, pharyngeal/esophageal irritation and bleeding, gastroesophageal reflux disease (GERD), anemia, dental erosion, as well as the effects of weight gain and resultant obesity, e.g., hypertension, diabetes, sleep apnea, etc. (see Chap. 15).
10. "*Tolerance, as defined by either of the following*:"
    (a) "*A need for markedly increased amounts of the substance to achieve intoxication or desired effect*."
    (b) "*A markedly diminished effect with continued use of the same amount of the substance*." Food tolerance has been demonstrated in animal studies (see Chap. 1). Over time rats with intermittent sugar and chow access have decreases in dopamine type 2 (D2) receptor messenger ribonucleic acid (RNA) levels in the nucleus accumbens compared with control rats on chow alone (Avena, Rada, & Hoebel, 2008). Evidence for tolerance in humans is indirect and implied and comes largely from clinical reports of bulimic patients eating larger and larger binge meals, eating more frequent binge meals, and/or gaining weight as the disorder progresses. The impaired satiety characteristic of full-blown bulimic-spectrum disorders can be seen as an indirect indicator of tolerance, as is the fact that individuals with BN and BED are typically overweight or obese (Brewerton, 1995; Dansky et al., 1998; Hudson, Hiripi, Pope, & Kessler, 2007). MRI studies indicate hypofunctioning of brain reward circuitry in response to a chocolate milkshake in patients with BN compared to

controls, which the authors speculated may be due to repeated bingeing on hyperpalatable foods (Bohon & Stice, 2011). In addition, the positive relationship between binge size, frequency, and BMI in individuals with BED also supports the notion of tolerance to food (Guss, Kissileff, Devlin, Zimmerli, & Walsh, 2002; Picot & Lilenfeld, 2003). As BMI increases, insulin resistance also increases, which can increase hunger and reduce satiety (Madden, Leong, Gray, & Horwath, 2012). In a prospective study over 8 years, Thomas, Butryn, Stice, and Lowe (2011) reported that substantial weight gain (or weight loss) resulted in a sevenfold increase in risk for subsequent onset of BN or subthreshold BN and that these individuals who developed a bulimic ED experienced greater increases in weight during the 2 years prior to ED onset when compared to healthy controls. In a prospective study of children over the course of 5 years, Tanofsky-Kraff and colleagues (2006, 2012) reported that binge eating predicted subsequent weight gain, increase in adipose tissue, and the development of signs of metabolic syndrome. In a prospective study of adolescents, Stice, Marti, Shaw, and Jaconis (2009) noted that subthreshold cases of BN and BED often progressed to threshold cases, thereby documenting a progressive increase in meal size and binge frequency.

11. "*Withdrawal*, *as manifested by either of the following*:"
    (a) "*The characteristic withdrawal syndrome for other* (*or unknown*) *substance* . . ."
    (b) "*The substance* (*or a closely related substance*) *is taken to relieve or avoid withdrawal symptoms*." A withdrawal syndrome from sugar has been described in animals (Avena, Bocarsly, Rada, Kim, & Hoebel, 2008; Avena, Rada, & Hoebel, 2008, 2009; Colantuoni et al., 2002; Ifland et al., 2009) (see Chap. 1) but remains to be clearly demonstrated in humans (Benton, 2010). However, there are clinical, anecdotal reports of "sugar withdrawal" in humans, which is characterized by irritability, headaches, and flu-like symptoms (Davis & Carter, 2009).

Taken together, the DSM-5 criteria for a substance-related disorder or addiction can be applied to ED behaviors, particularly binge eating, which commonly involves highly palatable, highly rewarding food. In a similar exercise, Cassin and von Ranson (2007) applied DSM-IV criteria for substance dependence to a group of women with BED and found that 92.4 % of them met criteria for food dependence. Likewise, Goodman (1990) applied DSM-III-R criteria for substance dependence to individuals with compulsive overeating and found significant phenotypic overlap. However, it is important to note that this phenotypic overlap between SUD and ED does not necessarily imply the same illness or mechanism.

13.3 The American Society of Addiction Medicine Definition of Addiction

Psychiatry and addiction medicine have had an interesting relationship over the years and have not always been in agreement (O'Brien, Volkow, & Li, 2006). The word "addiction" or "addictive" was thought to have a negative, stigmatizing connotation and therefore did not appear in any version of the Diagnostic and Statistical Manual of Mental Disorders until DSM-5 (American Psychiatric Association, 2013), where the classification of "substance-related disorders and addictive disorders" was listed for the first time.

The American Society of Addiction Medicine (ASAM, 2011) recently published an expanded definition of addiction. In summary, it reads as follows: "*Addiction is a primary, chronic disease of brain reward, motivation, memory and related circuitry. Dysfunction in these circuits leads to characteristic biological, psychological, social and spiritual manifestations. This is reflected in an individual pathologically pursuing reward and/or relief by substance use and other behaviors. Addiction is characterized by inability to consistently abstain, impairment in behavioral control, craving, diminished recognition of significant problems with one's behaviors and interpersonal relationships, and a dysfunctional emotional response. Like other chronic diseases, addiction often involves cycles of relapse and remission. Without treatment or engagement in recovery activities, addiction is progressive and can result in disability or premature death.*"

This definition is reminiscent of the DSM-5 definition as reviewed above. All of the clinical features and aspects described in the ASAM definition are seen in ED, although some of these may also be seen in other psychiatric disorders as well, such as impulse control disorders (see Chaps. 6 and 18). A common feature in both SUD and bulimic ED is the "inability to abstain" from harmful substances and/or behaviors. Like other chronic diseases, the ASAM definition notes that addiction often involves cycles of relapse and remission, which is true for many individuals with ED, as well as mood and anxiety disorders, which are highly comorbid to both SUD and ED. Without treatment or engagement in recovery activities, both ED and addictions can result in disability or premature death.

The ASAM expanded definition of addiction states that it is "*a primary chronic disease of brain reward, motivation, memory, and related circuitry, addiction substantially affects neurotransmission and interactions within reward structures of the brain, particularly the nucleus accumbens, anterior cingulate cortex, basal forebrain and amygdala, such that motivational hierarchies are altered and addictive behaviors, which may or may not include alcohol and other drug use, supplant healthy, self-care related behaviors.*" This has clearly been shown for ED, at least bulimic ED (see Chaps. 3 and 4). Addictions, SUD, and ED affect neurotransmissions and interactions between cortical and hippocampal circuits and brain reward structures, such that the memory of previous exposures to rewards (such as food) leads to a biological and behavioral response to external cues, thereby triggering craving and/or engagement in addictive and ED behaviors.

Problems in frontal cortex function in both SUD and ED contribute to altered judgment and the dysfunctional pursuit of rewards. Importantly, the frontal lobe morphology, connectivity, and functioning are still in the process of maturation during adolescence and young adulthood and do not fully mature until the mid- to late 20s (Lenroot & Giedd, 2008). Certainly, when children and adolescents are exposed to early substances or potentially addictive behaviors, there is a heightened risk in terms of developing an addiction and/or an ED, as well as their negative consequences. Most mental disorders, including SUD and ED, begin in childhood and adolescence (Kessler, Amminger, & Ustun, 2007).

The ASAM definition of addiction encompasses both the pursuit of pleasure and relief from negative affect. Typically, an addiction is a behavior that is performed to induce pleasure, at least initially, whereas a compulsion is something that is not, for the most part, pleasurable but instead results in relief from anxiety. Often these phenomena overlap, particularly once the behavior has become chronic. Frequently, individuals experience pleasure in getting relief from negative affect. Typically, after an addiction has progressed, the rewarding aspect or pleasure is diminished, and it becomes more of a sense of relief to mitigate the withdrawal symptoms. Similarly, binge eating may be initiated to induce pleasure but often serves as a method to relieve anxiety.

As noted in the expanded ASAM definition, both genetics and environment play a significant role in whether and how addictions develop. Genetic liability accounts for approximately 50 % of the likelihood that an individual will develop an addiction (ASAM, 2011) and 50–80 % of probability that someone will develop an ED (Bulik, Sullivan, Tozzi, Furberg, Lichtenstein, & Pedersen, 2006; Kendler, MacLean, Neale, Kessler, Heath, & Eaves, 1991; Klump, Miller, Keel, McGue, & Iacono, 2001). In other words, there is substantial evidence that SUD (like ED) aggregate in and are transmitted within families. Emerging research on genetic cross-transmission between disorders has been documented and is discussed in Chap. 5. We live in a toxic, fast-paced, highly stressful culture that is materialistic, image driven, food and pleasure oriented, and drug seeking. Exposure to trauma and other significant life stressors plays an important role in the development of both addictions and ED. There are clearly other factors that can contribute to the appearance of addiction and ED. Repeated engagement in drug use or other addictive behaviors causes a neuroadaptation at a neuronal level in motivational circuitry that leads to impaired control over further drug use or engagement in addictive behaviors. As a result, a reward deficit situation is created that leads to wanting more. Cognitive and affective distortions, which are often a target of treatment for both SUD and ED, impair perceptions and compromise the ability to deal with feelings, resulting in significant self-deception.

The ASAM definition notes the ABCs of addiction (see Table 13.2), which suggests that addiction is more than a behavioral disorder; it affects "*cognitions*, *emotions*, *interactions with others*, *ability to relate to family*, *community*, *themselves*, *and the Transcendent*." These criteria readily apply to bulimic-spectrum ED and to some extent AN-R (Kaye, Bulik, Thornton, Barbarich, & Masters, 2004).

Table 13.2 American Society of Addiction Medicine (ASAM) ABCs of addiction as applied to substance use disorders (SUD), behavioral addictions (BA), bulimic eating disorders (bED), and anorexia nervosa, restricting type (AN-R)

	SUD/BA	Bulimic ED	AN-R
• Inability to consistently **A**bstain	+	+	–[a]
• Impairment in **B**ehavioral control	+	+	±
• **C**raving	+	+	–[a]
• **D**iminished recognition of problems	+	+	+
• A dysfunctional **E**motional response	+	+	+

[a]Applies to dieting, exercising, and/or pursuit of the thin ideal

In summary, both the DSM-5 criteria for "other substance-related disorders" and the ASAM criteria for addiction (with a few exceptions) appear to be consistent with current understanding of the ED, particularly bulimic-spectrum disorders.

13.4 Similarities and Differences Between Eating Disorders and Addictions

Many people have major misconceptions about the nature of addictions and ED. They are not conditions that individuals choose to have or develop. These are not desired conditions even though, to some extent, lay persons and many health professionals see them this way.

The notion of behavioral addictions or process addictions was described many years ago (Korolenko, 1991; Goodman, 1990), but it is only recently that this notion has begun to be accepted by mainstream psychiatry and psychology (Goodman, 2008; Smith, 2012) (see Chap. 18). Dieting, exercise, binge eating, and purging can be described as process addictions even though binge eating involves a substance, i.e., food, albeit a heterogeneous one chemically. Gambling, sex and love addiction, computer/Internet, and compulsive buying are other presumptive behavioral or process addictions that have been described in both SUD and ED populations, particularly those with bulimic symptoms (Korolenko, 1991; Goodman, 1990). One commonality between these conditions is that afflicted individuals tend to be elevated on measures of novelty seeking, sensation seeking, and impulsivity (see Chap. 6).

13.4.1 Dieting, Restricting, and Starvation Dependence

Severe dieting, food restriction, and/or fasting can progress to the point of inducing a starvation state. Several investigators described the concept of starvation dependence in AN (Luby, Marrazzi, & Sperti, 1987; Marrazzi & Luby, 1986; Marazzi et al., 1990; Szmukler & Tantam, 1984). Based on clinical observations and sharing of fundamental features between dieting and addictions, they argued for an

"auto-addiction" model of chronic AN in which severe dieting is potentially addicting and AN is viewed as a dependence disorder. This model proposes that endogenous opiates are released during the initial period of dieting or prolonged food deprivation that creates a psychological "high" and, in turn, initiates and reinforces a state of starvation dependence. This theory goes a long way toward explaining the intractable nature of the disorder as well as what is so reinforcing about dieting. Dr. Tom Insel (2013), the Director of the National Institute of Mental Health, recently stated in regard to AN, "I think about it as an addiction." There are a variety of studies that shed light on this theory.

For example, Kaye, Pickar, Naber, and Ebert (1982) reported increased endogenous opioid activity in the CSF of severely underweight patients with AN, although the same authors later reported decreased CSF levels of beta-endorphin as well as its three sister peptides, beta-lipotropin, adrenocorticotropic hormone (ACTH), and pro-opiomelanocortin (POMC), in underweight AN patients (Kaye et al., 1987). Brambilla and associates (1995) reported that patients with AN (both restricting type (AN-R) and binge-purge type (AN-BP)) had significantly higher lymphocyte concentrations of beta-endorphin than in controls. This was especially true for patients with AN-BP. Marrazzi, Luby, Kinzie, Munjal, and Spector (1997) reported elevated levels of endogenous plasma alkaloids in patients with AN and BN in comparison to controls, although there have been no new studies in this century of this idea. Taken together, these data indicate that the opioid system is dysregulated in AN during the low weight state and they are compatible with the auto-addiction model of AN. Results using the opiate antagonist naltrexone in the treatment of ED have been mixed, with the best studies showing no effect compared to placebo (Marrazzi, Bacon, Kinzie, & Luby, 1995; Marrazzi, Markham, Kinzie, & Luby, 1995; Mitchell et al., 1989). Therefore, the auto-addiction model of AN has not been supported by pharmacologic studies to date.

Results from a recent fMRI study were interpreted to be in support of the starvation dependence model (Fladung et al., 2010). Patients with AN and healthy controls underwent a functional MRI during evaluation of visual stimuli. Subjects were shown different images consisting of underweight, normal weight, and overweight whole body images and asked to process each image in a self-referring way. Healthy controls had a pleasurable reaction to the normal weight body image (compared to the other two images), while AN patients had a much more pleasurable reaction to the thin body image (compared to the other two images). Given that activation in the ventral striatal reward system was higher during processing of underweight stimuli in the AN patients, the authors concluded that this differential activation toward disease-related stimuli was consistent with theories of starvation dependence.

Despite these findings that are compatible with the auto-addiction hypothesis of AN and the similarities of AN with addictions, many ED investigators are not convinced (Barbarich-Marsteller, Foltin, & Walsh, 2011). They emphasize "fundamental differences" between AN and SUD and conclude that "AN is not an addiction in and of itself." They note differences in several important areas, which are briefly reviewed in Table 13.3. The goals of SUD v. AN are different

Table 13.3 Differences between substance use disorders (SUD) and anorexia nervosa (AN) (adapted from Barbarich-Marsteller, Foltin, & Walsh, 2011)

	SUD	AN
Goal	Pursue drug	Pursue dieting/weight loss
Time course	Immediate	Immediate and long term
Reward	Intoxication	Hunger, self-control, thinness
Cultural consequences	Negative reinforcement	Positive reinforcement
Societal acceptance	Less acceptable	More acceptable
Addicted to?	Psychoactive substance	Absence of food intake
Obsessing about?	Using	Not using

in that the substance abuser is pursuing an immediate intoxication, while the person with AN is pursuing both the immediate and long-term effects of dieting, i.e., thinness and the illusion of control. The authors also point out important differences in the cultural and social consequences of SUD v. AN. Dieting and thinness are much more socially acceptable than the relative negative attitude toward and consequences of substance dependence. Finally, the other obvious difference is in what individuals with SUD v. AN are addicted to, i.e., using a psychoactive substance (drug) v. not using a substance (food). The authors do mention that "the excessive exercise observed in some individuals with AN may more closely resemble the pattern of drug use among substance abusers" (see below and Chaps. 7 and 28).

13.4.2 Exercise as Addiction

Excessive exercise is another problematic behavior associated with both AN and BN that has been described as an addiction (Berczik et al., 2012; Freimuth, Moniz, & Kim, 2011). This topic is extensively discussed in Chap. 7 and therefore will not be reviewed here in detail. However, the authors conclude that the concept of exercise addiction is indeed supported by the literature, although compulsive exercising may be a preferred term when it is associated with ED.

There have been a number of psychophysiological hypotheses to explain exercise addiction, and these include (1) a thermogenic hypothesis, (2) a catecholamine hypothesis, (3) an endorphin hypothesis (which is most widely known and empirically studied), (4) a serotonin hypothesis, and (5) a brain wave asymmetry hypothesis (Krivoschekov & Lushnikov, 2011). Additionally, there are two types of exercise addiction. In primary exercise addiction, the physical activity per se is an object of dependence. Secondary exercise addiction (most often seen in association with ED) appears to be related to decreasing body weight or to change the body's shape or size.

13.4.3 Binge Eating and Food Addiction

This section will discuss the issues of binge eating and food addiction. Patients with bulimic-spectrum disorders may binge on any type of foods, and the proportion of macronutrients in binge meals is no different from that of non-binge meals (Brewerton, Murphy, & Jimerson, 1994; Walsh, Kissileff, & Hadigan, 1989). However, carbohydrates are the predominant macronutrient ingested during binge eating, and these are often simple, high glycemic carbohydrates (or sugars) in combination with fats, thereby making these foods highly palatable and rewarding (Hadigan, Kissileff, & Walsh, 1989; Yanovski, 2003).

An emerging body of evidence over the last few years has characterized highly palatable foods as potentially addictive and evidence to support this idea that they act much like licit and illicit substances of abuse in the brain has been extensively documented (Avena, Gold, Kroll, & Gold, 2012; Avena, Rada, & Hoebel, 2008, 2009; Avena, Wang, & Gold, 2011; Benton, 2010; Fortuna, 2010; Gearhardt, Corbin, & Brownell, 2009; Gearhardt, White, & Potenza, 2011; Gearhardt et al., 2012; Gearhardt et al., 2011; Gold, Graham, Cocores, & Nixon, 2009; Hoebel, Avena, Bocarsly, & Rada, 2009; Joranby, Pineda, & Gold, 2005; Liu, von Deneen, Kobeissy, & Gold, 2010; Lustig, 2010; Sheppard, 2009). Specifically, sugar, fat, salt, and caffeine, which are common components of fast-food menus, have all been posited to have addictive properties (Cocores & Gold, 2009; Garber & Lustig, 2011). Eating such foods in excess may therefore become another strategy, just like any other addictive substance or behavior, that traumatized individuals use to numb themselves from unpleasant feelings and memories and to decrease emotional arousal (Brewerton, 2011) (see Chap. 17).

In support of this concept, Hirth, Mahbubur, and Berenson (2011) surveyed over 3000 women attending five public health clinics in Texas in regard to their (1) ED behaviors, (2) fast-food and sugary soda consumption, and (3) PTSD symptoms. The researchers reported a statistically significant link between PTSD symptoms and the frequency of (1) fast-food and sugary soda consumption and (2) ED symptoms, including severe dieting, purging, and compulsive exercising, but not with BMI. This study was the first to demonstrate a relationship between PTSD (and hence, trauma history) and eating specific types of foods known to be relatively unhealthy and associated with the concept of food addiction, i.e., highly palatable foods containing high concentrations of simple sugars and saturated fats, as well as salt and often caffeine (see Chaps. 1 and 4). The relationships between bulimic-spectrum ED and SUD to prior traumas and subsequent PTSD are discussed in detail in Chap. 17. Although the results of the Hirth et al. (2011) study did not find that women with PTSD had higher BMIs than those women without such symptoms, the women in this study were young, averaging only 20.8 years, and the effects of chronic excessive intake may not have had time to manifest. They were also engaging in more strategies to lose weight, i.e., dieting, vomiting, and smoking behaviors, which probably counteracted the inevitable weight gain that a steady diet of fast foods would incur.

The notion of food addiction is not a new idea, despite the surge in recent interest. Many individuals who have struggled with binge eating or compulsive overeating have independently and spontaneously identified the problem as a "food addiction." Many patients present with the chief complaint, "I have a food addiction," or "I'm addicted to food," or some variation of this statement. Interestingly, in a groundbreaking paper published well over 55 years ago, Randolph (1956) simultaneously described addictive drinking (of alcohol) and addictive eating, which he termed "food addiction." Although alcohol addiction continued to be a major focus of clinical research, nothing was written on the topic of food addiction until many years later.

However, largely inspired by the success of Alcoholics Anonymous (AA), Overeaters Anonymous (OA) was founded in 1960 in Los Angeles, California, by individuals self-proclaimed to be addicted to food. OA defined compulsive overeating as a progressive, addictive illness and focused on processed sugar as the addicting substance, a supposition that is no longer so implausible (Avena, 2010; Avena, Rada, & Hoebel, 2008, 2009) (see Chap. 1). Nevertheless, clinicians and researchers in the ED community have largely discounted that model and still do. This stance has inadvertently alienated many professionals in the addiction community as well as many patients who perceive their ED as an addiction. It is thought by many that one cannot possibly be addicted to food, since it is necessary for life, and the brain requires glucose to function properly. Carbohydrates and fats are basic energy sources. So how could food be addictive when it is required for survival? On the other hand, the modern Western diet contains inordinately high concentrations of high glycemic sugars and saturated fats that are engineered to be more and more irresistible and hedonically pleasing. The dosage and form of a substance determine its addictive potential, e.g., coca leaf (unprocessed) v. cocaine (processed).

Nevertheless, those individuals with ED have often described using food to self-medicate as well as being addicted not only to binge eating but also purging, dieting, and/or exercising. Often these behaviors are done in concert with each other in cyclic fashion, i.e., dieting, bingeing, and purging. Not only do these behaviors become habitual methods of regulating negative affect or mood (see Chap. 16), but eating foods with high hedonic value can rapidly, legally, and cheaply offer a measure of comfort, stimulation, and alleviation of psychic pain, however fleetingly. Of course, only more negative consequences follow as this pattern of behavior becomes habitual (addictive) in susceptible individuals who are exposed to frequent and high doses. A convergence of knowledge has led modern clinical neuroscience to recognize that certain foods can act like addictive substances in the brain despite the fact that they do have other peripheral metabolic effects that substances of abuse do not have.

Both animal and human experiments show that food intake and drug use each cause dopamine release in parts of the brain that mediate pleasure and emotion and that the degree of dopamine release correlates with the subjective sense of reward or experience of pleasure from both food and drug use. Similar patterns of brain activation as seen on fMRI in response to food and drug use have also been

described (Gearhardt et al., 2011; Volkow, Wang, Fowler, & Telang, 2008; Volkow & Wise, 2005) (see Chap. 4). In the case of food, reward mechanisms override homeostatic mechanism involved in regulation of feeding and metabolism (Lutter & Nestler, 2009).

Dr. Nora Volkow, the Director of the National Institute on Drug Abuse (NIDA), is a major proponent of food addiction in humans. Volkow and Wise (2005) stated, "To the degree that drugs and food activate common reward circuitry in the brain, drugs offer powerful tools to understand the neural circuitry that motivates food-motivated habits and how the circuitry may be hijacked to cause appetitive behaviors to go awry" (p. 555).

Dr. Bart Hoebel, the eminent Princeton neuroscientist who dedicated his career to studying the mechanisms of feeding and appetite regulation, and his colleagues at Princeton made the following statement: "Rats with intermittent access to food and a sugar solution can show both a constellation of behaviors and parallel brain changes that are characteristic of rats that voluntarily self-administer addictive drugs. In the aggregate, this is evidence that sugar can be addictive" (Avena, Rada, & Hoebel, 2008, p. 15).

Other studies demonstrate that food can stimulate the opiate system, and there are striking similarities in use and withdrawal patterns of sugar and classical drugs of abuse in certain animal models (Avena, Rada, & Hoebel, 2008) (see Chap. 1). There often appear to be reciprocal relationships among food and other substances. Individuals often gain weight when they stop smoking or drinking (Saules, Pomerleau, Snedecor, Brouwer, & Rosenberg, 2004). Recent research has shown that BMI is inversely proportional to alcohol intake, so the bigger one is, the less likely one is to drink (Kleiner et al., 2004). The authors noted that obese women have lower rates of alcohol use than those found in the general population of women. As BMI increases, lower rates of alcohol consumption are seen. Overeating may compete with alcohol for the same dopaminergic receptor sites in the brain, making alcohol ingestion less reinforcing. In addition, research shows that obese subjects who have lost weight following bariatric surgery sometimes start drinking excessively (see Chap. 9). King and colleagues (2012) reported that approximately 10–15 % of individuals who lose the weight from bariatric surgery and keep it off, 2–3 years down the road, are starting to report higher levels of drinking behavior. Taken together, these observations support the conclusion that food and classic addictive substances compete for the same pathways in the brain and may serve the same purposes psychologically. However, there may be other physiological explanations for these findings in the post-bariatric surgery population (see Chap. 9).

This reorientation of scientific research toward entertaining and validating the concept of food addiction has been a result of the fact that at least one-third of people in the USA are obese and that this number seems to be only increasing. Mortality and morbidity associated with obesity have become a major focus throughout medicine and public health. Using an extremely large database of two samples of over 39,000 subjects each, Grucza et al. (2010) demonstrated a link between risk for familial alcoholism and obesity, particularly in women. The

authors speculated that this link has emerged in recent years due to an interaction between a changing food environment and predisposition to alcoholism and related disorders.

13.4.3.1 Risk Factors

The argument that highly palatable foods can be addicting to certain people, especially traumatized people, is very compelling. As with other addictions, when exposed to substances such as alcohol, nicotine, illicit drugs, or behaviors, such as gambling, a certain subset of people are going to be highly rewarded by these substances or behaviors and will continue to use or engage in these behaviors. Others will say, "not for me," whereas others will want more and more, particularly those with reward deficiency. What do we know about those risk factors that will help us identify those who are at greatest risk for developing addictions? Interestingly, individuals with reduced dopamine type 2 receptor availability have a predisposition to both obesity and SUD (Tomasi & Volkow, 2013; Volkow et al., 2008; Volkow, Wang, Telang, et al., 2008; Volkow & Wise, 2005; Wang et al., 2001). Additionally, evidence suggests that engaging in addictive behaviors further downregulates D2 receptors and a reward deficiency syndrome is created. Given these interrelationships between addiction and obesity, a "reward deficiency syndrome" has been proposed to describe individuals with low D2 receptor density and polymorphisms of the D2 gene. This condition increases the risk for substance abuse, including alcohol dependence, heroin craving, cocaine dependence, methamphetamine abuse, nicotine sensitization, and glucose craving (Blum et al., 2011; Blum, Gardner, Oscar-Berman, & Gold, 2012; Blum, Liu, Shriner, & Gold, 2011).

In addition, obese subjects with BED have been reported to have an increased prevalence of the G/G (Comings & Blum, 2000) allele (A118G) of the μ-opioid receptor (OPRM1) (Davis et al., 2009), which has been associated with greater sensitivity to reward and an increased preference for sweet and fatty foods (Davis et al., 2011) and higher rates of substance addiction (Miranda et al., 2010; Ramchandani et al., 2011). Such sensitivity to reward is a personality trait that has been linked to both obesity and drug addiction. This predilection toward lower reward sensitivity is illustrative of the reward deficiency hypothesis that results in compensatory overconsumption (Comings & Blum, 2000).

Other risk factors for both SUD and ED are environmental, such as a history of sexual, physical, or emotional abuse or experiencing/witnessing interpersonal violence (see Chap. 17). Any traumatic experiences that produce PTSD or pPTSD symptoms are risk factors (Brewerton, 2004, 2007; Dansky, Brewerton, O'Neil, & Kilpatrick, 1997; Mitchell, Mazzeo, Schlesinger, Brewerton, & Smith, 2012). Given what is known about gene and environmental reactions, many genetic disorders will be manifested only when the environmental triggers are present. It is further validation of the self-medication hypothesis of PTSD or of addiction. Victims of violence not only resort to addictive and/or ED behaviors and smoking to alleviate psychic pain but also preferentially select highly palatable foods containing high concentrations of sugar, fat, salt, or caffeine or the combination, sometimes to the point of addiction, in an attempt to dampen arousal and facilitate

numbing and avoidance, which are specific symptoms of PTSD (Brewerton, 2011; Schoemaker, Smit, Bijl, & Vollebergh, 2002).

These environmental risk factors are particularly relevant to children. It is well known that traumatic experiences are extremely common in children, who are especially vulnerable to the complex neurodevelopmental changes of early stress and addiction. Children are also quite likely to be exposed to fast foods that can subsequently lead to the use of these foods to self-medicate negative mood states. It has already been reported that food addiction is very much a feasible concept in children (Merlo, Klingman, Malasanos, & Silverstein, 2009; Pretlow, 2011). It has become nearly impossible to find foods without added sugar, especially high fructose corn syrup (HFCS), in grocery stores. Not only are these foods highly palatable and potentially addicting, evidence has suggested that those that contain high concentrations of HFCS may be literally toxic (Lustig, 2010).

Interestingly, animal studies inform us further about the intimate interrelationships between eating, stress, and relief (Maniam & Morris, 2012). A highly palatable cafeteria diet consisting of high fat and high sugar content ameliorates anxiety and depression-like symptoms in rats exposed to very early life adversity, i.e., with early separation (Maniam & Morris, 2010). This effect is mediated by the effects of feeding on the glucocorticoid receptor gene, a very fundamental early effect that involves food as a direct comfort to the nervous system. The anatomical circuits underlying feeding and stress responses are interconnected and operate in dual directions. What this means is that palatable food can actually reduce central stress responses at a genetic level, so comfort food works at a deep biological level to soothe the early effects of stress.

13.4.3.2 The Yale Food Addiction Scale

Gearhardt, Corbin, and Brownell (2009) developed and validated the Yale Food Addiction Scale (YFAS), which was based on the DSM-IV criteria of substance dependence among other measures. The goal of this study was to determine if there was a subset of BED patients that met the criteria for "food addiction." The authors administered the YFAS to 81 obese patients with BED, along with a number of other measures of psychopathology. The investigators were able to classify 57 % of this group of BED patients as *also* having food addiction. Those who were classified as such had significantly higher levels of depression, more negative affect, more emotional dysregulation, more ED psychopathology, and lower self-esteem (Gearhardt et al., 2012). These YFAS scores were significant predictors of binge eating frequency above and beyond other measures. This food addiction group identifies, even within BED, a particularly disturbed variant characterized by greater ED psychopathology and associated pathology. In another study of 96 obese patients with BED, a classification of food addiction was met by 39 (41.5 %) of BED patients (Gearhardt, White, Masheb, & Grilo, 2013). Similarly, those meeting YFAS food addiction criteria had significantly higher levels of emotion dysregulation, negative affect, and eating disorder psychopathology and lower self-esteem than those not meeting these criteria. In addition, higher scores on the YFAS were related to an earlier age of first being overweight and dieting onset.

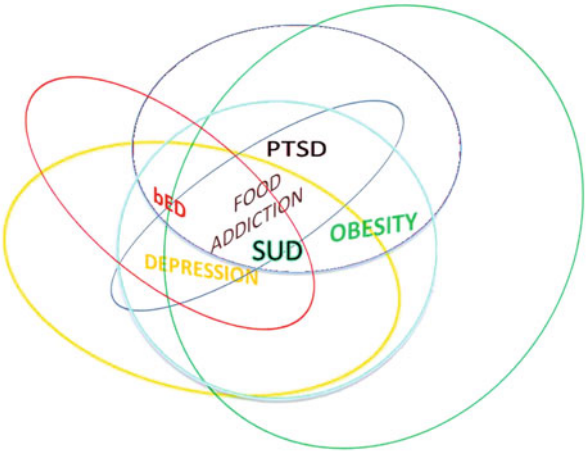

Fig. 13.1 Venn diagram depicting relationships between bulimic eating disorders (bED), substance use disorders (SUD), food addiction, posttraumatic stress disorder (PTSD), depression, and obesity

YFAS scores were also significant predictors of binge eating frequency above and beyond other measures. Unfortunately, the authors did not measure trauma history or PTSD in either of these studies. However, Mason, Flint, Field, Austin, and Rich-Edwards (2013) reported that 8 % of 57,321 women in the National Nurses' Study met criteria for food addiction using the YFAS, and the diagnosis of food addiction was significantly associated with a history of severe childhood physical and sexual abuse as well as obesity. Based on these findings, there appears to be a group of individuals (with and without a diagnosis of BED) who meet the criteria for "food addiction" based on the YFAS. See Fig. 13.1 that depicts the interrelationships between bulimic ED, food addiction, obesity, depression, SUD, and PTSD (Brady, Killeen, Brewerton, & Sylverini, 2000).

13.4.3.3 The Case Against Food Addiction

Several authors have been ardent critics of the food addiction hypothesis and have made a number of useful objections and arguments (Benton, 2010; Wilson, 2010; Ziauddeen, Farooqi, & Fletcher, 2012; Ziauddeen & Fletcher, 2013). The salient features of the case against food addiction will be summarized here.

1. One question involves the specific identity of the so-called addicting substance, and why it does not manifest as the preferred substance in laboratory studies of eating behavior? The macronutrient content of meals consumed by individuals with BN and BED has not been shown to substantially differ from that of controls, and carbohydrates (CHO) do not necessarily improve mood or relieve negative affect (Wilson, 2010). These are no doubt fair points. Nevertheless, the most commonly consumed types of food by binge eaters and non-binge eaters alike are CHO and FAT. Thus, the combination of CHO and FAT in much larger quantities, or doses, is in fact what binge eaters are most likely to consume (Brewerton, Murphy, & Jimerson, 1994; Hadigan et al., 1989), and the dose of a substance is an important factor in whether it causes addiction. What has not

been well documented in laboratory studies of binge eating is to what extent high glycemic versus low glycemic CHO are preferred by binge eaters, much less binge eaters who are also "food addicts." However, Hadigan et al. (1989) noted that patients with BN spent more of their mealtime eating dessert and snacks than did control subjects, and they also began their dessert and snack consumption earlier than control subjects. Tanofsky-Kraff and coworkers (2009) reported that children with loss of control (LOC) binge eating (a core feature of binge eating and food addiction) consumed more high-calorie snack and dessert-type foods than did those without LOC when presented with an array of different types of foods. In another study of patients with BED, Yanovski and colleagues (1993) reported that adult patients with BED consumed a greater percentage of energy as fat during a binge meal as well as a lesser percentage as protein than did subjects without BED. In other words, available data would suggest that it is highly palatable foods, which are both sugary and fatty that may be the potentially addicting combo. Nevertheless, the specific addicting agent or agents do need to be further clarified in controlled, scientific studies.

2. Wilson (2010) claims that "epidemiological data are inconsistent with an addiction model" of bulimic ED. Although higher rates of SUD are seen in bulimic ED, and vice versa, he rightly points out that substance abuse is not specific to ED and that depression is a more common comorbid disorder for both SUD and ED.
3. Wilson (2010) also notes some evidence that SUD and ED do not show evidence of a shared genetic or familial etiology. However, the results are mixed. This issue is discussed at length in Chap. 5.
4. One of the best arguments against the food addiction model is the simple fact that the core psychopathology of bulimic-spectrum ED is more complex than simply binge eating. This includes the "undue influence of body weight or shape on self-evaluation" as well as the "overvaluation of body weight or shape" (American Psychiatry Association, 2013) that is characteristic of all ED types and subtypes (Wilson, 2010). Individuals with BN, and to some extent, also those with BED, engage in significant dietary restraint and are distressed by their inability or difficulty controlling their eating. Wilson (2010) quotes Fairburn (1995) that "there is no equivalent phenomenon in SUD." However, this is highly disputable and at best doubtful. Certainly there are individuals with SUD who want to stop using but because of impaired control and/or distress intolerance repeatedly relapse and are subsequently distressed, demoralized, and/or depressed by their failed efforts to maintain abstinence.
5. Another important argument against the food addiction concept is the lack of evidence for withdrawal effects in humans, which has been discussed earlier. However, the food addiction field is new and good studies have not yet been designed or implemented to test this hypothesis in humans. In addition, it is notable that without exception every substance that has ever been found to be addicting in animals has subsequently been shown to be addicting in humans.
6. Wilson (2010) also correctly makes the point that outcome research clearly supports the efficacy of CBT in the treatment of BN and BED (see Chap. 24),

while the "diametrically opposed" 12-step approach has never been studied in bulimic ED (see Chap. 27). However, he does not mention that CBT is also effective for a variety of SUD and related comorbid disorders (see Chap. 24). In fact, in Project MATCH (Project MATCH Research Group, 1997), response to CBT was found to be no different from 12-step facilitation therapy or from motivational enhancement therapy (MET) in a large group of patients with alcohol abuse followed long term. Furthermore, the efficacy of CBT for BN and BED in no way disproves the food addiction hypothesis. It is highly questionable that CBT is "so effective" that it "represents a refutation of the addiction model." In many of the major trials of CBT in BED and BN, major comorbid disorders such as SUD were excluded, while other trials have shown that comorbidity is a negative prognostic factor (Castellini et al., 2012). Finally, the abstinence model does not necessarily "encourage dietary restriction" in the classical sense of dieting, generalized food restriction, or restrained eating. Instead certain foods or types of foods are eliminated, while a healthy meal plan consisting of all major food groups, vitamins, and minerals is encouraged or prescribed (see Chap. 23).

7. Politically and socially speaking, many people object to the term "addiction" in reference to food or ED given its negative connotations. As one prominent ED researcher said to me, the term has "baggage," and this is partly why the term was avoided for so long by the American Psychiatric Association (O'Brien et al., 2006). In addition, if addiction is a chronic disease, then what hope is there for those with ED to fully recover? Presumably a substantial number of ED patients do fully recover from their overt symptomatology (Fichter, Quadflieg, & Hedlund, 2006; Fichter et al., 2008; Strober, Freeman, & Morrell, 1997). However, there are important personality traits, such as perfectionism, obsessionality, harm avoidance, novelty seeking, and impulsivity that remain and may predispose to relapse. Is it accurate that patients with SUD can never fully recover? Probably not. But there has been intense philosophical debate as to whether anyone with a SUD or an ED is ever fully "recovered" versus always "recovering," which is more traditionally the position of 12-step programs. However, there is very little good, comprehensive data to illuminate this issue. The fact of the matter is that both SUD and ED patients have highly variable courses, with some doing extremely well and never relapsing on one end of the spectrum and some never improving and dying on the other.

13.4.4 Purging as an Addiction

What about purging as an addiction? Purging specifically refers to vomiting, laxative abuse, and/or diuretic abuse, each of which will be discussed in terms of its addictive potential. Vomiting is the most common compensatory behavior associated with BN, AN-BP, and subclinical ED (see Chap. 11).

13.4.4.1 Vomiting

Consider the following statement by a young woman in a blog on the Internet: "*I am only 17 years old, and I'm scared that I am becoming bulimic. One day I was crying very hard, so hard that I vomited, and after vomiting I felt relieved. What happened is that after that day, each time I'm either crying, sad, or angry, I either force myself, (which is so very easy to me), or naturally vomit. It's only after vomiting that I feel relieved and all right. I can't stop doing it because it makes me feel so much better. Am I addicted to it? Is it unhealthy? I haven't had any weight loss or anything like that.*"

This perspective provides a different twist on vomiting behavior, which in this case is not only being used as a modulator of mood but is also clearly becoming habitual to the point that she is concerned about whether she is "addicted." This case illustrates an under recognized aspect that vomiting is not just about "undoing" the effects of binge eating and getting rid of calories. It is often just as much about the emotional regulation effect that it has. Like binge eating, vomiting may have a distinct calming effect, both simultaneously decreasing negative affect and increasing positive affect (see Chap. 21). After binge eating, individuals are typically extremely anxious and even panicked about the increased intake and inevitable resultant weight gain, and vomiting is a way of relieving the physical and mental distress, calming down, getting rid of, and feeling better. Sometimes it happens that people get so upset that they actually vomit as a result of the emotional activation. This can happen naturally, as in "psychogenic vomiting," which appeared in the literature well before BN was described (Hill, 1968). In fact, Breuer and Freud (1893) noted that chronic vomiting was associated with emotional disturbances well over 100 years ago.

Vomiting results in release of the major stress hormone corticotropin-releasing hormone (CRH), which stimulates ACTH to release cortisol from the adrenal glands (Abraham & Joseph, 1986–1987; Kaye, Gwirtsman, & George, 1989). Plasma cortisol is known to increase after bingeing and purging, and with repeated episodes, it remains elevated for some time (Fullerton, Swift, Getto, & Carlson, 1986). So vomiting both relieves and creates stress at the same time. Whenever the stress hormone cortisol is stimulated, beta-endorphin is automatically released as well because it is cleaved off of the larger pro-opiomelanocortin molecule. When stressed, the body provides an endorphin pain relief. Evolutionarily, stress meant threat, which in turn meant possible impending pain, so a self-protective beta-endorphin release is produced, and this happens with vomiting. Over time, repeated vomiting causes a depletion of this peptide and opiate-receptor downregulation because of these chronic behaviors, and intermittent binge eating and semi-starvation only add to that. As discussed previously, the opiate effect of dieting results in decreased central beta-endorphin over a time, and more vomiting may be required to produce the same opioid effect.

This hypothesis is compatible with other findings. There are several studies that show that ED patients have a higher pain threshold, probably because of this opiate effect (Abraham & Joseph, 1986–1987; Stein et al., 2003). Brewerton and colleagues (1992) reported significantly decreased cerebrospinal fluid (CSF) levels

of beta-endorphin in women with BN in comparison to healthy controls. CSF concentrations of beta-endorphin were inversely correlated to measures of depression, suggesting that bingeing and vomiting aggravate mood disturbance, much like the secondary depression seen in SUD.

In desperation, some people have resorted to using emetine (syrup of ipecac) to induce vomiting. Emetine is a medicine previously used in emergency rooms to induce vomiting after poisonings. It is extremely toxic to muscle tissue, especially the heart, and its toxicity is cumulative over time. Syrup of ipecac used to be available over the counter, but it was taken off the shelves because of its toxic side effects. In addition, since 2010, its production has been discontinued. However, as of this writing, it can still be obtained online. The use of emetine to facilitate vomiting illustrates the compulsory or addictive nature of vomiting, which can become a goal that must be accomplished at all costs.

Recurrent, chronic vomiting may also result in dopamine depletion and therefore may predispose toward other addictive behavior. In a prospective study, Field et al. (2012) reported that female participants with purging disorder (PD) had a significantly increased risk of starting to use drugs (OR: 1.7) and starting to binge drink frequently (OR: 1.8).

13.4.4.2 Laxatives

The abuse of laxatives is a well-known compensatory behavior seen in patients with bulimic-spectrum disorders (see Chap. 12). The stimulant type of laxatives is the most dangerous since they typically produce excessive fluid and electrolyte losses leading to a variety of complications (see Chap. 15). Laxatives have very little effect on caloric absorption but rather cause evacuation of the colon and significant fluid and mineral losses. Laxatives, which are often taken in overdose, do produce weight loss resulting from diarrhea, loss of water weight, and resultant dehydration, as well as major discomfort due to abdominal and muscle cramping. The decrease in the number on the scale is highly rewarding, but then there is rebound water retention and edema that leads to anxiety and more abuse, and a vicious addictive cycle is established. Laxatives are not addictive per se in the classical sense (producing intoxication), but it is the process of using them repeatedly to excess with the mind-set that weight loss will be achieved no matter what that is problematic. Tolerance builds with stimulant laxatives, and abusers take more and more to achieve the desired effects. Therefore, the use of laxatives can be thought of as a behavioral or process addiction.

13.4.4.3 Diuretics

The use of diuretics is less common than vomiting or laxative abuse but nevertheless occurs in a substantial minority of patients. Diuretics have no effect on caloric absorption whatsoever, but individuals may resort to using them in an attempt to decrease bloating and swelling, which may be in part rebound phenomena from bingeing, vomiting, and/or laxative abuse. As with laxative abuse, weight loss is achieved via loss of water weight. The decreased weight on the scale has an extremely powerful reinforcing effect; however, chronic diuretic use leads to

rebound edema and in turn leads to further use. Diuretic abuse can cause major medical problems, including chronic kidney disease from hypokalemic nephropathy.

13.5 Treatment Implications

Why does it matter whether ED are called addictive disorders or not? How might one treat dieting, exercising, binge eating, and purging using the insights gleaned from an addictive perspective? From one standpoint, it may not matter at all. The treatment goals are similar if not identical for most behaviors. Normalization of weight and the cessation of dieting, excessive exercising, binge eating, purging, and the use of harmful substances to facilitate weight loss remain primary goals, followed by reduction of anxiety, normalization of mood, and resolution of traumatic or conflictual issues. Ultimately, acceptance of self, including body self, is a goal common to ED. However, how these goals are approached and achieved may be modified as discussed below using an addiction perspective.

13.5.1 Dieting

Diets don't work in the long term for the treatment of obesity, as most people regain all of their lost weight and more regardless of the type of diet or pharmacologic agent employed. Extreme dieting is a well-recognized risk factor for the initiation of bulimic-spectrum disorders (Brewerton, Dansky, Kilpatrick, & O'Neil, 2000) and a trigger for relapse. A core goal of treatment for all types of ED, including food addiction, is *abstinence from dieting*. Diet is a "4-letter word" in the ED world. Instead of using the word "diet," which implies restriction of intake or restrained eating, it is preferable to use the term "meal plan." Following a reasonable, well-balanced meal plan is an essential part of nutritional rehabilitation, weight restoration and/or stabilization, healthy eating, and a healthy life. This is an essential ingredient to the nutrition therapy and cognitive behavioral therapy for ED (see Chaps. 23 and 24).

13.5.2 Exercising

Since weight restoration is a primary goal in the treatment of AN, exercising and resultant energy expenditure are generally antagonistic to this process and often add danger to the medically compromised. ED patients who have been abusing exercise often need a rest. It is anti-therapeutic to be doing much of any kind of aerobic exercise, certainly, in the weight gain phase and in the early phases of treatment for BN—until medical stabilization. Initially, very gentle stretching or yoga or meditation is allowed and even encouraged as an anxiety-reducing strategy, but any intense exercise is not recommended. In a later phase of treatment, exercising to

moderation is encouraged in weight-restored patients with AN as well as in individuals with BN and BED. Exercising in moderation is obviously important to overall health and weight maintenance. The degree and frequency of exercise allowed and prescribed depend on the illness being treated and the clinical status at the time. Moderation is the rule, which is part of good CBT. Please see Chaps. 7 and 28 for a more detailed discussion of the treatment of exercise addiction and use of exercise as treatment.

13.5.3 Purging

Complete cessation of all purging behavior is a clear goal of treatment regardless of one's perspective on the addiction model of ED. Vomiting behavior and laxative abuse should be stopped as soon as possible, regardless of any possible withdrawal effects. Patients who have been taking high doses of diuretics chronically usually need to be tapered gradually off of these substances to avoid extreme rebound edema and associated triggering of any and all ED behaviors to compensate and respond to worsening negative affect. If constipation is a problem, the use of stool softeners, liberal fluids, and enhancement of dietary fiber intake are usually sufficient to treat this effectively (see Chaps. 12 and 15).

13.5.4 Binge Eating

The primary goal of any treatment approach is to move toward *abstinence from binge eating* while developing a healthy relationship with food. However, the comorbid presence of food addiction complicates this situation. One can become abstinent from binge eating, but not from food itself. However, the elimination of specific "binge foods," which patients may perceive they are addicted to, may be eliminated at least at the beginning of treatment in order to facilitate abstinence from binge eating. Again, it's a matter of moderation, and sometimes it may be a matter of identifying a hierarchal list of which foods are most triggering. The OA idea of avoiding highly refined, white processed carbohydrates is still an open, unresolved question, but as this chapter has attempted to illustrate, this approach may be highly relevant and appropriate for a subset of individuals with bulimic-spectrum disorders, perhaps those who score high on the YFAS. The high fluctuations in glucose levels that come from ingestion of foods containing high concentrations of simple sugars or high glycemic carbohydrates result in high levels of insulin that in turn drives essential amino acids, such as L-tryptophan and L-tyrosine into cells and across the blood–brain barrier to causes sudden surges in monoamine neurotransmitter levels (Brewerton, 1995). Glucose and insulin very much influence L-tryptophan and L-tyrosine uptake into the brain and subsequent neurotransmitter function, including serotonin, norepinephrine, and dopamine, all of which are prominently involved in ED, particularly serotonin in bulimic disorders (Brewerton, 1995) (see Chap. 3). The intermittent bingeing on "high

doses" of high glycemic carbohydrates can cause an intermittent surge of serotonin and dopamine, which in turn causes downregulation of serotonin and dopamine receptors and a reward deficit. Recent research has revealed that DA and insulin systems do not operate in isolation from each other but rather work together to regulate the motivation to engage in consummatory behavior and to adjust the associated level of reward. Insulin signaling regulates DA neurotransmission and affects the ability of drugs that target the DA system to exert their neurochemical and behavioral effects (Daws et al., 2011).

Carbohydrates are a necessary component of a healthy diet, but the kind of carbohydrates eaten may significantly influence the course of illness. There is sound reason to believe that working toward eating primarily low glycemic carbohydrates may be very advantageous in avoiding binge eating. As previously noted, the idea that some people with binge eating are addicted to high sugar, high fat foods has been empirically validated (Gearhardt et al., 2012, 2013; Gearhardt, White, & Potenza, 2011). No matter how stable one may become, eating certain foods can be triggering and destabilizing. This is not simply a psychological matter but a physiological one as well. Blouin et al. (1993) reported that patients with BN who received double-blind, placebo-controlled injections of glucose experienced heightened urges to binge at 10 and 60 min (compared to placebo), whereas healthy controls experienced reduced food cravings for sweets. Page and colleagues (2011) tested the hypothesis that circulating levels of glucose influence brain regions that regulate the motivation to consume high-calorie foods. The authors demonstrated that mild hypoglycemia preferentially activated limbic-striatal brain regions in response to food cues to produce a greater desire for high-calorie foods. However, euglycemia preferentially activated the medial prefrontal cortex and resulted in less interest in food stimuli. Higher circulating glucose levels predicted greater medial prefrontal cortex activation, but this response was absent in obese subjects. These findings demonstrate that circulating glucose modulates neural stimulatory and inhibitory control over food motivation and suggest that this glucose-linked restraining influence is lost in obesity.

Traditionally, the ED community has not been receptive to the idea of eliminating specific "binge foods." The dietary dogma has been that there are "no bad foods" and that all foods should be eaten in moderation. No foods should be eliminated according to this perspective unless there is a clearly and carefully diagnosed food allergy or intolerance, e.g., gluten intolerance with celiac disease. This viewpoint maintains that one can always eat a little bit of any "forbidden" food and that prolonged exposure will result in a desensitization and extinction of the fear of specific foods. However, as successful as CBT has been for BN and BED, it is not effective for everyone, with on average only 25–50 % of patients in RCT becoming completely abstinent, and relapse rates are high. Perhaps some people with bulimic ED get so triggered by highly refined sugar and fat that they are better off just not being exposed to it, at least for some time. Some patients don't want to be exposed to such foods, and the foundation upon which therapists and dieticians insist that they do is now on shaky ground. It is important to listen to patients, who often tell their treatment providers exactly what they need.

The usefulness of 12-step programs has been discussed in Chaps. 12 and 27, both as a self-help tool and as a therapist-facilitated approach. There is evidence of its utility in the treatment of SUD, but not ED; it hasn't been systematically studied in this population.

What is especially questionable to many professionals in the ED field is OA's prohibition on white flour and sugar. Anecdotally, many individuals report that avoidance of white flour and sugar is extremely useful. Furthermore, the success of low carbohydrate diets, such as the Atkins diet, for the treatment of obesity, attests to the feasibility of this approach, at least in some individuals (Casazza et al., 2012; Iqbal et al., 2010; Maeir et al., 2011). In the treatment of individuals with food addiction, OA can be offered in an unbiased manner as an option to be explored that may or may not work.

Another issue for ED professionals is that OA (like AA in the treatment of alcoholism) is a viable substitute for professional intervention. Conceptualizing an ED as an "addiction" does not imply that professional intervention in the treatment of an ED is not essential. Programs such as OA are considered as adjuncts to treatment, not the foundation of recovery. Twelve-step programs provide needed fellowship, have the advantage of being free and readily available, and are considered the most successful self-help programs worldwide. Patients can be encouraged to attend one or more meetings and decide for themselves if OA philosophy and practice is for them. They can also follow the 12 steps without adhering to the dietary guidelines. This kind of stance with individuals with bulimic ED can be useful (Giannini et al., 1998; Johnson & Sansone, 1993; McAleavey, 2008).

There is a lot of overlap between the types of therapies that are used in ED and addictions, which a large portion of this book reviews. When ED and SUD occur together, the rates of PTSD and prior trauma are higher than with each disorder alone (see Chap. 17). Often the PTSD symptoms are obscured or hidden until the ED symptoms and the addiction symptoms are well under control. In a major way, the treatment of ED and SUD and related comorbidity requires a phasic yet integrated approach to treatment. One starts with weight restoration, nutritional rehab, and/or detox first, and then one moves into the intensive psychotherapy phase, which is followed by the maintenance phase upon successful resolution of symptoms.

The decision of what to focus on first after a full medical and psychiatric evaluation is still debatable, but there is general consensus that it is a matter of which disorder or problem is most life threatening. What symptoms have the greatest lethality? Alcoholic delirium tremens is obviously a life-threatening condition that must take precedence. Severe hypokalemia and associated cardiac arrhythmia or seizures also have high priority. So sometimes it is the SUD and sometimes it is the ED that takes precedence. The medical and psychiatric sequelae of both ED and SUD need to be simultaneously addressed. This is a matter of good clinical judgment in collaboration with patients and their families.

Often when patients are not ready to change, one needs to start with motivational interviewing (MI) or motivational enhancement therapy (MET) to help them get ready to do the work that is required for recovery. Patients are often in different

stages of change depending on the symptom that is being discussed. Some patients are ready to work on ED symptoms but are not ready to address their SUD. Likewise, some patients are actively ready to confront their SUD but not the ED. Programs that offer comprehensive, integrated services for this comorbid condition provide the best opportunity for recovery from both disorders (see Chap. 21).

Conclusions

In summary, there continues to be a significant chasm in both the ED and SUD field in the conceptualization and treatment of patients that present with both an ED and SUD. In the past, similarities between these two disorders have centered on clinical presentation and behavioral similarities. However, emerging evidence in animal research, genetics, and neuroimaging has provided further evidence that supports the addictive nature of ED behaviors, including dieting (starvation dependence), compulsive exercising (exercise addiction), binge eating (food addiction), purging (purging addiction), as well as the addictive use of a variety of substances to promote weight loss (appetite suppressants, lipase inhibitors, ipecac, laxatives, and diuretics). All of these behaviors meet the contemporary definitions of addiction, including those delineated in the DSM-5 and ASAM definitions of addiction. Despite the fact that food and drugs of abuse act on the same or similar central reward networks, food consumption is also regulated by peripheral signaling systems which adds to the complexity of how the body regulates eating and manages pathological eating habits. Traditional pharmacologic and behavioral interventions for other SUD, however, may prove useful in treating obesity and ED, and the field is wide open in terms of the research that needs to be done in this area of overlap. The addiction field and the ED field need to work much more closely together in order to make further progress in research and treatment in order to further delineate the similarities and differences between these two classes of disorder.

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Assessment of Eating Disorders, Substance Use Disorders, and Addictions

14

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Abstract

Assessing eating disorders, substance use disorders, and addictions poses a number of challenges, particularly when these disorders co-occur. Although unstructured interview approaches are often utilized in clinical assessment of eating disorders, substance use disorders, and addictions, the use of semi-structured interviews and questionnaires can potentially increase the reliability of self-reported data. Several semi-structured interviews that assess both eating disorders and substance use disorders can be used, especially to examine diagnostic criteria. In addition, a number of interviews and questionnaires have been designed to assess eating disorders, substance use disorders, and addictions as separate conditions, allowing for the administration of such measures independently or in combination to assess co-occurring eating disorders, substance use disorders, and addictions. Biochemical measures can also be used to assess substance use. Technology-based assessment shows promise for facilitating increased self-disclosure and data reliability. Future research is needed to broaden the accessibility of technology-based assessment as well as to develop comprehensive measures of co-occurring eating disorders, substance use disorders, and addictions.

Keywords

Eating disorders • Substance use disorders • Addiction • Assessment • Chemical dependency • Questionnaires • Alcoholism

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As described in other chapters (e.g., Chaps. 11, 12, and 18), the co-occurrence of eating disorders (ED), substance use disorders (SUD), and addictions (ADC) is characterized by a complex interrelationship among causal and maintenance factors. This complexity creates challenges in both the treatment (see Treatment Perspectives of this volume) and the assessment of ED, SUD, and ADC, particularly given the fact that each of these conditions presents difficulties in assessment independent of their co-occurrence (see reviews by Peterson, 2010, and Hodgins, Diskin, & Stea, 2010). These challenges include broader issues related to limitations in self-report (Schacter, 1999) and more specific problems including the cognitive impact of nutritional deprivation and/or intoxication as well as complex diagnostic criteria that require metacognitive skills for patients and clients to describe accurately (e.g., undue influence of shape and weight in self-evaluation) (Peterson, 2010; Sobell & Sobell, 2003). In addition, the reliability of ED, SUD, and ADC assessment can be compromised by minimization and denial of symptoms as the result of deliberate misrepresentation or a lack of self-awareness (Anderson & Paulosky, 2004; Vitousek, Daly, & Heiser, 1991). Strategies for the accurate assessment of ED, SUD, and ADC include the use of specific instruments such as written self-report questionnaires and semi-structured interviews, and more objective measures including blood alcohol levels and serum toxicology screening. In addition, self-report instruments administered using technology show promise for enhancing data reliability.

Recent changes in the definitions and diagnostic criteria for ED, SUD, and ADC also complicate the accurate assessment of these conditions. As described in the recently published fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5; American Psychiatric Association, 2013), eating disorders are characterized by disturbances in eating behaviors and food intake that impair psychosocial functioning and/or physical health. The DSM-5 includes the following ED conditions: avoidant/restrictive food intake disorder (ARFID), anorexia nervosa (AN), bulimia nervosa (BN), and binge-eating disorder (BED). The DSM-5 also includes specific examples of other specified feeding and eating disorders (labeled eating disorder not otherwise specified in DSM-IV) as well as an unspecified category. Substance-related and addictive disorders in DSM-5 include ten separate categories of substances as well as gambling disorder. The DSM-5 defines SUD as a co-occurrence of cognitive, behavioral, and physiological symptoms indicating ongoing use in spite of related problems and includes criteria for impaired control, risky use, social impairment, and tolerance/withdrawal. In contrast to DSM-IV, DSM-5 has eliminated the distinction between substance abuse and substance dependence and, instead, has a single disorder category for each substance (e.g., alcohol, cannabis, hallucinogens, inhalants, opioids, sedatives, stimulants) that amalgamates the DSM-IV abuse and dependence criteria. Based on the DSM-5 criteria, assessment of ED, SUD, and ADC requires a comprehensive examination of behaviors, cognitions, psychosocial impairment, and physical symptoms.

The following sections summarize the most widely used assessment measures in these categories: (1) interviews that can be used to assess both ED and SUD/ADC,

(2) interviews that specifically measure ED or SUD/ADC, (3) questionnaires that assess both ED and SUD/ADC, and (4) questionnaires that specifically measure ED or SUD/ADC. Additional sections include technology-based assessment administration, data quality enhancement, clinical considerations, and directions for future research.

14.1 Interview-Based Assessment Measures for Both Eating Disorder and Substance Use Disorder Symptoms

Interview-based assessment instruments for ED and SUD/ADC symptoms, in particular, and psychiatric disorders, in general, are currently considered the "gold-standard" measures, based in part on the rationale that a well-trained interviewer can ensure that self-reported symptoms meet the designated criterion (e.g., DSM-5 criteria for a binge-eating episode) (Grilo, 2005; Wilson, 1993). Although limitations of semi-structured interviews include time burden, cost of training, and the possibility that some individuals may self-disclose more information using methods that do not involve face-to-face interactions (Keel, Crow, Davis, & Mitchell, 2002), these types of measures are widely used in research based on the premise that they yield the most accurate data.

The Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I; First, Spitzer, Gibbon, & Williams, 1995), currently being modified for DSM-5, is a semi-structured interview that can be used to diagnose both ED and SUD in adolescents and adults. After an introduction and overview, the trained interviewer administers a series of standard questions (as well as additional probes as necessary to determine if symptoms meet specific diagnostic criteria) to establish current and past diagnoses. The SCID-I can also be used to assess "subthreshold" diagnoses. Considered a gold-standard clinical interview in psychiatric research (Grilo, 2005), the SCID-I has been supported by a number of psychometric studies including those finding excellent inter-rater reliability among inpatients, outpatients, and controls (Loebbestael, Leurgans, & Arntz, 2011) as well as in substance-using adolescents (Martin, Pollock, Buckstein, & Lynch, 2000) along with acceptable ranges of test-retest reliability (Zanarini et al., 2000). The SCID-I is optimal for use in clinical populations (although a non-patient version is also available) in which the main objective is to determine current and/or past diagnostic status for ED and SUD as well as other co-occurring psychiatric disorders (e.g., mood and anxiety disorders). The DSM-5 revised version of the SCID-I will also include criteria for gambling disorder, which were not included in the earlier versions. Two validated DSM-IV clinical interview schedules are available that are similar in style and format to the SCID-I and can be readily incorporated into a SCID-I interview to assess gambling disorder (Grant, Steinberg, Kim, Rounsaville, & Potenza, 2004; Winters, Specker, & Stinchfield, 2002). Children and younger adolescents can be assessed using the child version of the Schedule for Affective Disorders and Schizophrenia (K-SADS; Kaufman, Birmaher, Brent, Rao, & Ryan, 1996) which includes items to diagnose ED and SUD.

The Structured Interview for Anorexic and Bulimic Disorders (SIAB-EX; Fichter, Herpertz, Quadflieg, & Herpertz-Dahlmann, 1998) was designed to assess eating disorder symptoms in adolescents and adults as well as co-occurring psychiatric symptoms and SUD including abuse of tranquilizers, alcohol, and illegal drugs. Like the SCID-I, the SIAB-EX can be used to determine current and past eating disorder diagnoses; in addition, the SIAB-EX also includes dimensional measures of eating disorder symptoms in the form of subscales and can be used to examine severity as well as the presence or absence of eating disorder diagnostic items and substance use. Two studies have supported the psychometrics features of the SIAB-EX including internal consistency, factor structure, and inter-rater reliability (Fichter et al., 1998; Fichter & Quadflieg, 2000, 2001). Because the SIAB-EX is one of the few ED assessment interviews that include dimensional measures of past as well as current symptom severity, it has been widely used in genetic studies to examine behavioral phenotypes (e.g., Halmi et al., 2005).

The Psychiatric Research Interview for Substance and Mental Disorders (PRISM; Hasin et al., 1996) is a semi-structured interview for adults that allows for the assessment of comorbid psychiatric disorders and SUD including distinctions among substance-related psychiatric symptoms and psychiatric disorders independent of SUD. This instrument provides diagnostic categorization as well as clinical information including age of onset and severity, which is helpful in determining whether different disorders may be etiologically or temporally linked. An updated version of this measure (PRISM-IV) displayed acceptable levels of reliability, including kappa coefficients of 0.76 for BN and 1.0 for AN (Hasin et al., 2006).

The Composite International Diagnostic Interview (CIDI; Wittchen, Kessler, & Ustun, 2001) was developed for nonclinician interviewers to ask highly structured questions of nonclinical study participants (ages 16 and older) about various types of psychiatric symptoms. The Substance Abuse Module (SAM) of the CIDI focuses on substance use and misuse (Robins, Babor, & Cottler, 1987; Robins et al., 1988). The CIDI also includes ED items pertaining to the diagnosis of AN and BN, although the CIDI is used less frequently in the ED literature. Its reliability and validity have been demonstrated (Cottler & Compton, 1993; Cottler, Robins, & Helzer, 1989), and it is in wide use among researchers with epidemiological and community samples.

14.1.1 Interview-Based Assessment Measures for Eating Disorders

The most widely used eating disorder-specific investigator-based interview is the Eating Disorder Examination (EDE; Fairburn, 2008), with the most recent version (16.0D) having been updated from a previously published version (12.0D, Fairburn & Cooper, 1993). The EDE includes 39 items and four subscales: Restraint, Eating Concern, Shape Concern, and Weight Concern. It also provides raw frequency data for eating disorder symptoms including purging and binge-eating behavior, which is subclassified into the frequency of episodes meeting DSM-5 criteria for binge eating

(objective bulimic episodes, OBEs) as well as other types of episodes (e.g., subjective bulimic episodes, objective overeating episodes). The EDE primarily focuses on the past 28 days prior to the interview; however, some of the questions focus on the last three months in order to establish a DSM-5 diagnosis. The interview begins with an orientation that includes the use of a calendar and an adaptation of the Timeline Followback (TLFB; Sobell & Sobell, 2003) method to provide memory anchors in order to improve the accuracy of symptom recall. A number of studies have examined the reliability and the validity of the EDE (see review by Berg, Peterson, Frazier, & Crow, 2012). Test-retest reliability scores for 2- to 14-day intervals have generally been good (range = 0.50 to 0.88) with the exception of subjective bulimic episodes (0.17–0.40) (Berg et al., 2012). Inter-rater reliability data for the EDE have generally been acceptable (range = 0.65 to 0.99) with slightly more variability in internal consistency coefficients (0.44 to 0.85) (Berg et al., 2012). The EDE has been widely used in treatment and descriptive studies of eating disorders and can be used with adolescents as young as 13, and a version for children is also available (Bryant-Waugh, Cooper, Taylor, & Lask, 1996). Although it is available in the public domain (https://webedit6.medsci.ox.ac.uk/psychiatry/research/researchunits/credo/assessment-measures), the EDE requires extensive training and expertise in both the interview in particular and EDs in general. In addition, because the EDE focuses on the past three months, this instrument is not suitable for the assessment of past ED symptoms, behaviors, or diagnoses.

The Yale-Brown-Cornell Eating Disorders Scale (YBC-EDS; Mazure, Halmi, Sunday, Romano, & Einhorn, 1994) is a semi-structured interview to assess preoccupations and rituals specific to EDs among adolescents and adults. In administering this instrument, the interviewer determines the types of preoccupations and rituals that the interviewee is experiencing currently as well as in the past. The interviewer then determines the frequency of occurrence, associated impairment, motivation for change, and overall functioning. Psychometric studies of the YBC-EDS have supported its reliability and validity (Mazure et al., 1994; Sunday & Halmi, 2000).

14.1.2 Interview-Based Assessment Measures for Substance Use Disorders

One of the most widely established methods for SUD assessment in adults and adolescents is the TLFB (Sobell & Sobell, 2003). In this approach, the interviewer collaborates with the patient, client, or research participant to establish specific memory anchors using a calendar. The patient or participant is then asked to recall specific events of alcohol and/or drug use as well as periods of abstinence for a designated time frame (e.g., one week, one month). Empirical evidence supports both the reliability and validity of TLFB, and this approach can be especially useful with variable consumption patterns and/or when detailed frequency estimates are needed (see Sobell & Sobell, 2003). The TLFB has also been validated for assessing

gambling behaviors (Hodgins & Makarchuk, 2003; Weinstock, Whelan, & Meyers, 2004). Form 90 (Project Match, 1993; Tonigan et al., 1997) integrates the TLFB with additional measurement of treatment and psychosocial functioning along with alcohol and drug consumption for the past 90 days. This approach also incorporates a grid averaging method (Miller & Marlatt, 1984) for time efficiency. Psychometric data support the reliability and validity of Form 90 if it is administered by interviewers who have been adequately trained (Sobell & Sobell, 2003).

The Addiction Severity Index (ASI; McLellan, Luborsky, Woody, & O'Brien, 1980) is an interview-based measure that assesses problem severity and treatment need in a variety of domains: alcohol use, drug use, employment, medical issues, psychiatric problems, family and social relationships, and legal problems. The ASI has considerable psychometric data supporting its reliability and validity (see review by Makela, 2004) and is widely used in both clinical and research settings. The ASI includes composite scores based on both self-report and interviewer-based ratings. More recently, a computer-administered version of the interview has been found to be promising in terms of eliciting increased self-disclosure (Butler, Villapiano, & Malinow, 2009) and providing a more time-efficient assessment method that does not require staff training or administration. A module assessing gambling is also available (Petry, 2007).

The Lifetime Drinking History (LDH; Skinner & Sheu, 1982) interview can be used to determine lifetime patterns of alcohol consumption, including a modified version that includes a maximum severity category (Sobell, Sobell, Leo, & Cancilla, 1988). Although psychometric data support the reliability and validity of the LDH, it lacks the precision of other methods, particularly in assessing current patterns, and is primarily useful for obtaining a chronological overview (Sobell & Sobell, 2003). An updated version, the Cognitive Lifetime Drinking History (Russell et al., 1997) can also be administered by computer.

The CRAFFT (Knight et al., 1999) is a screening instrument that can be used to assess substance use disorders among individuals under the age of 21. CRAFFT is an acronym for the screening questions (e.g., "Have you ever ridden in a CAR. . ."), which can also be administered in a questionnaire format. This instrument has psychometric data to support its use (Knight, Sherritt, Schrier, Harris, & Chang, 2002) and is widely used in research and clinical settings. The Adolescent Diagnostic Interview (Winters & Henly, 1993) can also be used to assess and diagnose drug and alcohol problems in adolescent samples.

14.1.3 Using Questionnaires Versus Interview-Based Instruments

Interview and questionnaire methods can be used to quantify symptomatology (e.g., a specific symptom such as binge-eating frequency), establish a diagnosis, identify individuals with symptoms or at risk of disorders within nonclinical samples, and measure change over time. For this reason, although semi-structured interviews are often considered the ideal method of assessment in psychiatric research, questionnaire-based self-report measures have several unique advantages over

interview-based methods and, at times, are more appropriate for certain types of assessment. Often, questionnaires are selected because they cost less and are easier to administer. In addition, they can be given to large samples (e.g., in epidemiology research) and are more easily adapted to technology-based administration. Interestingly, research investigations comparing self-report and interview-administered versions of the same instruments have yielded mixed conclusions, with some symptoms reported with greater severity on questionnaire-based measures and others on interview-based measures (Berg, Peterson, Frazier, & Crow, 2011, 2012; Fichter & Quadflieg, 2000). For this reason, questionnaires may overestimate the severity of symptoms (Mond, Hay, Rodgers, Owen, & Beumont, 2004). However, in a notable finding by Keel and colleagues (2002) in a study of eating disorder symptoms, the investigators found better agreement between questionnaires and interviews conducted by phone than between questionnaires and interviews conducted in person. Perhaps the one area within ED assessment in which questionnaire-based measures are particularly problematic is dietary restriction. Given that self-report measures of dieting and dietary restraint correlate quite poorly with actual dietary intake (Stice, Fisher, & Lowe, 2004), these data should be interpreted as intent to diet that is not necessarily manifested in actual food consumption. In summary, questionnaires are generally easier and less costly to administer, may enhance self-disclosure, and can reduce the risk of interviewer bias (Fichter & Quadflieg, 2000; Peterson & Miller, 2005). In contrast, interview-based methods may reduce the risk of false-positive screens or diagnoses as well as enhance reliability (Peterson & Miller, 2005; Peterson & Mitchell, 2005). Conducting assessment interviews by phone rather than in person may also enhance self-disclosure (Keel et al., 2002).

14.2 Questionnaires to Assess Both Eating Disorders and Substance Use Disorders

Despite the co-occurrence of ED, SUD, and ADC, few questionnaires are available that comprehensively assess these symptoms in a single instrument. The questionnaire version of the SIAB-EX (Fichter & Quadflieg, 2000) is an eating disorder questionnaire that includes several items related to SUD. The Eating Disorder Questionnaire (Mitchell, Hatsukami, Eckert, & Pyle, 1985; Mitchell, 2005) is a database measure for adolescents and adults that includes items about current and past eating disorder behavior, alcohol and drug use, weight history, treatment history, family history, and medical symptoms. ED and SUD/ADC co-occurrences have commonly been assessed using several items in larger epidemiological studies (e.g., Gadalla & Piran, 2007; Neumark-Sztainer, Story, Dixon, & Murray, 1998; Piran & Robinson, 2011). In the majority of research studies, particularly those using clinical samples, separate questionnaires for ED and SUD/ADC have been administered rather than a single comprehensive measure.

14.2.1 Questionnaires to Assess Eating Disorder Psychopathology

Widely used with adolescents and adults, the Eating Disorder Examination—Questionnaire (EDE-Q; Fairburn, 2008) contains similar items and subscales (i.e., Restraint, Eating Concern, Shape Concern, Weight Concern) as well as symptom frequencies as the interview version. Although the EDE-Q overlaps to a certain extent with the EDE, these measures should not be used interchangeably given differences in response patterns (Berg et al., 2013). Psychometric studies support the test-retest reliability from 1 to 14 days (range = 0.51–0.92, with the exception of subjective bulimic episodes: 0.39) and the internal consistency (range = 0.70–0.93) of the EDE-Q (Berg et al., 2012).

The Eating Disorder Inventory (EDI; Garner & Olmsted, 1984) was developed over three decades ago to assess eating disorder symptoms as well as associated psychological variables. The most recent edition, the 91-item EDI-3 (Garner, 2004), includes the original eating disorder subscales (Drive for Thinness, Bulimia, Body Dissatisfaction) and nine additional subscales (Low Self-Esteem, Personal Alienation, Interpersonal Insecurity, Interpersonal Alienation, Interoceptive Deficits, Emotional Dysregulation, Perfectionism, Asceticism, and Maturity Fears). The EDI-3 is widely used in clinical settings with adolescents and adults because it includes norms that can help the clinician illustrate a patient's or client's scores in a way that is useful for treatment planning. Psychometric data have supported its reliability and validity (Garner, 2004; Clausen, Rosenvinge, Friborg, & Rokkedal, 2011; Stanford & Lemberg, 2012) although results of studies examining its factor structure have been more inconsistent (Clausen et al., 2011). Developed recently, the Eating Pathology Symptoms Inventory (EPSI; Forbush et al., 2013) was developed iteratively using factor analysis and has eight factors: Body Dissatisfaction, Binge Eating, Cognitive Restraint, Excessive Exercise, Restricting, Purging, Muscle Building, and Negative Attitudes toward Obesity. In contrast to other questionnaires, the EPSI was developed as a comprehensive measure that is suitable for use with males as well as females. Psychometric data suggested high levels of internal consistency (0.84–0.89) as well as test-retest reliability (average = 0.73) (Forbush et al., 2013).

For diagnostic assessment, the Eating Disorder Diagnostic Scale (EDDS; Stice, Fisher, & Martinez, 2004; Stice, Telch, & Rizvi, 2000) is a brief 22-item questionnaire that can be used to establish eating disorder diagnoses in adolescents and adults. The EDDS has been found to have excellent reliability and validity as well as diagnostic specificity and sensitivity (Stice et al., 2000; Stice, Fisher, & Martinez, 2004). Although interview-based methods like the SCID-I are generally considered optimal diagnostic instruments, the EDDS can be used as a screening tool and in large samples when interviewing is not feasible. Given that the EDDS was developed for DSM-IV diagnosis, the extent to which the EDDS is a valid measure for DSM-5 criteria has not been established.

Several brief screening instruments are available for eating disorder symptoms in adolescents and adults, including the SCOFF (Morgan, Reid, & Lacey, 1999; Perry et al., 2002). The five items (e.g., "Do you make yourself sick? Would you say

that food dominates your life?") can be administered easily in all types of settings to identify individuals at risk for an eating disorder. Although originally designed as an interview, the psychometrics of the questionnaire version have been found to be comparable (Hill, Reid, Morgan, & Lacey, 2010; Perry et al., 2002). The BULIT-R (Thelen, Farmer, Wonderlich, & Smith, 1991) and the Bulimia Investigatory Test Edinburgh (BITE; Henderson & Freeman, 1987) can be used to screen for symptoms of bulimia nervosa. Used for the assessment of binge eating in both clinical and community samples including field trials for BED, the Questionnaire for Eating and Weight Patterns—Revised (QEWP-R; Spitzer et al., 1993) includes items about each of the diagnostic criteria for BED and can be administered to adults and adolescents. Although psychometric data supporting the validity of the QEWP-R are inconsistent, it has been found to be a useful preliminary screening instrument for detecting binge eating, including obese samples (Barnes, Masheb, White, & Grilo, 2011).

Another widely used questionnaire of ED attitudes and behaviors is the Eating Attitudes Test (Garner & Garfinkel, 1979), one of the original measures designed to assess ED symptoms as well as determine ED risk. The EAT includes items to assess symptoms of anorexia nervosa and has been translated into several languages in addition to English. Both 40- and 26-item versions of this test have demonstrated reliability and validity (Garner, Olmsted, Bohr, & Garfinkel, 1982), and it has been used in both clinical and community settings as an eating disorders screening instrument.

A brief, 30-item questionnaire developed to measure ED symptoms and pathology over time among individuals as young as 10 years old as well as adults is the Minnesota Eating Behavior Survey (MEBS; von Ranson, Klump, Iacono, & McGue, 2005). It assesses eating pathology on a continuum, both in a total score and as measured with four subscales (Body Dissatisfaction, Weight Preoccupation, Binge Eating, and Compensatory Behaviors). While it has been used with both male and female participants, like many ED measures, the MEBS' Weight Preoccupation items appear to more adequately assess variables that are traditionally feminine, such as concern with the size of thighs and hips, than masculine-oriented symptoms, such as concern with muscularity. Evaluations of the MEBS' psychometrics have been positive, except the Compensatory Behaviors subscale, which has shown limited reliability (von Ranson, Cassin, Bramfield, & Fung, 2007; von Ranson et al., 2005).

Another measure that can be used to assess eating disorder in youth is the Kids Eating Disorder Survey (KEDS; Childress, Brewerton, Hodges, & Jarrell, 1993), a questionnaire that has been used in epidemiological studies with children. Additional research has documented the psychometric features of the KEDS including its reliability and validity (Childress, Jarrell, & Brewerton, 1993).

In general, caution is merited when using self-report questionnaire to assess psychiatric diagnoses. Some symptoms of eating disorders involve fairly complex concepts, such as binge eating and the undue influence of weight and shape in self-evaluation, which can be difficult to assess accurately by questionnaire alone, especially among nonclinical samples. Providing the opportunity for respondents

to ask clarifying questions about items is one way to help safeguard the validity of responses. An alternative is two-stage screening of nonclinical samples, in which a questionnaire administered in one stage is followed by in-depth diagnostic interviews of high scorers in a second stage.

Finally, an aspect of body dissatisfaction that is more stereotypically masculine, and less often assessed, is the desire to be more muscular. However, it is worth noting that these concerns can also affect women. Among several questionnaires tapping concerns about muscularity (see Tod, Morrison, & Edwards, 2012) is the 15-item Drive for Muscularity Scale (McCreary & Sasse, 2000). Although this measure was developed for use with adolescents, subsequent work has demonstrated its reliability and validity when used with men and women as well (Wojtowicz & von Ranson, 2006).

14.2.2 Questionnaires to Assess Substance Use Disorder

The TLFB method, designed as an interview, can also be administered as a self-report questionnaire (Sobell & Sobell, 1996), using pen and pencil or computer administration. Similarly, the CRAFFT for youth can be administered in self-report format. In addition to the TLFB, quantity frequency measures (QF; see reviews by Room, 2000; Sobell & Sobell, 2003) are widely used written self-report measures that vary according to whether they assess average use (e.g., the average number of days in which a substance was used and the average amount on those days) or multidimensional variables including volume; however, QF approaches are generally less accurate than TLFB (Sobell & Sobell, 2003). A paper and pencil version of the ASI can also be used to assess SUD severity.

The Alcohol Use Disorders Identification Test (AUDIT; Babor, Higgens-Biddle, Saunder, & Monteiro, 2001) has become the preeminent alcohol screening tool, surpassing the Michigan Alcohol Screening Test (MAST; Selzer, 1971) and the CAGE (Ewing, 1984), which were widely used for a number of decades. The AUDIT is comprised of ten items assessing drinking quantity, negative consequences, and impaired control. This measure was developed for a World Health Organization study and, therefore, uses items that are cross-culturally neutral. The three consumption items have also been validated as a very brief screen for use in large-scale epidemiological studies (Bush, Kivlahan, McDonell, Fihn, & Bradley, 1998). Another widely used alcohol scale to assess dependence more specifically is the Alcohol Dependence Scale (Skinner & Allen, 1982). The AUDIT has also been modified to assess other drug use problems (Drug Use Identification Test; DUDIT; Voluse et al., 2012), although it is less well validated. The Drug Abuse Screening Test (DAST; Skinner, 1982) also provides a reliable drug use problem screen using 10 items and can be used with adolescent as well as adults. Consequences of alcohol and drug use can be assessed using the Drinker Inventory of Consequences (DrinC) (Miller, Tonigan, & Longabaugh, 1995) and the Inventory of Drug Use Consequences (IDUC; Blanchard, Morgenstern, Morgan, Lobouvie, & Bux, 2003). The DUDIT, DAST, and IDUC do not differentiate

among the specific drugs of abuse, whereas a newer scale, the Alcohol, Smoking, Substance Involvement Screening Test (ASSIST; WHO Assist Working Group, 2002) assesses seven different drug classes plus alcohol and tobacco. Several studies have supported the reliability and the validity of the ASSIST (Humeniuk et al., 2008; WHO Assist Working Group, 2002).

A number of questionnaires can be used to assess substance use and substance use disorders in youth. The Young Adult Alcohol Consequences Questionnaire (Read, Kahler, Strong, & Colder, 2006) is a measure of consequences of alcohol consumption that includes eight separate domains (Social-Interpersonal Consequences, Impaired Control, Self-Perception, Self-Care, Risk Behaviors, Academic/Occupational Consequences, Physical Dependence, Blackout Drinking) identified by confirmatory factor analysis. A brief version has also been developed with psychometric support of its reliability and validity for a six-week time period (Kahler, Hustad, Barnett, Strong, & Borsari, 2008). Additional screening questionnaires with psychometric support that can be used in adolescent samples include the Personal Experience Inventory (Winters & Henly, 1989) and the Adolescent Substance Abuse Subtle Screening Inventory (Lazowski & Miller, 2001; Miller, 1985).

A variety of screening tools have been developed for gambling disorders in both clinical and community samples (see review by Hodgins & Stinchfield, 2008). The South Oaks Gambling Screening (Lesieur & Blume, 1987) was almost universally used until the development of newer instruments such as the Problem Gambling Severity Index (PGSI; Ferris & Wynne, 2001) and the National Opinion Research Center DSM-IV Screen for Gambling Problems (NODS; Gerstein et al, 1999).

Questionnaires are available that focus specifically on the negative consequences associated with alcohol, other drug use, and gambling. These questionnaires basically provide comprehensive or brief checklists of potential consequences of SUD (Blanchard, Morgenstern, Morgan, Lobouvie, & Bux, 2003; Forcehimes, Tonigan, Miller, Kenna, & Baer, 2007; Tonigan & Miller, 2002). Questionnaires can also be used to assess a variety of other domains with clinical or theoretical importance including craving, relapse precipitants, family history, readiness to change, and self-efficacy (Hodgins et al., 2010: Hodgins & Stinchfield, 2008).

14.3 Technology-Based Assessment Administration

Assessment measures of ED, SUD, and ADC, in particular, and psychopathology/behavioral health, in general, are increasingly administered and scored using technology. Although computer scoring of certain measures (e.g., the EDI) has been available for several decades, there is an increasing focus on the use of technology-based momentary measurement in psychopathology research. Called ecological momentary assessment (EMA; Stone & Shiffman, 1994), this approach uses experience sampling by having individuals' record feelings, behaviors, attitudes, and thoughts in "real time" using handheld technology including cellular

phones. Typically, EMA devices signal users to make recordings several times each day. In addition, EMA users also typically provide recordings when a specific behavior occurs (e.g., ingestion of food, alcohol, or drugs; self-induced vomiting). EMA has been used to assess precipitants of eating disorder symptoms (Smyth et al., 2007) and drug and alcohol use as well as the process of relapse (see review by Shiffman, 2009). In addition, EMA is more frequently being used in ED and SUD/ADC research to measure treatment outcome (see Shiffman, 2009). Interestingly, EMA has been found to correlate with biochemical markers including blood alcohol level and may be more accurate than TLFB in self-reported substance use (Shiffman, 2009). Ideally, EMA can be adapted for use within clinical as well as research settings. Preliminary data suggest that the "reactive" effects of EMA in altering eating disorder or substance use patterns are minimal (Shiffman, 2009; Stein & Corte, 2003), supporting the reliability and feasibility of this innovative measurement approach.

Although EMA is particularly novel in its use of technology-based assessment to examine variables in "real time" (minimizing retrospective recall bias while maintaining ecological validity that can be compromised within a laboratory setting), technology-delivered assessment can be useful with more traditional, non-momentary approaches as well. Administering questionnaires using computers has a number of advantages over paper and pencil measures. First, clients, patients, and research participants often prefer computer-based administration. Second, this process minimizes error in data entry as well as increasing time efficiency (particularly data entry). Third, and potentially most critical, increasing evidence suggests that computer administration of questionnaires and interviews may increase self-disclosure and data reliability compared to face-to-face administration, particularly in SUD (Butler et al., 2009; Caldwell & Jan, 2012). For these reasons, computer-based assessments should be utilized in research and clinical settings whenever sensitivity (e.g., detecting ED symptoms or substance use) is especially important.

14.4 Laboratory and Observational Methods

Serum and urine toxicology as well as breathalyzer (for alcohol) tests can be used to assess recent substance use ingestion. Other biomedical markers of alcohol that include elevated levels of gamma-glutamyltransferase, carbohydrate-deficient transferrin, and alanine aminotransferase are described in Chap. 15. Although no laboratory test is available for the diagnosis of ED, medical assessment including a physical examination and electrolyte screening is often advised (Crow & Swigart, 2005) to assess medical risk. Behavioral paradigms including feeding laboratories (e.g., Samuels, Zimmerli, Devlin, Kissilef, & Walsh, 2009) as well as test meals in which individuals are observed eating (Anderson & Paulosky, 2004) can also provide useful data about eating patterns, food consumption avoidance, binge-eating behavior, as well as overall energy and macro-/micronutrient intake. Similarly, researchers have utilized drinking paradigms in laboratory settings

(e.g., Conrad, McNamara, & King, 2012) to assess consumption behavior and psychological factors related to use.

14.5 Enhancing Data Reliability and Data Quality

Several strategies can be used to enhance the reliability of assessment information among individuals with ED and/or SUD/ADC (see Anderson & Paulosky, 2004; Hodgins et al., 2010; Miller & Rollnick, 2012; Peterson, 2005; Sobell & Sobell, 2003; Vitousek et al., 1991) including (1) providing positive reinforcement for accurate self-report; (2) establishing rapport in both clinical and research settings; (3) using motivational interviewing techniques to manage resistance, increase engagement, and explore ambivalence (Miller & Rollnick, 2012); (4) asking for specific behavioral examples (e.g., "How many bottles of wine did you drink?," "How large was the ice cream container?"); (5) having staff members who are not involved in the individual's treatment team conduct assessment interviews and administer/collect questionnaires; (6) utilizing telephone interviews and computer-based questionnaires as a potential strategy to enhance self-disclosure; (7) using objective data (e.g., blood alcohol levels) whenever possible; (8) seeking collateral information from family members and other treatment providers; and (9) using timeline anchors to increase recall (e.g., TLFB, described above). In addition, Sobell and Sobell (2003) have suggested several strategies that are particularly helpful in the context of SUD assessment: (1) ensure that the individual is not under the influence of drugs or alcohol at the time of assessment, (2) provide written confirmation of data confidentiality, and (3) administer assessment methods in a conducive setting (e.g., not during incarceration). Although these strategies will not guarantee increased self-disclosure and data reliability, they can be used to optimize the likelihood that patients, clients, and research participants will provide accurate information to the best of their abilities.

Finally, certain assessment instruments may need to be adapted in the context of coexisting ED and SUD/ADC. For example, interviews and questionnaires to assess depression typically include items about disturbance in eating and weight. These items may need to be omitted and total and subscale scores prorated given the likelihood of endorsement by individuals with ED symptoms. Similarly, assessment instruments that specify a quantity of alcohol consumed as a threshold for possible intoxication (e.g., five or more drinks as specified by the SCID-I) may have to be modified given that individuals with ED who are underweight and/or nutritionally compromised may become intoxicated after consuming smaller amounts of alcohol than non-ED individuals.

14.6 Clinical Considerations

Given the high rates of suicide (Harris & Barraclough, 1998) and psychiatric comorbidity (Steiger & Israel, 2010) among individuals with ED, SUD, and ADC, patients, clients, and research participants should be screened carefully on an ongoing basis for suicidal ideation, plan, intent, and risk (Shea, 1999) as well as other psychiatric symptoms. In addition, all clinical and research staff should be trained in both suicide assessment and crisis management. As mentioned above, ongoing medical assessment is also a necessity among individuals with ED, SUD, and ADC to monitor medical risk and health status.

Although the majority of ED, SUD, and ADC measures are used to determine diagnosis, symptoms, severity, and psychosocial impairment, most of these instruments do not provide a longitudinal perspective of these co-occurring disorders. Given the variability of temporal and sequencing patterns for the onset and course of ED, SUD, and ADC among individuals in which these conditions co-occur (e.g., Baker et al., 2013) as well as the important implications for treatment, mapping out a historical timeline with onset, offset, relapse, and remission dates of ED and substance use patterns can be useful. As described above in the context of TLFB, the use of calendars with dates of historical importance can improve the quality of historical data recall.

Although self-monitoring measures are used less often in ED and SUD/ADC research because of the potential reactivity by which recording impacts behavioral patterns (Sobell & Sobell, 2003; Wilson, 1993), written self-monitoring (or self-monitoring with handheld technology like cellular phones) of eating patterns and substance use (as well as gambling and impulsive behaviors) can be extremely useful initially for treatment planning as well as throughout treatment to identify targets of treatment and to examine progress. The self-monitoring forms included in Fairburn (2008) can be modified for the assessment of substance use along with eating intake. Although not necessarily accurate (Anderson & Paulosky 2004) and, as a result, not ideal as a sole method of assessment, self-monitoring forms are nonetheless a useful measurement tool in the context of treatment (Fairburn, 2008). In addition, the "reactivity" of self-monitoring that can be problematic in research is potentially advantageous in clinical settings, given the likelihood that self-monitoring will actually improve the behavior being monitored (Wilson, 1993). Similarly, symptom recall (e.g., asking in an interview or questionnaire whether any eating disorder symptoms or drug or alcohol consumption occurred within the past 24 hours) may be not be entirely accurate but can be clinically useful. TLFB can also be used as a clinical tool for SUD (Sobell & Sobell, 2003) and can be expanded to include eating disorder symptoms as well (e.g., similar to EDE procedures).

In contrast to research settings, clinicians can rely on unstructured interviewing techniques (e.g., Berg & Peterson, 2013; Peterson, 2005) along with the semi-structured interviews and questionnaires described above to assess ED, SUD, and ADC. Clinicians can also interview family members and significant others for additional information about ED, SUD, and ADC symptoms and behaviors, a

procedure that can be impractical given confidentiality restrictions in the context of research. Collateral information from other sources can be extremely useful in understanding and diagnosing ED, SUC, and ADC with accuracy, although the extent to which consulting with individuals other than the client, patient, or research participant can compromise trust in the clinician should be carefully considered and, ideally, decided collaboratively. At minimum, obtaining previous treatment records is extremely helpful in assessing ED, SUD, and ADC.

Directions for Future Research and Conclusions

As reviewed in this chapter, interviews, questionnaires, technology-based methods, laboratory measures, and test meals can be used to assess ED, SUD, and ADC. Accurate assessment is complicated by several factors including the complexity of symptom presentation along with the potential for symptom denial and minimization (which may or may not be intentional). One main limitation in the assessment of these co-occurring conditions is the fact that few available measures address ED, SUD, and ADC comprehensively. Instead, the combined use of separate measures of ED and SUD/ADC is recommended, along with the SCID-I for diagnostic purposes given that it includes both ED and SUD/ADC symptom categories (a combination that is currently a common practice in research) (Grilo, White, Barnes, & Masheb, 2013; Root et al., 2010). Perhaps the most pressing need in research is the development of instruments specifically designed to assess co-occurring ED, SUD, and ADC comprehensively. Ideally, interviews and questionnaires could be used as dimensional measures (e.g., severity measures of distress, behavioral frequency of ED and substance use) as well as diagnostic tools to facilitate DSM-5 diagnosis. Instruments designed to assess both current and past ED, SUD, and ADC symptoms would also be a significant contribution to this literature.

Additional research is needed in technology-based assessment, particularly using handheld devices including cellular phones. Used in research in the context of EMA, the need for momentary data that can be utilized in clinical settings is significant, particularly given the potential for increased reliability and use in treatment. In particular, the integration of technology-based assessment with interventions (e.g., interactive programs that could provide feedback and treatment techniques based on momentary data related to eating and substance use patterns) holds promise for clinical as well as research settings. Finally, although a number of questionnaires and interviews are available to assess ED, SUD, and ADC, more data are needed to establish their reliability and validity, particularly among samples characterized by co-occurring ED and SUD/ADC disorders, as well as in samples with heterogeneity in age, ethnic status, and gender.

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Medical Complications of Eating Disorders, Substance Use Disorders, and Addictions

15

Pauline S. Powers and Nancy L. Cloak

Abstract

Eating and substance use disorders are common psychiatric disorders that often occur together and are associated with high premature mortality rates, most often due to medical complications. The medical complications are presented for each set of disorders and the interactions among them are described. Other co-occurring psychiatric disorders (i.e., bipolar disorder) and medical illnesses (i.e., diabetes mellitus) and their effect on management are discussed. Correlates of the medical complications, including age, severity, the duration of each disorder, and additional medical and psychiatric comorbidities, are reviewed. Identification of medical complications utilizing the medical history, review of systems, physical examination, and laboratory testing is described, and the impact of medical complications on choice of treatment setting is outlined. Management of commonly encountered medical issues (including the refeeding syndrome and withdrawal from various substances) is discussed, and therapeutic use of medical information to decrease denial and increase early entry into appropriate treatment is described. Finally, the urgent need to educate healthcare professionals in management of patients with these disorders is emphasized.

Keywords

Eating disorders • Substance use disorders • Medical complications

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15.1 Introduction

Eating disorders (EDs) and substance use disorders (SUDs) are common psychiatric conditions that frequently occur together. Both have high Standardized Mortality Ratios (SMRs) due to medical complications and suicide. These conditions can be difficult to treat and when they occur together are likely to be even more difficult. Understanding the independent and combined medical complications of these disorders is crucial in treatment planning and implementation, as evidenced by their prominence in guidelines from the American Psychiatric Association (Yager et al., 2006), the American Society of Addiction Medicine (Mee-Lee & Schulman, 2001), and the National Institute of Clinical Excellence, (2004). Although other psychiatric disorders are also associated with high SMRs, their direct medical complications, at least at this time, are less clear than among ED or SUD.

There is currently preliminary, but very exciting, research suggesting commonalities in the neurobiological pathways of ED and SUD (Kaye, 2010; Volkow & Baler, 2014; Walsh, 2013). Also, there is the clinical commonality of continued use of substances or restrictive and compensatory behaviors despite medical, psychological, and social consequences. Recent work suggests that there may be psychobiological explanations for the denial (or unrealistic optimism) about possible negative consequences so often seen in both sets of disorders (Dillard, Midboe, & Klein, 2009; Klein, Ullsperger, & Danielmeier, 2013; Rinn, Desai, Rosenblatt, & Gastfriend, 2002; Vandereycken, 2006).

15.1.1 Prevalence

The estimated lifetime prevalence of ED is 11.7 % (Hudson, Hiripi, Pope, & Kessler, 2007), and the estimated lifetime prevalence of SUD is 14.5 % (Kessler et al., 2005). This means that at some time during their lives, approximately 37 million Americans will have an ED and approximately 46 million Americans will have an SUD.

Although the exact prevalence rate of comorbid ED and SUD is unknown, the evidence is clear that they often co-occur and that it is more likely in patients who binge or purge, irrespective of their specific ED diagnosis (Harrop & Marlatt, 2010; Nokleby, 2012). Table 15.1 illustrates lifetime comorbidity estimates from the National Comorbidity Survey Replication (Hudson et al., 2007) that included 2,980 subjects. Among patients with anorexia nervosa (AN), bulimia nervosa (BN), and binge eating disorder (BED), the lifetime prevalence of any substance use disorder was 27 %, 36.8 %, and 23.3 %, respectively (see Table 15.1). Piran and Robinson (2011) looked at the relationship between ED and SUD in over 1,000 young women and found that the cluster of binge eating, dieting, and purging was significantly correlated with binge drinking, drinking associated with negative consequences, and with cocaine use. The cluster of dieting and purging was associated with the use of stimulants/amphetamines.

Table 15.1 Lifetime comorbidity estimates of eating disorders and substance use

Eating disorder	Alcohol use disorder (%)	Drug use disorder (%)	Any substance use disorder (%)
Anorexia nervosa	24.5	17.7	27.0
Bulimia nervosa	33.7	26.0	36.8
Binge eating disorder	21.4	19.4	23.3

Adapted from Hudson et al. (2007)

Few studies estimating comorbidity have included boys and men with ED, and most have not included as SUD the use of nicotine, caffeine, or energy drinks. Most studies also have not included the misuse of medications that have established therapeutic use for other illnesses. Nor has the inappropriate omission of needed medications to promote weight loss usually been considered in estimates of SUD. An additional problem is that most studies begin with patients diagnosed with ED to determine how many also have SUD; few studies begin with patients who have SUD and assess for possible comorbidity of ED (Nokleby, 2012).

15.1.2 Premature Mortality

The Standardized Mortality Ratio (SMR) has been used to estimate premature mortality. The SMR is the ratio of the number of deaths among individuals with an illness in a study population compared to the number of deaths expected based on age- and sex-specific rates in a standard population. If the ratio is greater than one, individuals with the illness have a greater chance of premature death than those without the illness. The SMR for AN is the highest among ED (5.86), and among SUD the SMR for drug injection is dramatically high at 14.68 (see Fig. 15.1 for details). For patients with both ED and SUD, few data are available. One small study by Keel et al. (2003) found that the severity of alcohol use disorder (AUD) was consistently a predictor of fatal outcome among AN patients.

15.1.3 Substances Misused

Legal substances including alcohol, nicotine, and caffeine are the most commonly used and misused substances by patients with ED (Anzengruber et al., 2006; Greenfield, Back, Lawson, & Brady, 2010; Holderness, Brooks-Gunn, & Warren, 1994; Pisetsky, Chao, Dierker, May, & Striegel-Moore, 2008). (See Chap. 12.) Medications with legitimate treatment indications may also be misused by patients with ED, often as a coping strategy. Among these medications and their approved uses are the following: stimulants (for attention deficit disorder), opiates (for severe pain relief), sleep medications (for insomnia), and marijuana (approved in some states for a variety of uses including terminal cancer). Certain other drugs with

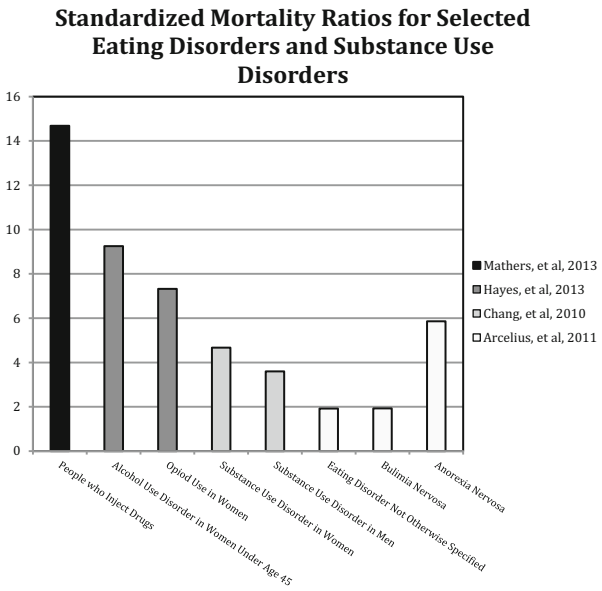

Fig. 15.1 Anorexia nervosa has the highest Standardized Mortality Ratio (SMR) of any eating disorder. People who inject drugs are at a very high risk for premature death

approved uses are misused as part of the ED including laxatives, diuretics, and weight loss medications. Eating disorder patients may use laxatives to compensate for food consumption; diuretics may be misused to promote rapid weight loss or to decrease perceived or real edema. Weight-loss drugs, including fat absorption inhibitors or appetite suppressants, may also be misused. Illegal substances, especially cocaine (Jonas, Gold, Sweeney, & Pottash, 1987) and, less often, methamphetamine and 3,4-methylenedioxymethamphetamine (ecstasy) are also used.

Some medications (i.e., insulin or pancreatic enzymes) may be withheld as part of the ED to compensate for food consumption. Withholding insulin can result in hyperglycemia and weight loss due to excretion of the excess blood glucose into the urine. Withholding pancreatic enzymes can result in malabsorption and subsequent weight loss.

15.2 Correlates of Medical Complications

Multiple factors contribute to the likelihood of medical complications in patients with comorbid ED and SUD including age of onset, severity and duration of either disorder, additional comorbidities, efficacy of previous treatments, available support systems, and innate vulnerability of the patient.

Although there is disagreement on whether ED and SUD simply co-occur or are actually comorbid, a common psychopathological course is often seen. For example, many patients with AN develop BN, typically because the patient's hunger eventually leads to increased caloric intake that in turn elicits extreme anxiety that is relieved with various purging strategies. In this weight-restored, but still ill,

condition, the patient may utilize various drugs (typically amphetamines or cocaine) to decrease appetite. In addition, the patient may utilize alcohol or other drugs to decrease anxiety or to decrease awareness of obsessions about weight, shape, or size. One consequence of this common pathway is often an interactive set of medical complications, including previous enduring effects of semi-starvation (i.e., osteoporosis or decreased grey matter of the brain), effects of purging behavior (i.e., electrolyte disturbances, cardiac arrhythmias, or gastrointestinal complications), and effects of alcohol misuse (i.e., hypertension or liver toxicity).

15.2.1 Comorbid Psychiatric Disorders

People with both ED and SUD frequently have additional comorbidities, including both psychiatric and medical disorders. Anxiety disorders are common among ED patients, particularly obsessive–compulsive disorder (OCD) that co-occurs between 35 and 44 % of patients with AN or BN (Kaye, Bulik, Thornton, Barbarich, & Masters, 2004). A relationship between BN and bipolar disorder, particularly bipolar disorder II, has also been noted (Lunde, Fasmer, Akiskal, Akiskal, & Oedegaard, 2009). OCD and bipolar disorder also may have medical complications. Patients with OCD may have skin picking, which can result in scarring and infection (Odlaug & Grant, 2010) and hair loss from hair pulling. The medical complications associated with bipolar disorder include results of accidents associated with impulsive behavior, migraine headaches, and a variety of complications associated with the treatment of bipolar disorder (Krishnan, 2005). Perugi and Akiskal (2002) have noted a connection between atypical depression, bipolar spectrum disorder, and ED (particularly BED and BN) and argue that there is a cyclothymic-anxious-sensitive temperament that is linked to all these conditions. One study by Perugi et al. (1997) found that among a consecutive series of 315 OCD patients, 15.7 % had bipolar disorder (mostly bipolar disorder II). Among all these conditions that frequently occur together (SUD, ED, OCD, atypical depression, and bipolar disorder), the sequence in which they develop is probably highly variable and currently is poorly understood. The spectrum concept is in line with current hypotheses that various neurocircuits overlap and interact in ways that may explain the comorbidity so often seen in ED patients. (See Chaps. 11 and 12.)

15.2.2 Comorbid Diabetes Mellitus

There is evidence that Type 1 diabetes mellitus increases the risk of ED (Fairburn, Peveler, Davies, Mann, & Mayou, 1991); estimates are that between 11.5 and 27.5 % meet diagnostic criteria for an ED, most often BN or BED (Grylli, Hafferi-Gattermayer, Schober, & Karwautz, 2004; Smith, Latchford, Hall, & Dickson, 2008). Among patients with Type 2 diabetes mellitus, BED is estimated to occur in 6.5–9.0 % (Davison, 2003).

One problem with determining an appropriate ED diagnosis is that neither the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV-TR) (American Psychiatric Association, 2000) nor the DSM-5 (American Psychiatric Association, 2013) specifically includes insulin omission as a compensatory behavior, and thus most diabetic patients with an ED have been diagnosed with eating disorder not otherwise specified (EDNOS). Nonetheless, insulin omission and the resultant uncontrolled blood sugar levels are associated with an increased likelihood of renal, ophthalmic, and gastrointestinal complications (Takii et al., 2008). Gagnon, Aime, Belanger, and Markowitz (2012) have summarized the findings that suggest the possibility of insulin omission as a compensatory behavior. These include marked fluctuations in weight (especially weight loss), restrictive dietary behaviors, multiple episodes of diabetes-related hospitalizations or ketoacidosis, or unexplained elevations in hemoglobin A1c.

Some medical complications, such as delayed gastric emptying and peripheral neuropathies, are common to both ED and diabetes. The precipitant for the dangerous behavior of insulin omission is typically fear of weight gain, either due to insulin itself or following food consumption, particularly carbohydrates.

15.2.3 Influence of Age

Another important correlate of medical complications among patients with ED or SUD is age. Unlike several other very serious psychiatric disorders (including schizophrenia and bipolar disorder), adolescent onset of AN is associated with a better prognosis and thus a decreased likelihood of serious medical complications, if successfully treated soon after onset (Walsh, 2013). BN and BED typically begin later than AN and, like AUD, may wax and wane over many years. Therefore, associated medical complications are highly variable.

Older adults with either disorder usually have a history of an ED or SUD in earlier life, but there may have been long symptom-free periods. Among adults 60 years and older, SUDs (particularly alcohol and prescription drugs) are known to be a common unrecognized problem (Center for Substance Abuse Treatment, 1998; Dar, 2006). With age, medical complications from SUD are more common. For example, excessive drinking increases the risk for coronary heart disease, hypertension, and stroke—all conditions whose incidence increases with age. Alcohol use is one of the three leading causes of falls in the elderly (Watson et al., 2013). Information about the relationship between age and medical complications among ED patients is sketchy. In 2008–2009, while the highest rate of hospitalizations for ED was 28 % among patients aged 19–30, 18 % of patients hospitalized for ED were between ages 45 and 65 and 7 % were aged 65 and older (Zhao & Encinosa, 2011). Although these data are from an impressive national database, it is likely that these rates are underestimates, since many patients are hospitalized for medical complications that are not necessarily diagnosed as secondary to an ED. This may be particularly true for older ED patients, since weight loss from other causes

commonly occurs in elderly patients, and binge and/or purge behavior may not be acknowledged by the patient.

15.3 Identifying Medical Complications

While ED and SUD are both associated with significant and sometimes fatal medical complications, existing guidelines do not address the medical evaluation and management of patients who have both conditions. This section synthesizes information from practice guidelines for the treatment of patients with ED (Banker et al., 2012; National Institute of Clinical Excellence (NICE), 2004; Yager et al., 2006; Yager et al., 2012), SUD (Center for Substance Abuse Treatment, 2006; Connery & Kleber, 2007; Kleber et al., 2006; Mee-Lee & Schulman, 2001), and co-occurring disorders (Center for Substance Abuse Treatment, 2005b). In addition, the medical assessment of patients who have co-occurring SUD and ED is addressed, the role of medical problems in determining level of care (placement) decisions is reviewed, and the detection and initial management of serious complications are outlined. For clinicians who do not have access to a medical provider with expertise in ED and SUD, strategies for managing some common complaints that can affect routine management are also discussed.

15.3.1 Medical History and Review of Systems

Because medical complications of ED and SUD can affect every organ system, a thorough medical history, review of systems, and physical examination are necessary. The medical history for patients with co-occurring ED and SUD focuses first on identifying risk factors for urgent, life-threatening complications such as the refeeding syndrome and alcohol and/or sedative-hypnotic withdrawal. Some complications might require referral for medical care while their ED and SUD are being treated. Examples might include HIV or hepatitis B or C infection related to intravenous drug use. Issues that may complicate treatment or compromise the patient's willingness to remain in treatment also need to be identified. Examples might include chronic pain in individuals with opioid use disorders or the abuse of laxatives and cessation of which could result in significant discomfort from constipation. Both of these could impair a patient's ability to participate in treatment. While the focus is on identifying conditions that require immediate attention, clinicians should also remember that patients with ED and SUD frequently do not follow up on recommendations for routine preventive health care, and these issues should be addressed at some point in treatment.

Clinicians should be aware that patients frequently minimize their symptoms in general, or focus on one area as "the problem," minimizing other contributions to their difficulties. For example, a patient may present for treatment of a cocaine use disorder and neglect to mention a substantial intake of alcohol or the use of self-induced vomiting to control his or her weight when he or she is unable to obtain

Table 15.2 Major medical complications of eating disorders and selected substance use disorders

	Disorder or substance							
System	Anorexia nervosa[a]	Bulimia nervosa	Binge eating disorder[b]	Alcohol	Stimulants[c]	Opioids	Marijuana[d]	Tobacco
Eye/ear/nose/throat	Optic neuropathy Lagophthalmos	Enamel erosion Dental caries Parotid enlargement Increased (salivary) amylase		Parotid enlargement Increased (salivary) amylase Oral cancer	Optic neuropathy Keratitis Enamel erosion Dental caries/tooth loss Gingival ulceration Chronic rhinitis Perforated nasal septum	Pupillary miosis	Conjunctival injection	Cataract Macular degeneration Periodontal disease Oral cancer
Respiratory	Aspiration pneumonia Spontaneous pneumothorax Spontaneous pneumomediastinum		Sleep apnea Pulmonary hypertension	Aspiration pneumonia Sleep apnea	Pneumothorax Pulmonary edema Pulmonary hemorrhage Bronchiolitis obliterans Hypersensitivity pneumonitis Pulmonary hypertension Interstitial fibrosis	Respiratory depression Bronchospasm Pulmonary edema Aspiration pneumonia	Bronchitis Lung cancer (likely)	Bronchitis Chronic obstructive pulmonary disease Laryngeal cancer Lung cancer Pulmonary hypertension
Cardiovascular	Bradycardia Hypotension Syncope Mitral valve prolapse QTc prolongation Sudden cardiac death Peripheral edema Pericardial effusion Emetine cardiomyopathy Acrocyanosis	Emetine cardiomyopathy Hypotension Syncope	Myocardial infarction Hypertension Congestive heart failure Atrial fibrillation	Alcoholic cardiomyopathy Hypertension Tachycardia Arrhythmias Sudden cardiac death	Myocardial infarction Cardiomyopathy Myocarditis Hypertension Aortic dissection Arrhythmia Sudden cardiac death	Hypotension	Tachycardia Increased risk for myocardial infarction Hypertension (HMAs)	Ischemic heart disease Aortic aneurysm Peripheral vascular disease

Gastrointestinal	Delayed gastric emptying Reflux, esophagitis Constipation Elevated transaminases Superior mesenteric artery syndrome	Reflux, esophagitis Mallory–Weiss tears Esophageal rupture Constipation Cathartic colon Pancreatitis Acute gastric dilatation	Reflux Esophagitis Gall bladder disease Fatty liver disease Pancreatitis Cancer of the esophagus, colon, liver, gallbladder, pancreas	Reflux Esophagitis Gastritis Pancreatitis Increased pancreatic amylase Malabsorption Elevated transaminases Alcoholic fatty liver Alcoholic hepatitis Cirrhosis GI cancers (esophagus, stomach, pancreas, liver, colon) GI bleeding (varices, Mallory–Weiss tears, gastritis)	Intestinal ischemia Gastroduodenal perforation Acute liver injury (MDMA)	Vomiting Delayed gastric emptying Constipation	Vomiting (HMAs) Xerostomia Constipation	Esophageal cancer Stomach cancer Pancreatic cancer
Fluids/renal/electrolytes	Dehydration Refeeding syndrome (hypophosphatemia, hypomagnesemia, hypokalemia) Nephrogenic diabetes insipidus Hyponatremia	Dehydration Hypokalemia Hyponatremia Hypomagnesemia Metabolic alkalosis Pseudo-Bartter's syndrome	Renal cancer	Dehydration Hypokalemia Hyponatremia Hypocalcemia Hypomagnesemia Hepatorenal syndrome Rhabdomyolysis	Dehydration Rhabdomyolysis Hyponatremia (MDMA, bath salts) Nephrosclerosis	Nephropathy Rhabdomyolysis	Acute kidney injury (HMAs) Hypokalemia (HMAs)	Bladder cancer Renal cancer
Reproductive/obstetric	Amenorrhea Oligomenorrhea Infertility Unplanned pregnancy Low birth weight Low testosterone (males)	Amenorrhea Oligomenorrhea Polycystic ovarian syndrome Miscarriage	Cancer of the breast, ovaries, endometrium, prostate Gestational diabetes Fetal macrosomia	Amenorrhea Oligomenorrhea Sexual dysfunction Infertility Fetal alcohol syndrome Low testosterone (males)	Placental abruption Low birth weight Prematurity Microcephaly	Amenorrhea Oligomenorrhea Unplanned pregnancy Low testosterone (males) Preeclampsia Low birth	Low birth weight Child development problems (cognitive, behavioral)	Prematurity Low birth weight Sudden infant death Cervical cancer Placental abruption Spontaneous abortion

(continued)

Table 15.2 (continued)

	Disorder or substance							
System	Anorexia nervosa[a]	Bulimia nervosa	Binge eating disorder[b]	Alcohol	Stimulants[c]	Opioids	Marijuana[d]	Tobacco
				Low birth weight Prematurity Breast cancer		weight Prematurity Miscarriage Neonatal withdrawal		
Endocrine/ metabolic	Osteoporosis Low T3, T4 Hypercortisolemia Hyperlipidemia Hypoglycemia Impaired temperature regulation Growth retardation	Hyperlipidemia Poor diabetes control	Type 2 diabetes Hyperlipidemia	Hypoglycemia Hyperglycemia Hyperlipidemia Hyperuricemia Osteoporosis	Hyperprolactinemia	Osteoporosis (males)		Osteoporosis
Hematologic	Anemia Leukopenia Thrombocytopenia		Non-Hodgkin lymphoma Multiple myeloma	Anemia Leukopenia Thrombocytopenia Coagulopathy				Acute myeloid leukemia Hypercoagulability
Dermatologic	Xerosis Carotenodermia Lanugo hair	Russell's sign			Skin lesions (excoriations)			Accelerated aging
Neuromuscular	Skeletal myopathy Peripheral neuropathy Wernicke's syndrome Decreased brain volume	Seizures Migraine	Osteoarthritis	Skeletal myopathy Peripheral neuropathy Wernicke's syndrome Withdrawal seizures Cerebral hemorrhage Decreased brain volume Cerebellar dysfunction Central pontine myelinolysis	Seizures Hyperthermia Cerebral infarction Cerebral hemorrhage Decreased brain volume		Seizures (HMAs)	Cerebrovascular disease

Psychiatric	Depression Anxiety Mood lability Insomnia Cognitive impairment	Depression Anxiety Mood lability Insomnia	Association with depression	Depression Anxiety Mood lability Insomnia Cognitive impairment Psychosis Withdrawal delirium	Anxiety Insomnia Paranoia Mood lability Psychosis Delirium Cognitive impairment	Depression Cognitive impairment	Anxiety Paranoia Cognitive impairment Psychosis Withdrawal syndrome	Risk for alcohol use disorders in adolescents Withdrawal syndrome
Other				Withdrawal syndrome Accidents, injuries Death from overdose	Accidents, injuries Multiple complications from IV use	Withdrawal syndrome Accidents, injuries Death from overdose Multiple complications from IV use	Accidents, injuries	Withdrawal syndrome

Source: Boghdadi and Henning (1997); Brewerton and George (1993); Brewerton, George, and Harden (1993); Bulik and Reichborn-Kjennerud (2003); Callaghan, Allebeck, and Sidorchuk (2013); Chase, Neild, Sadler, and Batey (2005); Darke, Kaye, McKetin, and Duflou (2008); Gowing, Henry-Edwards, Irvine, and Ali (2002); Grucza, Przybeck, and Cloninger (2007); Hall and Degenhart (2009); Hatsukami and Severson (1999); Hermanns-Clausen, Kneisel, Szabo, and Auwärter (2013); Hoffman, Zerwas, and Bulik (2011); Hochlehnert et al. (2010); Katzman (2005); Large, Sharma, Compton, Slade, and Nielssen (2011); Mehler (2011); Micali, Simonoff, and Treasure (2007); Mitchell and Crow (2006); Prosser and Nelson (2012); Warner (1993); Wipfli and Samet (2009)

[a]Complications listed are those of restricting anorexia nervosa; patients with anorexia nervosa who purge may also develop complications listed under "bulimia nervosa"

[b]Complications shown are those of obesity, which is present in 70 % of individuals with binge eating disorder; as yet, there is little evidence to support the presence of medical complications of binge eating disorder in patients who are not obese

[c]This category includes cocaine; amphetamine and methamphetamine; ecstasy (3,4-methylenedioxymethamphetamine, MDMA), which has both stimulant and hallucinogenic properties; and synthetic cathinones ("bath salts")

[d]This category includes marijuana and synthetic cannabinoids—herbal marijuana alternatives (HMAs)—such as spice and K2

Table 15.3 Key elements of the medical history for patients with eating disorders and substance use disorders

History
Eating disorder
Food intake (24-h recall; intake over past 1–2 weeks)
Weight history (highest, lowest, recent changes—degree and rate of recent loss is especially important)
Purging behaviors
Type (vomiting, laxatives, diuretics, exercise, fasting, insulin omission)
Any use of ipecac (quantify)
Amount (frequency and duration)
History of medical hospitalizations or ER visits related to eating disorder
Substance use disorder
All substances used, including alcohol, caffeine/energy drinks, tobacco
Quantify duration, amount, and time of last use, especially for alcohol, sedative-hypnotics, and opioids
History of complicated withdrawal (for alcohol and sedative-hypnotics) including seizures or delirium
Behaviors creating infectious disease risk (IV drug use, unprotected sex, exchanging sex for drugs, homelessness, or incarceration)
History of medical hospitalizations or ER visits related to substance use disorder
Both disorders
Misuse of substances related to eating disorder (e.g., using stimulants to suppress appetite, getting high to avoid eating)
Use of substances to control appetite or weight (tobacco, stimulants, diet pills, laxatives, diuretics, caffeine)

cocaine. To assist patients with being candid, it may be helpful to explain why you are asking for detailed information about issues that they may not feel are important or are uncomfortable discussing: namely to assess for potentially life-threatening medical complications and to guide the development of a comprehensive treatment plan. Many patients are not aware that they are at risk for serious medical complications, and education about this aspect of their disorders can have therapeutic value. Therefore, in addition to routine questions about past medical and surgical history, medications, and general systems review, specific questions about behaviors and symptoms commonly associated with the ED and SUD are recommended, as listed in Table 15.3.

Of particular importance are questions that assess risk factors for life-threatening complications. These include the refeeding syndrome and complicated alcohol or sedative-hypnotic withdrawal. The refeeding syndrome occurs in almost 8 % of severely underweight AN patients (Vignaud et al., 2010). Historical factors predisposing patients to refeeding syndrome include fasting or very low caloric intake, weight loss exceeding 15 % in the preceding 3–6 months (even if normal or above-normal weight at presentation), history of alcohol and/or drug abuse, and behaviors that may cause electrolyte abnormalities, such as self-induced vomiting

and laxative or diuretic use (Rio, Whelan, Goff, Reidlinger, & Smeeton, 2013). Alcohol withdrawal is complicated by seizures in 6–15 % of cases and by delirium in 4–15 % of cases (Eyer et al., 2011). Historical factors predisposing patients to complicated alcohol and/or sedative-hypnotic withdrawal include duration and amount of drinking/drug use, history of complicated or multiple withdrawals, and concurrent medical illnesses.

Obtaining an accurate history may be complicated by patients' tendency to minimize their use of substances; they may also focus on their "drug of choice" and not initially disclose their use of other substances, unless specifically asked. It can often be helpful to describe the dangers and discomforts of withdrawal and sharing your concern for protecting them from these outcomes. A review of systems may have therapeutic as well as diagnostic value, because patients may not link physical symptoms that trouble them to their ED or SUD, and making this connection can be motivating.

15.3.2 Physical Examination

Accurate measurements of height, weight, and vital signs, including orthostatic vital signs, are key components of the physical examination. Patients' weights should be measured with sensitivity to their frequent extreme discomfort with being weighed, and some patients may prefer to be weighed backwards. Weights should be obtained in a hospital gown, and heights should be measured carefully. For adolescents, weight and height should be compared with the patient's pediatric growth chart. The physical examination should likewise include an evaluation of key findings indicative of the medical complications of ED and SUD, as outlined in Table 15.4. Findings such as fever (temperature $>$ 100.4 °F), markedly abnormal vital signs, decreased or fluctuating level of consciousness, clonus, and hallucinations should prompt urgent referral to an emergency room.

15.3.3 Laboratory Studies

15.3.3.1 Laboratory Studies for ED Patients

Laboratory and imaging studies complement the history and physical examination and serve to detect potentially dangerous complications that have few or vague symptoms (such as hypokalemia), further evaluate symptoms and signs detected in the initial evaluation, identify asymptomatic medical complications (such as HIV or osteoporosis), and evaluate alternative causes for symptoms (such as hyperthyroidism as a cause for weight loss and anxiety). Additionally, laboratory testing can be used to detect unreported substance use, including some laxatives and diuretics that may be abused by patients with ED. Finally, some laboratory tests and procedures are recommended as part of routine preventive health care and should be included in the evaluation if the patient has not had them. Screening tests pertinent to treatment of individuals with ED and SUD include those for cervical cancer,

Table 15.4 Key aspects of the systems review and physical examination for patients with eating disorders and substance use disorders

	Potential findings and implications	
System	History	Physical examination
General	Fevers, chills, night sweats (infectious complications, alcohol/opioid withdrawal)	Fever (infectious complications, alcohol/opioid withdrawal, stimulant intoxication) Hypothermia (malnutrition)
HEENT	History of head trauma (e.g., when intoxicated), visual problems or double vision (anorexia, vitamin deficiencies), nasal pain or bleeding (from snorting drugs), lacrimation (opioid withdrawal), mouth sores, dental caries, sore throat, and/or parotid enlargement (self-induced vomiting)	Signs of head trauma Miosis (opioid intoxication) or mydriasis (opioid or sedative withdrawal) Nystagmus (Wernicke's) Lacrimation Nasal septal perforation Perimyolysis (self-induced vomiting) Parotid enlargement Oral lesions (cancer)
Respiratory	Cough (infectious complications, reflux), shortness of breath (pulmonary edema, chronic obstructive pulmonary disease, pulmonary hypertension), hemoptysis (lung cancer)	Hyperinflation, prolonged expiratory phase (COPD) Crackles (aspiration pneumonia, pulmonary edema)
Cardiovascular	Chest pain (myocardial ischemia, reflux, pericardial effusion), palpitations (arrhythmias), syncope or near syncope (arrhythmias, dehydration)	Tachycardia (stimulant intoxication, opioid or sedative withdrawal, refeeding syndrome, dehydration) Orthostatic hypotension (dehydration) Bradycardia (malnutrition) Irregular rhythm Mid-systolic click (mitral valve prolapse) Acrocyanosis (malnutrition) Diminished pulses (peripheral vascular disease)
Gastrointestinal	Heartburn (reflux), early satiety (delayed gastric emptying in eating disorders, stomach cancer), abdominal pain (pancreatitis, hepatitis, gall bladder disease, superior mesenteric artery syndrome), diarrhea (opioid withdrawal, laxative abuse), constipation (malnutrition, laxative abuse), vomiting (opioid or alcohol/sedative withdrawal, cannabinoid hyperemesis), hematemesis (Mallory–Weiss tear or esophageal rupture/variceal bleeding)	Abdominal tenderness Abdominal masses (cancer, aneurysm) Fecal occult blood
Neuromuscular	Generalized weakness (myopathy, electrolyte abnormalities), peripheral sensory loss (neuropathy), gait problems or falls (neuropathy,	Proximal weakness (myopathy) Sensory deficit (neuropathy) Ataxia, Romberg sign (cerebellar dysfunction, Wernicke's)

(continued)

Table 15.4 (continued)

System	Potential findings and implications	
	History	Physical examination
	cerebellar dysfunction, Wernicke's), seizures (alcohol/sedative withdrawal or hyponatremia)	Tremor (alcohol/sedative withdrawal) Clonus (alcohol/sedative withdrawal)
Endocrine/ reproductive	Poor diabetes control, sexual dysfunction, menstrual irregularity or amenorrhea, infertility, symptoms of sexually transmitted disease (e.g., genital ulcers, discharge)	Gynecomastia (males), testicular atrophy (alcohol use); signs of sexually transmitted disease
Hematologic	Easy bruising (thrombocytopenia), enlarged "glands" (lymph nodes; due to infectious or neoplastic complications)	Bruises Lymphadenopathy
Dermatologic	Excessive hair growth (lanugo), hair loss, brittle nails, yellowish skin (jaundice, carotenodermia)	Lanugo (anorexia nervosa) Hair loss, brittle nails (malnutrition) Yellowish discoloration (jaundice or carotenodermia) Excoriations (stimulant use) Needle tracks Evidence of accidental trauma or deliberate self-harm
Psychiatric	Insomnia, confusion, poor memory and concentration, depression, anxiety, obsessive–compulsive behaviors, suicidal ideation, self-harm (all present in both eating and substance use disorders), hallucinations (intoxication with stimulants, marijuana; withdrawal from alcohol and sedative-hypnotics)	Depressed or anxious affect Fluctuations in attention or level of consciousness (delirium) Responding to internal stimuli Impaired attention or memory on cognitive testing

chlamydial infection, diabetes mellitus, chronic obstructive pulmonary disease, and lipid disorders. A detailed discussion of preventive health care is beyond the scope of this chapter; details are available at the Agency for Health Care Research and Quality website (http://www.ahrq.gov/professionals/clinicians-providers/guidelines-recommendations/guide/index.html).

Suggested routine laboratory and imaging studies for patients with ED and SUD are listed in Table 15.5, along with some potential abnormal findings. Indications for osteoporosis screening in patients with ED and SUD may change with the US Preventive Services Task Force updated guidelines issued in 2011, which recommend dual-energy X-ray absorptiometry (DXA) screening for individuals with a risk for fracture equal or greater than that of a 65-year-old woman (9.3 %). Fracture risk can be calculated using the WHO FRAX tool (http://www.shef.ac.uk/FRAX/). Many individuals with ED and SUD will likely fall into that range. Current guidelines recommend DXA for female patients with AN who have had amenorrhea for 6 months or longer; however, since weight, rather than duration of amenorrhea, is the strongest predictor of bone mass in patients with AN (Baker,

Table 15.5 Routine laboratory and imaging studies for patients with eating disorders and substance use disorders

Study or test	Potential abnormalities and comments
Laboratory studies	
Complete blood count	Leukopenia, anemia, thrombocytopenia: malnutrition, bone marrow suppression from alcohol Anemia: GI bleeding Leukopenia: HIV infection Leukocytosis: bacterial infection
Comprehensive metabolic panel	Glucose: ↓ malnutrition, ↑ insulin omission or undetected diabetes Sodium: ↓ water loading, heavy alcohol use; ↑ severe dehydration with stimulants Potassium: ↓ vomiting, laxatives, diuretics, refeeding, heavy alcohol use; ↑ rhabdomyolysis with opioid OD or stimulants Chloride: ↓ vomiting; ↑ laxatives Bicarbonate: ↑ vomiting; ↓ laxatives, alcoholic ketosis, rhabdomyolysis Urea nitrogen: ↑ dehydration, renal failure (e.g., rhabdomyolysis), GI bleeding Creatinine: ↑ renal failure (e.g., rhabdomyolysis); ↓ muscle wasting (normal may be relatively elevated in malnutrition and cirrhosis; therefore, creatinine clearance should be calculated) Calcium: ↓ rhabdomyolysis, significant or prolonged hypomagnesemia Aspartate transaminase, alanine transaminase: ↑ malnutrition, refeeding, alcoholic hepatitis, hepatitis B or C, nonalcoholic fatty liver disease, rhabdomyolysis Albumin: ↓ advanced alcoholic liver disease; rarely secondary to eating disorder alone Total bilirubin: ↑ advanced alcoholic liver disease
Phosphate	↑ rhabdomyolysis; ↓ malnutrition, refeeding syndrome
Magnesium	↓ malnutrition, heavy alcohol use, laxatives, refeeding syndrome
Amylase	↑ self-induced vomiting, pancreatitis, heavy alcohol use; also obtain lipase if pancreatitis is suspected
Urinalysis	Decreased specific gravity in water loading, myoglobin casts in rhabdomyolysis
Thyroid studies	Euthyroid sick syndrome (low T3, low-normal T4, low-normal TSH) in malnourished patients
Pregnancy test	Amenorrheic females can ovulate and are at risk for unplanned pregnancy
Gonadotropins and sex steroids	↓ LH, FSH, estradiol (or testosterone) in malnutrition; ↑ estradiol and ↓ testosterone with heavy alcohol use; ↓ testosterone with opioids (variable); also check prolactin if amenorrhea persists despite abstinence and weight restoration
Lipid panel	↑ heavy alcohol use, bingeing; ↑ cholesterol in anorexia nervosa
Urine drug screen	Most commercial tests screen for benzodiazepines, barbiturates, cocaine, amphetamines, marijuana, PCP, and opioids. See text for discussion

(continued)

Table 15.5 (continued)

Study or test	Potential abnormalities and comments
Hepatitis B and C serologies	
HIV screening	High-risk patients (those with substance use disorders fall into this group) should be screened annually[a]; some states may require separate consent form
Serologic test for syphilis	False-positive RPR or VDRL may be seen in IV drug users and should be confirmed by a treponemal test (e.g., FT-ABS)
Tuberculosis testing	For IV drug users, HIV-positive patients, patients with history of incarceration or homelessness, and symptoms of TB
GGT and CDT	Consider gamma-glutamyl transferase (GGT) and carbohydrate-deficient transferrin (CDT) in combination, to evaluate for heavy drinking (>4 drinks/day) in patients who may be underreporting; see text for details
Other studies	
Electrocardiogram	Arrhythmias; bradycardia in malnutrition; tachycardia in stimulant intoxication, alcohol or sedative withdrawal, refeeding syndrome; prolonged QTc in electrolyte abnormalities, severe malnutrition; evidence of ischemia (ST segment depression, T wave inversion) may be seen with stimulant intoxication
DXA (dual-energy X-ray absorptiometry)	See text for discussion. Contraindicated in pregnancy

[a]Branson, B. M., Handsfield, H. H., Lampe, M. A., Janssen, R. S., Taylor, A. W., Lyss, S. B., & Clark, J. E. (2006). Revised recommendations for HIV testing of adults, adolescents, and pregnant women in health-care settings. Centers for Disease Control and Prevention. Available at http://www.cdc.gov/mmwr/preview/mmwrhtml/rr5514a1.htm

Roberts, & Towell, 2000; Turner et al., 2001), DXA should be considered for all individuals with low weight, especially if there is a history of smoking and/or alcohol use. Other patients for whom DXA should be considered include those with long-standing AUD (Berg et al., 2008) and those who have had gastric bypass surgery (Vilarrasa et al., 2009).

Some substances are misused almost exclusively by patients with ED for purposes of appetite suppression and weight control and are not assessed by traditional laboratory tests for substances of abuse. These include laxatives, ipecac, caffeine, thyroid hormone, diet pills, and diuretics. It is important to detect the use of these substances as they can both cause specific medical complications (e.g., cardiomyopathy from ipecac use, cathartic colon from laxative abuse) and exacerbate the medical complications of ED and SUD (e.g., caffeine abuse interacting with alcohol withdrawal to cause seizures). The most important assay is a careful history in which the patient is asked specifically about the use of each of these substances; however, in selected cases, laboratory testing is available and can be useful (Table 15.6). A wide variety of other substances may also be used including performance-enhancing agents, growth hormones, and various supplements advertised as effective for weight loss. Patients often do not volunteer information about these substances, and it is wise to enquire systematically about their possible use.

Table 15.6 Prevalence, diagnosis, and medical complications of substances frequently misused specifically by patients with eating disorders

Substance	Prevalence	Diagnosis	Complications
Laxatives	15–75 % (lifetime)	History Fecal magnesium (magnesium-containing laxatives) Specific laboratory tests are available for anthraquinones (senna, cascara), bisacodyl, phenolphthalein, and polyethylene glycol, but are not 100 % sensitive or specific	Dehydration with secondary hyperaldosteronism Electrolyte disturbances: hypokalemia, hypomagnesemia, metabolic acidosis (acute) and alkalosis (chronic) Chronic constipation Cathartic colon Rectal prolapse Renal tubular injury and renal failure Withdrawal syndrome (constipation, obstipation)
Ipecac	7–28 % (lifetime)	History Serum/plasma assays available	Complications of vomiting Arrhythmias Hypotension Skeletal myopathy Cardiomyopathy (based on cumulative exposure)
Caffeine	45 % (current excessive use vs. 13 % in controls)	History Serum assay available	Arrhythmias Seizures Death in overdose Increased heart rate and blood pressure Vomiting, reflux, increased gastric acid secretion Impaired fetal growth Exacerbation of dehydration (mild diuresis if used alone) Tremor, muscle twitches Anxiety Insomnia Psychosis reported in patient with anorexia nervosa Withdrawal syndrome (headaches, lethargy) Impaired calcium absorption
Diet pills[a]	32 % (lifetime)	History	Adverse effects of caffeine (caffeine and guarana, see above) Nausea, vomiting (hoodia) Paresthesias (hoodia)
Diuretics	31 % (lifetime)	History Urine electrolytes[b] Diuretic screen urine assay available (detects several common diuretics)	Dehydration, hypotension, syncope Electrolyte abnormalities (hypokalemia, hypomagnesemia, hyponatremia, hypocalcemia)

(continued)

Table 15.6 (continued)

Substance	Prevalence	Diagnosis	Complications
			Metabolic alkalosis Renal failure
Thyroid hormone	6.7 %	History Thyroid studies (TSH, T4, thyroglobulin: in thyroid hormone abuse, TSH will be low, T4 elevated, and thyroglobulin low; in other causes of hyperthyroidism, T4 and thyroglobulin are both elevated)	Tachycardia, arrhythmias Weight loss Hair loss Heat intolerance, sweating Diarrhea Hyperreflexia Tremor Amenorrhea

Source: Bakke et al. (2010); Burgalassi et al. (2009); Cohen (1980); Curatolo and Robertson (1983); Roerig et al. (2003); Roerig, Steffen, Mitchell, and Zunker (2010); Sepkowitz (2013); Shaul, Farrell, and Maloney (1984); Steffen, Mitchell, Roerig, and Lancaster (2007); Woodside, Walfish, Kaplan, and Kennedy (1991)

[a]Ingredients vary; the most common ingredients of over-the-counter diet pills include caffeine, green tea extract, guarana extract, hoodia, and bitter orange extract

[b]In the setting of dehydration, increased urine sodium, potassium, and chloride are suspicious for diuretic abuse unless there is underlying kidney disease

15.3.3.2 Laboratory Tests to Detect Substance Use

Laboratory testing plays a key role in the evaluation of SUD. Accurate interpretation of results requires knowledge of the limitations on detection (e.g., which substances are assayed, and how long they are present in the body), and the potential for false positive and negative results. Most drug screening tests include assays for benzodiazepines, barbiturates, cocaine, amphetamines, marijuana, phencyclidine, and opioids. Synthetic opioids such as methadone, oxycodone, hydromorphone, fentanyl, buprenorphine, meperidine, and hydrocodone are not detected unless specifically assayed. Urine is the usual source for testing because it can be collected easily and noninvasively, but specimens are easily adulterated or substituted. Commonly used parameters for detecting an invalid specimen include temperature, pH, specific gravity, and creatinine, but clinical laboratories do not routinely measure these, nor do they typically assay for oxidizing adulterants.

It is recommended that clinicians contact their laboratory regarding the availability of validity testing for specimens. In addition, the laboratory should be able to provide a list of drugs that cross-react with its assay and thus cause false positive results. Commonly prescribed drugs that can cause false positives include bupropion, ranitidine, and trazodone (amphetamine); some fluoroquinolones and rifampicin (opioids); dextromethorphan, diphenhydramine, and venlafaxine (phencyclidine); and fluoxetine, haloperidol, risperidone, sertraline, and verapamil (LSD), but this list is only partial. Gas chromatography with mass spectroscopy (GC/MS) is considered the gold standard for confirming positive screening assays but requires that specimens be sent out to a reference laboratory and thus is not usually practical for most clinical needs; it should be considered if medicolegal issues are anticipated. The duration of detection is variable and depends on the

Table 15.7 Approximate detection time for screening urine immunoassays

Drug	Approximate duration of detection after last use
Alcohol	7–12 h
Amphetamine	1–4 days
Barbiturates	Short acting (e.g.,. pentobarbital, secobarbital), 4–6 days Intermediate acting (e.g., amobarbital), 3–8 days Long acting (e.g., pentobarbital), 10–30 days
Benzodiazepines	Short acting (e.g., triazolam), 24 h Intermediate acting (e.g., alprazolam, clonazepam, lorazepam), 1–12.5 days Long acting (diazepam), 3–8 days (up to 24 days for nordiazepam)
Marijuana	Single use: up to 3 days Moderate use: up to 4 days Heavy use: up to 10 days Chronic, heavy use: up to 30 days
Opioids	Most opioids: 1–3 days Methadone: 3–11 days
Phencyclidine	1–10 days
LSD	4 h
GHB	12 h

Source: Substance Abuse and Mental Health Services Administration. (2012). *Clinical drug testing in primary care. Technical Assistance Publication (TAP) 32* (HHS Publication No. (SMA) 12-4668). Rockville, MD: Substance Abuse and Mental Health Services Administration

dose, route of administration, pattern of use, laboratory cutoff, and individual metabolism; approximate detection time for screening urine immunoassays is listed in Table 15.7. Because alcohol is usually present in blood or urine for only a few hours after use, blood tests for gamma-glutamyl transferase (GGT) and carbohydrate-deficient transferrin (CDT) may be used in combination if heavy drinking (>4 drinks/day) is suspected in patients who may be underreporting; a formula based on the results has shown 90 % sensitivity and 98 % specificity (Hietala, Koivisto, Anttila, & Niemela, 2006).

15.4 Medical Issues Affecting Patient Placement Decisions

Choosing an appropriate level of care is a critical component of the initial assessment of patients with both ED and SUD, and while factors such as risk for harm to self or others and the absence of social support play a role, medical risks are a key consideration. Placement should be guided by the results of a thorough history, physical examination, and laboratory evaluation, as discussed above. The first question to address is whether the patient requires referral to an emergency room for possible medical admission.

A history of seizure, syncope, chest pain, abdominal pain, gastrointestinal bleeding, or shortness of breath and physical examination findings such as fever (temperature > 100.4 °F), markedly abnormal vital signs (pulse <40 or >100

(unless explained by stimulant use or alcohol withdrawal), blood pressure <90/60 mmHg or >180/110 mmHg or with orthostatic drop >20 mmHg), decreased or fluctuating level of consciousness, clonus, or hallucinations should prompt urgent referral to an emergency room, as should significant abnormalities on the electrocardiogram (ECG) or metabolic panel. Mild electrolyte abnormalities (e.g., potassium >3.0, magnesium >1.0, phosphate >1.0) can usually be replaced orally if the patient is cooperative, but more severe abnormalities require IV replacement, which is important because most inpatient/residential ED and SUD facilities do not provide IV therapy.

A second question to address is whether or not the patient needs the treatment intensity provided by an inpatient setting (or a residential setting with 24-h nursing care and laboratory). While nonmedical factors such as psychiatric comorbidity, severity of illness, availability of support, and failure at lower levels of care also influence this decision, key medical considerations are the risk for refeeding syndrome and/or the need for medically managed substance withdrawal.

15.4.1 Refeeding Syndrome Placement Choices

The refeeding syndrome is a potentially life-threatening complication of reinitiating nutrition in severely malnourished individuals. Its pathophysiology involves shifts of phosphate from the blood into the intracellular space as a result of the insulin response to glucose. Since total body phosphate is depleted due to malnutrition, the blood phosphate decreases, resulting in low adenosine triphosphate levels in tissues and a host of serious complications, including congestive heart failure, respiratory failure, rhabdomyolysis, blood dyscrasias, delirium, and seizures. Potassium also shifts intracellularly and this can result in severe hypokalemia. Risk factors include fasting or very low caloric intake, weight loss exceeding 15 % in the preceding 3–6 months (even if normal or above-normal weight at presentation), history of alcohol or drug abuse, and electrolyte abnormalities. An inpatient setting is required due to the need for close monitoring of food intake, frequent vital sign checks, daily laboratory assessments, and administration of oral phosphate and potassium. Monitoring and prevention of refeeding syndrome is discussed below.

15.4.2 Alcohol Withdrawal Placement Choices

Although there is some evidence that mild to moderate uncomplicated alcohol withdrawal can be safely managed in the outpatient setting (Hayashida et al., 1989), an inpatient setting will be required for most patients with co-occurring ED and SUD who have been misusing alcohol, sedative-hypnotics, or opioids. This is consistent with the SAMHSA guidelines for detoxification, which state, "for alcohol, sedative-hypnotic, and opioid withdrawal syndromes, hospitalization (or some form of 24-h medical care) is often the preferred setting for detoxification, based on principles of safety and humanitarian concerns" (Center for Substance

Abuse Treatment, 2006, p. 16). In particular, patients who are underweight, who have lost significant amounts of weight, or who are purging by any means are medically compromised, and this in turn heightens the risk for alcohol or sedative-hypnotic withdrawal delirium, which can be life threatening. While opioid withdrawal is not dangerous, intensive support and supervised opioid taper and/or adjunctive medications are needed to manage the severe discomfort that almost always precipitates a relapse outside a structured setting. Management of withdrawal syndromes is discussed below.

15.4.3 Placement Choices for Patients with Both an ED and SUD

Since most inpatient facilities specialize in the treatment of SUD or ED, but not both, the question often arises regarding the appropriate placement of a patient suffering from both disorders. (See Chap. 21.) From a medical standpoint, if a patient is at risk for refeeding syndrome but does not have a severe alcohol, sedative-hypnotic, or opioid use disorder, an inpatient ED unit would generally be the best initial placement. Conversely, patients who are not at risk for refeeding syndrome but who require medically managed withdrawal are best initially managed in an inpatient SUD unit. Placement of patients who require medically managed withdrawal and who are at risk for the refeeding syndrome should be decided on a case-by-case basis. An important deciding factor is the expertise and comfort level of the nursing staff with substance withdrawal protocols and complications. If the staff on an ED unit is not experienced with managing alcohol, sedative-hypnotic, or opioid withdrawal, it is usually best for the patient to be detoxified in an SUD unit with close oversight by a medically trained clinician familiar with the prevention and treatment of refeeding syndrome.

15.5 Management of Some Commonly Encountered Medical Issues

Medically trained clinicians who treat patients with ED and SUD should be familiar with the monitoring and prevention of refeeding syndrome and medically managed withdrawal and should also be able to manage, with consultation, less serious but frequently encountered problems such as gastrointestinal complaints, mild electrolyte abnormalities, amenorrhea, and osteoporosis.

15.5.1 Refeeding Syndrome Management

The refeeding syndrome is a rare but potentially lethal complication of nutritional restoration that is caused by hypophosphatemia resulting from intracellular shifts of phosphate in response to insulin. Prevention begins with identifying individuals at risk and measuring serum electrolytes (including magnesium and phosphorous)

Table 15.8 Refeeding syndrome risk factors and preventive strategies

Refeeding syndrome risk factors	Strategies to prevent refeeding syndrome
Very low weight (BMI < 15)	Start calories at 25–30 kcal/kg/day
Rapid weight loss	Advance by 300 calories every 3–4 days
No or limited food intake 1 week prior	Monitor vital signs and edema at least daily
Large amount of weight loss	Monitor electrolytes and phosphorous daily for a week, then biweekly
Electrolyte abnormalities	
Alcohol or substance abuse	

before initiating refeeding. Initial daily calorie intake should be 25–30 kcal/kg, and calories should be advanced by 300 calories every 3–4 days until intake is adequate to produce 2–3 lb. of weight restoration per week. In patients who are judged to be at risk for refeeding syndrome, electrolytes, including magnesium and phosphorous, should be obtained daily for a week, then biweekly in patients who show initial abnormalities. Vital signs should be measured at least daily, as tachycardia, which may be relative (i.e., a heart rate of 80 in a patient whose usual heart rate is 40), can be an early harbinger of the refeeding syndrome.

Risk factors for refeeding syndrome and some strategies for preventing it are listed in Table 15.8. Additionally, in patients who are at risk for the refeeding syndrome, clinicians should monitor finger stick glucose levels in the mornings, as these patients are also at risk for hypoglycemia when they produce insulin in response to refeeding but have very low glycogen reserves. Thiamine should be administered before initiating refeeding, as Wernicke–Korsakoff syndrome has been reported in individuals with AN even in the absence of alcohol abuse (Altinyazar, Kiylioglu, & Salkin, 2010; Saad, Silva, Banzato, Dantas, & Garcia, 2010). Usually electrolyte abnormalities can be managed by electrolyte replacement, rather than by reducing calories, but if edema develops the caloric advancement may need to be held. Recently some (Katzman, 2012; Kohn, Madden, & Clark, 2011) have questioned the need for low initial calorie intakes and slow advancement, but this is based on studies of otherwise healthy adolescents and as yet there is no research supporting more rapid refeeding protocols in adults.

15.5.2 Alcohol and Sedative-Hypnotic Withdrawal Management

Signs and symptoms of acute alcohol withdrawal generally start 6–24 h after the last drink and may begin while there is still alcohol present in the body. Symptoms include anxiety, irritability, difficulty sleeping, decreased appetite, nausea, and hypersensitivity to sound and light. Brief auditory and visual hallucinations may also occur. Signs include dilated pupils, tremor, sweating, elevated pulse and blood pressure, and sometimes fever, although infection must be ruled out. The most severe manifestations include seizures and delirium. Seizures usually occur within

the first 48 h, although they have been reported days after the last drink. The most important risk factor is a history of previous withdrawal seizures; long history of alcohol use disorder, poor nutrition, and electrolyte abnormalities and history of head injury also contribute to risk for alcohol withdrawal seizures. Delirium does not develop suddenly but occurs as a progression from severe withdrawal symptoms and is related to the amount and duration of alcohol consumption, severity and number of prior withdrawal episodes, increasing age, general health, and nutritional status. It is characterized by the presence of other signs of alcohol withdrawal together with confusion, disorientation, and fluctuating levels of attention and awareness; patients are often agitated and appear to hallucinate.

Patients with ED who are malnourished (including those who are at normal weight) and/or who purge by vomiting or laxative or diuretic abuse are at risk for withdrawal-related seizures and delirium. Patients presenting with delirium should be managed in a general medical setting initially; most other patients are candidates for medically managed withdrawal in inpatient SUD or ED facilities.

Although there is increasing interest in the use of anticonvulsants for managing alcohol withdrawal due to the adverse effects and addictive potential of benzodiazepines (Polycarpou et al., 2005), data are still limited, and at present, benzodiazepines remain the treatment of choice for managing the discomfort of withdrawal and preventing seizures and delirium (Amato, Minozzi, & Davoli, 2011).

15.5.2.1 Benzodiazepines and Alcohol Withdrawal

There are two basic approaches to managing alcohol withdrawal with benzodiazepines: symptom-triggered dosing or scheduled dosing. The former is best based on a structured measure of alcohol withdrawal signs and symptoms, usually the Clinical Institute Withdrawal Assessment of Alcohol Scale, revised (CIWA-Ar; Stuppaeck et al., 1984). This scale should be administered by trained nursing staff every 4 h while the patient is awake, and medication is given depending on the score. This approach has been shown to reduce the given quantity of benzodiazepine and to shorten the duration of treatment without increasing the risk of withdrawal complications (Daeppen et al., 2002).

However, a fixed schedule of dosing is probably safer when the patient has a history of withdrawal seizures or delirium (especially during a supervised detoxification), and if the patient has a medical condition such as coronary artery disease in which the autonomic instability of withdrawal might be unsafe, or when the nursing staff are not trained in the use of the CIWA-Ar. If a fixed dose is given, allowance should be made for repeating doses if needed for symptom control. Another consideration is the choice of benzodiazepine: when rapid control of symptoms is needed, shorter-acting agents such as diazepam or lorazepam are preferred, but longer-acting agents such as chlordiazepoxide may be preferable for preventing seizures. There are few data to guide this, however, and those that exist do not show clear differences. However, benzodiazepines that are metabolized in the liver (all except lorazepam, oxazepam, and temazepam) should be avoided in patients with significant liver disease, and only lorazepam is reliably absorbed via the

Table 15.9 Suggested medical management for alcohol withdrawal

	Symptom-triggered	Scheduled dose[a]
Monitoring	Vital signs q4–8 h while awake	Vital signs q4–8 h while awake
	CIWA-Ar q4 h while awake[b]	
Medications	Oxazepam 30 mg q4 h prn CIWA-Ar > 8 Oxazepam 60 mg q4 h prn CIWA-Ar > 15 Thiamine 100 mg daily Folate 1 mg daily Multivitamin 1 daily	Oxazepam 30 mg q6 h × 4 doses then 15 mg q6 h × 8 doses Thiamine 100 mg daily Folate 1 mg daily Multivitamin 1 daily
Ancillary medications for use with either regimen		
Acetaminophen and/or nonsteroidal anti-inflammatory agents as needed for pain (limit dose if liver disease present) Antiemetics (e.g., ondansetron, promethazine) as needed for nausea and vomiting		

Based on Mayo-Smith, M. F. (1997). Pharmacological management of alcohol withdrawal: A meta-analysis and evidence-based practice guideline. *JAMA*, *278*, 144–151

[a]Patients receiving medication on a scheduled basis should be monitored closely and additional medication should be given if scheduled doses are inadequate; this may take the form of "call MD" orders, such as "call MD for tremulousness, diaphoresis, pulse > 100," etc.

[b]May discontinue monitoring when CIWA-Ar score < 8 for 24 h

intramuscular route. All patients with alcohol withdrawal should receive thiamine, and those who have hallucinations not responding to benzodiazepines alone may respond to low doses of an antipsychotic (e.g., haloperidol or olanzapine 2–5 mg), although these patients should be monitored very carefully for delirium. Table 15.9 outlines an approach to alcohol withdrawal.

15.5.2.2 Sedative-Hypnotic Withdrawal

Management of sedative-hypnotic withdrawal may be similar to that for alcohol withdrawal, i.e., the drug is discontinued and withdrawal managed with benzodiazepines, as outlined above. However, there are no data regarding the relative safety and efficacy of symptom-triggered sedative withdrawal, and individuals who are using high doses are probably best managed using a scheduled dose regimen. In cases in which patients were initially treated with the sedative for an anxiety disorder, it may be preferable to institute a longer taper, although relapse risk is high, unless the patient can remain in a structured setting. This involves substituting a long-acting benzodiazepine for the one abused, if necessary (e.g., clonazepam for alprazolam), and then providing this in divided doses (2–3 times per day) for 1–2 weeks before initiating a gradual taper at a rate of 10 % per week. The taper may need to be slowed for the final 25 %. At the beginning of the taper, patients should be started on another form of treatment for their anxiety disorder (an SSRI and/or psychotherapy). The onset of withdrawal symptoms depends on the half-life of the agent. For example, for short-acting benzodiazepines symptoms begin within a day and reach their peak in 4–5 days. Onset for longer-acting benzodiazepines begins about the third day after cessation of the drug and reaches

peak intensity in about 10 days. However, the duration of symptoms may be protracted and last for months (Vikander, Koechling, Borg, Tonne, & Hiltunen, 2010).

15.5.2.3 GHB Withdrawal

Gamma-hydroxybutyrate (GHB) withdrawal can be life threatening and frequently requires high doses of benzodiazepines for management. Clinicians should always ask about the use of this agent in patients who present with sedative-hypnotic withdrawal syndrome, since it is detectable in urine assays for only 12 h after last use, and there should be a low threshold for referral to a general medical setting (van Noorden, van Dongen, Zitman, & Vergouwen, 2009).

15.5.3 Opioid Withdrawal

Because agonist therapy is the most effective treatment for chronic IV opioid dependence (Center for Substance Abuse Treatment, 2005a), maintenance on methadone or buprenorphine–naloxone should be considered for all individuals with opioid use disorders. Methadone maintenance treatment is only available at licensed centers (directory available at http://www.methadonecenters.com/) and patients need to meet eligibility criteria, which include a history of problematic use for at least a year plus prior ineffective treatment attempts. Buprenorphine–naloxone can be prescribed by physicians who have completed Substance Abuse and Mental Health Services Administration (SAMHSA)-approved training and obtain a DATA 2000 waiver and an additional DEA number (Substance Abuse and Mental Services Administration (SAMHSA), 2000). Although there have been no studies of patients with ED who are maintained on methadone or buprenorphine–naloxone, this may represent the best option for patients who have a lengthy history of opioid use disorder with multiple relapses, as it would allow them to stabilize the opioid use disorder while addressing the ED. For other patients, detoxification may be the preferred option; this would include patients who have less severe opioid use disorders or who cannot access or decline agonist therapy.

Opioid withdrawal is not dangerous, but can be extremely uncomfortable, and this frequently leads to relapse. Symptoms include anxiety, nausea, vomiting, diarrhea, and muscle and bone aches; and signs include dilated pupils, piloerection, lacrimation, rhinorrhea, and increased heart rate and blood pressure. As for alcohol withdrawal, a structured instrument, the Clinical Opiate Withdrawal Scale (COWS), is available to standardize the assessment of withdrawal (Wesson & Ling, 2003). To manage discomfort and reduce relapse, several strategies for pharmacological management have been developed. Two of these involve use of opioid agonists (methadone and buprenorphine or buprenorphine–naloxone) and one involves the off-label use of clonidine, an alpha-2 adrenergic agonist. The latter reduces activation of the locus couruleus, a noradrenergic nucleus in the pons that becomes hyperactive during opioid withdrawal.

15.5.3.1 Opioid Withdrawal in ED Patients

While methadone tapering is the method that has been in use the longest, it can only be used in facilities that are specifically licensed to prescribe it for the treatment of opioid dependence. Typically, methadone is given in 5 mg increments, beginning after the first signs of opioid withdrawal are observed, up to a total of 20 mg in the first 24 h (30 mg in individuals with large habits) until symptoms are stabilized and then tapered by 10–20 % per day in inpatients. Ancillary medications such as acetaminophen and NSAIDs for pain, antiemetics for nausea, and loperamide for diarrhea are also employed.

Given the limitations on methadone's availability and its potential for prolonging the QTc interval (although this occurs primarily at doses much higher than those used for detoxification), it is generally not the medication of choice for detoxifying patients with co-occurring ED. Additionally, a recent Cochrane Review comparing methadone tapers to other forms of pharmacological management (mainly clonidine and buprenorphine–naloxone) found that there were no differences among the treatments in terms of completion of treatment or number of participants abstinent at follow-up (Amato et al., 2013). Another review found that the severities of withdrawal symptoms with methadone and buprenorphine–naloxone were similar, but duration of symptoms was significantly less with buprenorphine–naloxone compared with methadone, and that buprenorphine was more effective than clonidine in terms of reducing withdrawal symptoms and keeping patients in treatment (Gowing, Ali, & White, 2009).

Because clonidine's potential to produce hypotension may be problematic in many ED patients who already have low blood pressures due to malnutrition and/or dehydration, buprenorphine–naloxone is probably the preferred medication for managing opioid withdrawal in patients with co-occurring ED. Unfortunately, as yet there are no clear guidelines regarding dose and duration of buprenorphine–naloxone when it is used for detoxification. A protocol used by the multicenter NIDA trials of outpatient buprenorphine–naloxone detoxification is presented in Table 15.10, along with some guidelines for the use of clonidine.

15.5.4 Common Problems Encountered During Withdrawal from Alcohol, Sedative-Hypnotics, and Opioids

In addition to medical management, patients who are withdrawing from alcohol, sedative-hypnotics, and opioids will need support and education about the time course of symptoms and the need for additional treatment beyond the withdrawal process. Early in the withdrawal period, they may have limited ability to participate in other aspects of the treatment program, and a judicious balance, agreed upon by all staff members, should be struck between allowing extra rest during this time and not permitting patients to completely retreat from therapeutic activities.

Withdrawal from alcohol, sedative-hypnotics, and opioids often produces decreased appetite and gastrointestinal symptoms, and patients may use these symptoms as reasons to avoid eating or to disguise purging. This should be

Table 15.10 Suggested medical management for opioid withdrawal

Buprenorphine–naloxone[a]	Clonidine[b]
Day 1: Verify that patient has signs of opioid withdrawal (e.g., COWS score > 10) prior to administering buprenorphine–naloxone, then give buprenorphine–naloxone 4 mg/1 mg sublingually; repeat in 1–2 h if signs of withdrawal persist Day 2: Buprenorphine–naloxone 8 mg/2 mg Day 3: Buprenorphine–naloxone 16 mg/4 mg Day 4: Buprenorphine–naloxone 14/3.5 mg Day 5: Buprenorphine–naloxone 12 mg/3 mg Day 6: Buprenorphine–naloxone 10 mg/2.5 mg Day 7: Buprenorphine–naloxone 8 mg/2 mg Day 8: Buprenorphine–naloxone 6 mg/1.5 mg Day 9: Buprenorphine–naloxone 6 mg/1.5 mg Day 10: Buprenorphine–naloxone 4 mg/1 mg Day 11: Buprenorphine–naloxone 4 mg/1 mg Day 12: Buprenorphine–naloxone 2 mg/0.5 mg Day 13: Buprenorphine–naloxone 2 mg/0.5 mg	Measure blood pressure before each dose and hold medication if blood pressure is <90/60 Clonidine 0.1 mg every 4–6 h for signs of withdrawal (e.g., COWS score > 10); may increase by 0.1–0.2 mg/day until withdrawal symptoms are consistently suppressed (typical maximum dose is 0.8 mg/day) Stabilize for 3–5 days then taper by 0.1–0.2 mg/day
Ancillary medications to use with either regimen	
Acetaminophen and/or nonsteroidal anti-inflammatory agents as needed for muscle and bone aches Loperamide as needed for diarrhea Dicyclomine as needed for abdominal cramping Antiemetics (e.g., ondansetron, promethazine) as needed for nausea and vomiting Hydroxyzine as needed for anxiety and restlessness Zolpidem or trazodone[c] as needed for insomnia	

[a]Based on Amass, L., Ling, W., Freese, T. E., Reiber, C., Annon, J., Cohen, A., … Horton, T. (2004). Bringing buprenorphine-naloxone detoxification to community treatment providers: the NIDA clinical trials network field experience. *The American Journal on Addictions*, 13(Suppl. 1), S42–S66

[b]Not FDA-approved for treatment of opioid use disorders

[c]Not FDA-approved for treatment of insomnia

suspected if gastrointestinal complaints persist beyond the usual time course of withdrawal or are disproportionate to other signs and symptoms of withdrawal. Patients may require a bland diet or dietary supplements during this time, but they should be expected to eat and should be given appropriate medications to control their symptoms. Finally, some patients may present with both alcohol/sedative-hypnotic and opioid use disorders; in these cases, it is usually best to stabilize the patient on an opioid agonist during the period of alcohol or sedative-hypnotic withdrawal, and then taper the opioid agonist.

15.5.5 Withdrawal from Stimulants, Nicotine, and Marijuana

Withdrawal from stimulants, nicotine, and marijuana is not dangerous and does not require medical management, but it can interfere with patients' ability to fully participate in treatment. Patients who have been using high doses of stimulants may "crash," requiring extra rest and sleep, and they should also be monitored for depression and especially suicidal ideation. Nicotine withdrawal can cause anxiety, depression, irritability, and poor concentration that can impair the ability to participate in treatment and confound the assessment of other psychiatric disorders, including withdrawal from other substances.

There is a variety of nicotine replacement products, including gum, lozenge, nasal spray, inhaler, and patch; probably the delivery method that is least disruptive to the patient's overall treatment program is the patch, since the other methods require multiple administrations per day. The patch dose is based on the number of cigarettes per day as shown in Table 15.11. Heavy smokers will be underdosed by standard single-patch therapy, and high-dose nicotine patch treatment has been shown to be safe and well tolerated (Dale et al., 1995).

Marijuana withdrawal occurs in the majority of chronic, daily users and is characterized by irritability, decreased appetite, anxiety, restlessness, sleep disturbances (including strange dreams), and sometimes depressed mood, tremulousness, and sweating. The onset is 1–2 days after cessation and the full syndrome typically persists for 1–2 weeks, although sleep disturbance may last longer. As for nicotine withdrawal, these symptoms can interfere with patients' ability to participate in treatment and mimic symptoms of psychiatric disorder or other withdrawal syndromes. Currently there is no specific treatment for marijuana withdrawal; one study showed improvement in symptoms with quetiapine, but this was accompanied by an increase in marijuana cravings (Cooper et al., 2012).

15.5.6 Gastrointestinal Complaints

Gastrointestinal complaints are common in patients who have ED and SUD; many of the medical complications of the disorders affect the gastrointestinal system, and withdrawal from some substances can also produce gastrointestinal symptoms that may complicate nutritional rehabilitation, as discussed above. Three commonly encountered complaints that can interfere with treatment are bloating and early satiety, reflux and regurgitation, and constipation. Patients with AN have well-documented delays in gastric emptying for solid food, although studies differ regarding whether gastric emptying for liquids is delayed; studies are also mixed regarding whether delayed gastric emptying is present in patients with BN. They are also mixed regarding whether or not improvement occurs with weight gain (Hadley & Walsh, 2003).

The symptoms of bloating and excessive fullness compound patients' psychological difficulties with eating and may contribute to purging behavior. Management consists of small, frequent meals that are low in fiber, with liquid replacement

Table 15.11 Recommended initial dosing of nicotine patch therapy based on number of cigarettes smoked

Cigarettes smoked per day	Patch dose (mg/day)[a]
<10	7
10–20	14
21–40	21
>40	42

[a]Nicotine patches are available in 7 mg/day, 14 mg/day, and 21 mg/day

of part of the caloric intake if this is ineffective. Medications such as metoclopramide and erythromycin that enhance gastric emptying may be tried if symptoms are severe, but these have significant side effects, including QTc prolongation and extrapyramidal symptoms, and should be used rarely and with great caution. If symptoms persist after significant nutritional improvement, a nuclear medicine gastric emptying study is indicated.

Gastroesophageal reflux likely occurs as a result of delayed gastric emptying and presents with symptoms of heartburn and involuntary regurgitation of food. While there are no studies to guide the management of reflux specifically in patients with ED and SUD, if they also report symptoms of delayed gastric emptying, the interventions discussed above should be implemented. According to guidelines for the management of reflux, in the setting of typical symptoms, further diagnostic evaluation is not needed, and patients should be given an 8-week empiric trial of daily to twice daily proton pump inhibitor therapy. Patients whose symptoms persist despite this treatment or who experience alarm symptoms such as dysphagia, involuntary weight loss, persistent vomiting, or anemia should be referred for upper endoscopy (Katz, Gerson, & Vela, 2013). Constipation is almost universally present in patients who are malnourished and frequently leads to overuse of laxatives. It is useful to educate patients about the reflex hypofunctioning of the colon that occurs in response to decreased calorie intake; other contributing factors include electrolyte abnormalities and dehydration.

In addition to nutritional rehabilitation, adequate hydration with a low dose of a fiber supplement such as psyllium should be given automatically. If the patient does not have a bowel movement after a few days, polyethylene glycol preparations are safe and effective, and glycerin suppositories can be used concurrently to facilitate rectal emptying (American College of Gastroenterology, 2005). Patients who have been abusing laxatives should be started on a polyethylene glycol product at the inception of treatment. This can be given once or twice daily and gradually tapered.

15.5.7 Moderate Electrolyte Abnormalities

While more severe electrolyte abnormalities (e.g., potassium <3.0, magnesium <1.0, phosphate <1.0) should be replaced intravenously in a medical setting, milder abnormalities can usually be corrected with oral medications. Phosphorous is generally given as 250–500 mg four times per day and magnesium as magnesium

oxide 400–800 mg/day. Potassium should be given in amounts sufficient to correct the total body deficit, which is about 100–200 mEq/L for a potassium level between 3.0 and 3.5; this can be accomplished with 20–40 mEq of potassium per day, with daily monitoring of levels. Additionally, dehydration and hypomagnesemia should be corrected concurrently, as both result in renal losses of potassium. Oral hydration should consist of fluids other than plain water, e.g., sports drinks. If there is severe metabolic alkalosis (e.g., bicarbonate level >35), rehydration should be given intravenously. Asymptomatic patients with chronic, mild hyponatremia (e.g., sodium >125) can be managed with nutritional rehabilitation and free water restriction (1–2 L/day); this almost always requires that the patient be managed in an inpatient setting to prevent excessive water drinking.

15.5.8 Amenorrhea and Osteoporosis

Although amenorrhea is no longer part of the diagnostic criteria for AN, a majority of patients with AN will present with it. The first step in evaluation always is to rule out pregnancy, while educating patients that pregnancy can occur in women even when menses are irregular or absent. On laboratory evaluation, patients have a typical "hypothalamic amenorrhea syndrome" with low levels of all gonadal and gonadotrophic hormones. The treatment is weight restoration; in adolescents, attaining a body weight 90 % of average was associated with resumption of menses within 6 months in 86 % of patients (Golden et al., 1997). There are no comparable data in adults. However, clinicians frequently encounter patients who reach their target weight yet do not resume menstruating. These patients should be evaluated for persistence of ED behaviors, such as diets low in fat and protein and excessive exercise, as these factors can also cause amenorrhea even in normal weight individuals (e.g., female athletes). Psychological factors are also associated with persistence of amenorrhea in patients with ED (Poyastro-Pinheiro et al., 2007) and may improve with psychotherapy. While measurement of hormone levels (including prolactin) and referral to a gynecologist are warranted for patients with persistent amenorrhea, the use of hormonal agents to produce bleeding is discouraged, as this may remove an incentive for recovery, and they are not effective for treating low bone density.

Low bone density is also present in a majority of adult patients with AN: in one series, osteopenia was found in 51 % and osteoporosis in 31 % (Winston, Alwazeer, & Bankart, 2008). The treatment is weight restoration and resumption of menstruation, although full normalization of bone mass may not occur (Dominguez et al., 2007). Studies of oral contraceptives, calcium and vitamin D, and exercise have not shown benefit (Mehler & MacKenzie, 2009), although it is worthwhile to test for and treat vitamin D deficiency. There have been three randomized controlled trials of bisphosphonates in patients with AN and low bone density, two showing benefit (Miller et al., 2011; Nakahara et al., 2006) and the other showing none (Golden et al., 2005). Because these agents are teratogenic and long-term safety and efficacy in premenopausal women has not been well demonstrated, a thorough informed

consent process should be conducted, and most clinicians reserve them for extreme cases (e.g., multiple fractures) and require written consent. A single randomized trial in adolescent girls found transdermal estrogen to be effective in improving bone density (Misra et al., 2011), but longer-term efficacy and generalizability to adults are unknown. There are no studies of treatment for low bone density in boys or men with ED. Bone density tests may have the unexpected benefit of enhancing motivation for treatment: patients are often stunned to see a graphic depiction of their bone density marked on a curve showing normal bone density for age that shows their bones are as weak as those of a normal 70-year-old.

15.6 Therapeutic Use of Medical Information

Denial is very common among patients with ED and SUD. As noted earlier in this chapter, there is recent evidence that both neurobiological and psychodynamic factors contribute to denial. Denial often results in delayed treatment and worse medical complications and contributes to an increased likelihood of premature death. For both sets of disorders, several strategies have been used to try to overcome denial and increase the likelihood that patients will enter treatment and have a better chance for a successful treatment outcome.

For ED, an explicit plan to educate patients (and their families) has been utilized to decrease denial by showing the relationship between symptoms, signs, laboratory tests, and behavior (Powers, 1984). This is often most effective when presented to the patient by a physician, particularly the primary care physician, and when print or visual data are available. For example, a patient with AN who purges by vomiting might complain of palpitations (a symptom) and have arrhythmias on physical examination (a sign) and hypokalemia and an abnormal ECG with a prolonged QTc interval (abnormal laboratory testing). The physician could explain that purging by vomiting can lower serum potassium levels (hypokalemia) that, in combination with semi-starvation, can result in a prolonged QTc interval on the ECG and arrhythmias that can increase the chance of a fatal arrhythmia. Showing the patient the ECG and the actual printed laboratory results may help overcome some of the denial. The booklet produced by the Academy for Eating Disorders (Banker et al., 2012) provides a clear description of common presenting symptoms, signs, laboratory abnormalities, imaging studies, and ECG as well as the elements of a comprehensive evaluation.

Although there are many reports of efforts to decrease denial among patients with SUD (particularly alcohol use disorders), only a few have used confrontation at the time that medical complications occur to decrease denial. Early exceptions were among motor vehicle accident patients or burn patients with alcohol use disorders (Bostwick & Seaman, 2004; Powers et al., 1994). More recent reports that have focused on trying to intervene early (prior to severe complications) have found that emotive anti-alcohol messages (especially those related to medical complications) result in defensiveness and may actually increase the level of denial (Brown & Locker, 2009; Brown & Richardson, 2012). It may be that less

confrontational strategies based on symptoms, signs, or laboratory testing and the relationship to alcohol use behavior would be more effective, particularly if this occurs with the primary care physician.

Conclusions

ED and SUD are common and are associated with multiple medical complications that result in very significant elevated premature mortality rates. The combination of an ED and SUD is even more dangerous. Detection of either disorder can be difficult especially in the early stages of the illness. Recognition and diagnosis of the common medical complications for both conditions occurring together can be the foundation for a treatment plan most likely to be effective. Unfortunately, many physicians are unaware of the medical complications of ED (Powers & Cloak, 2013), and stigma toward both sets of conditions is rampant. Specialists in ED and SUD need to educate their colleagues about these medical complications and their management in order to increase the possibility of a successful treatment outcome. The perplexing and frustrating denial that is so frequently seen in both sets of disorders is currently best understood as a complex interactive set of psychodynamic and neurobiological circuits, most likely based on genetic and epigenetic factors.

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The Role of Negative Affect in Eating Disorders and Substance Use Disorders

16

Brian J. Cook, Stephen A. Wonderlich, and Jason M. Lavender

Abstract

Affect regulation is one mechanism that has been implicated in the development and maintenance of both eating disorders and substance use disorders. Specifically, the affective processing model of negative reinforcement posits that negative affect, as a symptom of withdrawal, is the main impetus in substance use disorder development and maintenance. Similarly, a recent transactional model of emotion dysregulation posits that individuals with eating disorders display heightened emotional sensitivity and reactivity, which in turn predisposes these individuals to eating disorder behaviors (e.g., binge eating, purging, etc.) as a means of attempting to modulate heightened negative affect. While affect regulation is similar in eating disorders and substance use disorders, differences in precursors of negative affect, cognitions, and withdrawal symptoms are present in these two forms of psychopathology. Despite these differences, affect regulation models in both eating and substance use disorders have begun to influence treatment. Thus, understanding the role of negative affect may be a key component of treating substance use disorders and eating disorders independently, as well as the co-occurrence of these disorders.

Keywords

Affect regulation • Emotion dysregulation • Eating disorders • Substance use disorders

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16.1 The Role of Negative Affect in Eating Disorders and Substance Use Disorders

Substance use disorders (SUD) and eating disorders (ED) share many clinical similarities. For example, both disorders typically begin in adolescence or early adulthood, include behaviors which may function to maintain the disorder despite harmful consequences, have a high tendency to relapse, and alter the way the individual relates to others (Goodman, 2008). Such similarities suggest that these two disorders may share common mechanisms of development and maintenance. Accordingly, several etiological models have been presented that may explain the co-occurrence of ED and SUD, including neurobiological (see Chap. 3), personality (see Chap. 6), and genetic (see Chap. 5) theories which highlight factors common to both disorders. While each of these theories elucidates possible mechanisms of etiology, they often fail to identify common mediating variables which may clarify the process of SUD and ED development (Harrop & Marlatt, 2010). Affect regulation is one mechanism that has been implicated in the development and maintenance of both ED (Haynos & Fruzzetti, 2011) and SUD (Baker, Piper, McCarthy, Majeskie, & Fiore, 2004) and has been identified as a mediator of both disorders (Gadalla & Piran, 2007; von Ranson, McGue, & Iacono, 2003). Thus, understanding the role of negative affect may be a key component of treating co-occurring SUD and ED.

Examining affect regulation as a possible mechanism of both SUD and ED broadly fits the conceptualization typically referred to as the self-medication hypothesis of SUD. Simply stated, the self-medication hypothesis posits that individuals use drugs or alcohol in an attempt to relieve symptoms of mental disorders (Khantzian, 1985). The simplicity of this hypothesis is broadly appealing but fails to identify specific biobehavioral processes that substance use is thought to influence (Henwood & Padgett, 2007; Lembke, 2012). Negative affectivity may be a clinically and scientifically useful behavioral process to account for the commonalities between SUD and ED, and alleviation of negative affect has been thoroughly researched as a vital component of both SUD (Baker et al., 2004; Measelle, Stice, & Springer, 2006) and ED (Engel et al., 2013; Haynos & Fruzzetti, 2011; Smyth et al., 2007).

This chapter will review two clinically oriented models that include affect as a key construct. First, we will review the role of affect regulation as a key component of a negative reinforcement model of SUD (Baker et al., 2004). Second, we will discuss a model that posits that emotion dysregulation is key in understanding the development and treatment of ED (Haynos & Fruzzetti, 2011). We will focus our discussion of each model on relevant research with a particular emphasis on ecological momentary assessment (EMA) studies. EMA entails multiple assessments conducted "in the moment" in a naturalistic environment (Shiffman, Stone, & Hufford, 2008). The main advantages of this type of data collection are that EMA avoids the problems associated with retrospective recall bias in self-report assessments when they are not conducted in real time and allows detailed study of antecedents and consequences of behavior, which is important for the study

of affect regulation. Moreover, EMA has been successful in examining relationships among affect in SUD (Shiffman, 2009) and ED (Smyth et al., 2007). Finally, we will compare and contrast the SUD and ED literature that has examined the role of affect in the etiology of these disorders.

16.2 Substance Use Disorders

Negative affect regulation has been a widely studied antecedent to substance use and has been included in multiple models of SUD development and maintenance. The initial models that included affect as an explanatory variable suggested that withdrawal syndrome symptoms, including negative affect, appear after a single or very few instances of substance use. Consequently, as the level of the ingested substance begins to fall, withdrawal symptoms increase, and subsequent substance use represents an attempt to manage or alleviate such symptoms (Hull, 1943). Subsequent models have continued to consider the link between negative affect and substance use. For example, the fundamental assertion of Solomon's (1980) opponent-process model of SUD is that every behavioral process has a pleasant or unpleasant affective valence which is followed by an opponent process. Thus, substance use (A process) produces a pleasurable affective state, but when withdrawal symptoms including negative affect (B process) increase, the substance use is repeated to maintain the magnitude of the A process over the B process. Moreover, negative affect is also implicated in theories that emphasize external factors and cues to substance use. For example, social learning theory posits that when negative affect is high, self-efficacy for dealing with external problems (e.g., interpersonal stressors, etc.) is low. Consequently, substance use to alleviate underlying tension may be triggered, in part, by self-regulatory-related expectancies regarding an inability to inhibit drinking (Jung, 1994).

The inclusion of negative affect in each of these theories suggests that affect regulation is a robust phenomenon that may play a pivotal role in SUD etiology. Interestingly, few models have focused primarily on affect regulation as the key factor in understanding the development of SUD. One notable exception, however, is the affective processing model of negative reinforcement (Baker et al., 2004). Simply stated, this model conceptualizes the etiology of SUD in the following four steps. First, the initial instance of substance use is performed in an attempt to experience the desirable effects elicited by the substance (e.g., drinking alcohol produces calming and euphoric effects). Second, as the amount of substance ingested is metabolized and eliminated by the body, unpleasant symptoms of withdrawal begin to present themselves (e.g., hangover symptoms such as anxiety, nausea, headache, etc.). Third, the individual wants to alleviate these unpleasant withdrawal symptoms and therefore uses the substance again in an attempt to elicit the pleasurable effects previously experienced (e.g., the individual drinks more alcohol (aka "hair of the dog") to feel better). Fourth, the individual learns that substance use alleviates the unpleasant withdrawal symptoms and may also alleviate other similar unpleasant experiences (e.g., drinking in response to negative

affect resulting from interpersonal conflicts). Thus, this model posits that symptoms of withdrawal syndrome act to motivate continued substance use and that negative affect is the primary symptom responsible for this process.

Overt physical symptoms of withdrawal (e.g., tremors, insomnia, shaking, etc.) are commonly associated with SUD. Including these symptoms in explanations of SUD becomes problematic because each class of substance may produce very different physical symptoms during withdrawal. In other words, withdrawal symptoms seem to be specific to the type of substance used (WHO, 1992). Therefore, any etiological explanation that includes withdrawal symptoms must focus on symptoms that are universal to all substance use. Negative affect is one symptom that is universal to all substance use (Marlatt & Gordon, 1980). Thus, the universality of negative affect as a withdrawal symptom is the crucial supposition of the affective processing model of negative reinforcement.

While negative affect in general is identified as the key component of Baker and colleagues' (2004) model, it may be more appropriate to say that the intensity and awareness of negative affect are the key considerations in understanding SUD. Specifically, negative affect is a symptom of withdrawal that is likely to be present after the very first substance use episodes. At this point in the development of substance use problems, the level of awareness of negative affect is most likely very low, often only at a preconscious level of awareness, and acts primarily as an interoceptive (internal) cue. For example, an individual may not consciously recognize their current unpleasant emotional state as negative affect resulting from substance use, but is able to detect that additional substance use alleviates this feeling. Furthermore, negative affect intensity may increase and enter conscious awareness as a result of abstinence from substance use or as a consequence of other general stressors (external cues). Thus, the negative affect may be increased by the combination of interoceptive and exteroceptive cues, which increases the number of cues for substance use.

Baker and colleagues (2004) present three main arguments for why the rise in negative affect intensity is a key component to understanding its role in SUD development and maintenance. First, negative affect intensity is related to hot and cool processing of motivational reasoning (Metcalfe & Mischel, 1999). Simply stated, cool processing is best described as a nonemotional basis for motivating behavior, while hot information processing is influenced by emotional memories and less able to be modified by declarative statements (Öhman & Mineka, 2001). Cool processing predominates at low levels of awareness of negative affect but gives way to hot processing as negative affect awareness and intensity increase. Thus, as negative affect intensity increases, the individual not only becomes more aware of the unpleasant emotional state they are experiencing but also recalls the memory of the pleasant emotional state induced by previous substance use episodes. Consequently, the individual is motivated to use substances by the emotional memory of the pleasurable state experienced during previous substance use episodes and the expectancy that it will produce the same outcome.

Second, Baker and colleagues (2004) argue that affect intensity influences an individual's ability to maintain information, set and accomplish goals, make plans,

and follow instructions. Tasks such as these are referred to as cognitive control. At very low levels of negative affect, the individual is not influenced by affective state, and cognitive control is not necessary to prevent the engagement in substance use. Alternatively, when negative affect is very high, cognitive control is impaired because information processing is negatively impacted by emotional factors which influence adaptive decision making. Thus, one major difference between low and high levels of awareness of negative affect is that cognitive control is significantly impaired by the appetitive emotional value of the substance being used at high levels of negative affect.

Third, substance use is not an entirely individual activity. For example, awareness of future unavailability of a drug, social cues for substance use, and interpersonal stressors are largely related to factors that are out of the control of an individual. These motivations for substance use are referred to as modulators. Baker and colleagues (2004) state that at low levels of negative affect, the use of substances tends to be proceduralized and at high levels decision making is based primarily on emotional factors. That is, the individual may be unaware of the external motivational impetus for substance use at low and high levels of negative affect but is aware of the availability of drugs. Therefore, individuals may be most susceptible to modulators at moderate levels of negative affect.

The affective processing model presents the fundamental groundwork to argue that SUD are learned responses to withdrawal-induced negative affect cues which produce a negatively reinforcing reduction in negative affect. Motivation for substance use may arise from interoceptive (e.g., recognition of increased negative affect) or exteroceptive (e.g., modulators) sources. As the unpleasant stimuli are presented, substances are used to ameliorate negative affect. Central to this learning process is that negative affect, whether arising from withdrawal or other external factors unrelated to substance use, is reduced when substance use is performed. This reduction of unpleasant affect is the consequence that would be thought to maintain the disorder. In other words, it is rewarding.

16.3 Ecological Momentary Assessment Research in Substance Use Disorders

Research cited by Baker and colleagues (2004) to support each aspect of the affective processing model of negative reinforcement provides a sound justification supporting the model and adequately addresses many criticisms of affective regulation models. However, recent advances in EMA may provide another method of elucidating the role of affect in SUD (Shiffman, 2009). Interestingly, EMA studies of smoking, alcohol, and other substances have provided new insights into affect regulation that may be specific to each type of substance.

Cross-sectional and retrospective designed studies have consistently supported the role of negative affect regulation in smoking studies (Shiffman, 1993), in spite of some inconsistency (Shiffman, 2009). More recent EMA research has indicated that mood and craving predicted smoking 4 h prior to the behavior, but not 2 h prior

(Berkman, Dickenson, Falk, & Lieberman, 2011), and that smokers that abstained from smoking on their chosen quit day experienced greater increases in negative affect than those who failed to quit (Yeh, McCarthy, & Baker, 2012). Collectively, these EMA studies may point to temporal relationships among affect and smoking that are currently not fully understood.

Similarly, EMA research examining alcohol use has yielded encouraging results that may support the role of affect regulation in substance use. The EMA literature appears to consistently suggest that affect regulation may play a role in several different drinking scenarios (1) when negative affect is experienced early in the week and drinking occurs on the weekend, (2) negative affect is increased early in the day and drinking occurs later that same evening, and (3) drinking is assessed using binge-drinking criterion or only first drink of a drinking episode as the operational definition of drinking (Shiffman, 2009). Moreover, EMA studies that have looked at drinkers who also smoke have found that alcohol is appraised as more negatively reinforcing than cigarettes and that drinking, but not smoking, decreases negative affect (Piasecki et al., 2011). Thus, Baker and colleagues' (2004) associations of affect regulation and negative reinforcement-based learning may be supported.

EMA examinations of other substance use have also revealed interesting findings. Specifically, Carrico and colleagues (2013) report that negative affect was not associated with any measure of methamphetamine use, but positive affect was associated with coping and self-efficacy for managing drug use. Similarly, negative affect is not directly associated with cocaine and heroin use, but is associated with temptation to use (Waters, Marhe, & Franken, 2012), stress preceding use (Preston & Epstein, 2011), and a gender difference indicating women experience greater negative affect after use than do men (Kennedy, Epstein, Phillips, & Preston, 2013). Finally, in a series of EMA studies, Buckner, and colleagues (Buckner, Crosby, Silgado, Wonderlich, & Schmidt, 2012; Buckner, Crosby, Wonderlich, & Schmidt, 2012; Buckner, Zvolensky, & Eckera, 2013; Buckner et al., 2011) report that social anxiety predicted cannabis use. The motivational role of modulators and exteroceptive factors outlined in Baker and colleagues' (2004) model may also be supported by Buckner, Crosby, Wonderlich, and Schmidt (2012) finding that the presence of other cannabis users facilitated marijuana use.

Taken together, SUD studies that have employed EMA designs have found support for the basic assertion that affect regulation plays a pivotal role in substance use while also suggesting some differences that were previously unable to be detected with cross-sectional or retrospective designs. Recent advances in statistical analyses, mobile technology such as GPS-enabled smartphones, and the ability to collect real-time physiological data may advance our understanding of the nuances of affect regulation in SUD (Shiffman, 2009). Thus, EMA designs have the potential to further evidence the associations identified by previous studies and advance our understanding of SUD through better measurement of variables that may not yet be included in conceptual and/or theoretical models. Future EMA research is encouraged to continue exploring and examining the role of affect in SUD.

16.4 Negative Affect in Eating Disorders

A substantial body of empirical research suggests that negative affect is an important factor to consider in relation to ED psychopathology. For example, evidence from both population-based studies and research using clinical samples suggests that mood and anxiety disorders commonly co-occur with ED (Hudson, Hiripi, Pope, & Kessler, 2007; Kaye, Bulik, Thornton, Barbarich, & Master, 2004). Furthermore, beyond diagnostic co-occurrence, evidence suggests that those with ED tend to display elevated subthreshold symptoms of anxiety and depression (e.g., Kaye et al., 2004; Wagner et al., 2006), as well as higher levels of various forms of negative affect (e.g., anger, guilt, hostility; Allen, Scannell, & Turner, 1998; Waller et al., 2003).

Additional support for the relevance of negative affect to ED is provided by research suggesting that those with ED display a pattern of co-occurring personality disorders and traits associated with negative emotionality, including borderline personality disorder features and obsessive-compulsive personality disorder features, as well as broader personality constructs such as neuroticism (e.g., Cassin & von Ranson, 2005). Finally, numerous recent studies have focused on examining emotion-based constructs that are highly associated with negative affect. Among these are negative urgency, a construct defined by the tendency to act rashly in the face of negative affect (Cyders & Smith, 2008), and distress tolerance, which refers to the ability to experience and tolerate negative affective states (Simons & Gaher, 2005). Consistent with the findings noted previously, evidence suggests that elevated negative urgency and poor distress tolerance are associated with ED symptoms, particularly binge eating (Corstorphine, Mountford, Tomlinson, Waller, & Meyer, 2007; Fischer, Settles, Collins, Gunn, & Smith, 2012). Taken together, these distinct but intersecting lines of research point to the importance of considering the role of negative affect in the etiology, maintenance, and treatment of ED.

The specific role of negative affect in ED has been emphasized to varying degrees in existing etiological/maintenance and treatment models. For example, the escape theory of binge eating (Heatherton & Baumeister, 1991) posits that binge eating behavior is motivated by a desire to divert attention from the negative affective experience associated with aversive self-perceptions. In another model that is focused on a more specific facet of negative affect, Strober (2004) highlighted the role of fear and anxiety in AN, suggesting that individuals with AN exhibit a propensity toward anxiety and fear learning processes, as well as a greater resistance to extinction of learned fear responses. Additionally, although emotion processes are not a primary focus in the model, Fairburn, Cooper, and Shafran's (2003) transdiagnostic theory of ED maintenance addresses the role of mood intolerance, particularly with regard to associations with binge eating and compensatory behaviors.

Numerous models of ED psychopathology thus address the role of negative affect. Accordingly, numerous recent emotion-based psychotherapeutic treatments for ED have emerged. Included among these interventions are dialectical behavior therapy (DBT; Safer, Telch, & Chen, 2009), integrative cognitive-affective therapy

(ICAT; Wonderlich et al., 2014), and emotion acceptance behavior therapy (EABT; Wildes & Marcus, 2011). Each of these interventions focuses heavily on the role of negative affect in precipitating and maintaining ED behaviors, with a particular emphasis on providing skills for managing negative affective states. In sum, existing theoretical and treatment models of ED psychopathology vary to some extent in the role or relative emphasis on negative affect, although more recent theoretical models and emerging treatments focus more heavily on the importance of negative affect and related emotion constructs.

16.5 Emotion Dysregulation Model of Eating Disorders

Although several of the ED psychopathology models discussed above address negative affect as a relevant factor, a recent model proposed by Haynos and Fruzzetti (2011) provides a useful framework for conceptualizing the role of negative affective states and related emotion constructs in ED. This proposed transactional model of emotion dysregulation focuses specifically on AN. However, it can also be applied to ED more broadly, and thus the discussion of the model here will focus on ED psychopathology as a whole. The basic theory of this model is that individuals with ED display an underlying emotional vulnerability characterized by elevated emotional sensitivity and reactivity, which in turn predisposes these individuals to frequently experience elevated emotional arousal in response to various stimuli (e.g., cognitions, environmental stressors, etc.). This frequent experience of heightened negative affect promotes ED behaviors (e.g., binge eating, purging, etc.) as a means of attempting to modulate affect, thus functioning as a form of maladaptive emotion regulation. Additionally, variables are also posited to influence the underlying emotional vulnerability. Specifically, both weight loss/starvation state and invaliding responses from others (which result due to deficits in emotional expression by the eating disordered individual) are also proposed to contribute to and exacerbate emotional vulnerability, thus perpetuating the cycle and maintaining ED symptoms over time. The focus of this brief review of the literature relevant to this model will be on the primary emotion components within the model (for further explanation of the inaccurate expression and invalidating environmental components of the model, see Haynos & Fruzzetti, 2011).

There is growing evidence suggesting that individuals with ED display a broad pattern of deficits in various dimensions of emotion regulation (Harrison, Sullivan, Tchanturia, & Treasure, 2010; Racine & Wildes, 2013), and existing empirical findings lend support to the various associations addressed in the Haynos and Fruzzetti (2011) emotion dysregulation model of ED psychopathology. For example, evidence for an underlying emotional vulnerability can be found in research suggesting that those with ED display an attentional bias to emotionally evocative cues (Shafran, Lee, Cooper, Palmer, & Fairburn, 2007), individuals who binge eat display greater fluctuations in anxiety and depression than those who do not binge eat (Lingswiler, Crowther, & Stephens, 1989), and days characterized by

fluctuating levels of negative affective states are common in bulimia nervosa (BN; Crosby et al., 2009) and anorexia nervosa (AN; Lavender et al., 2013).

Additionally, as noted previously, the presence of heightened negative emotional arousal in ED is evidenced by the high rates of co-occurring affective disorders (Hudson et al., 2007; Kaye et al., 2004), as well as elevated negative affective states and symptoms (Allen et al., 1998; Wagner et al., 2006; Waller et al., 2003). Perhaps most importantly, an extensive literature supports an association between these negative affective states and ED symptoms, particularly behaviors such as binge eating, purging, dietary restriction, etc. For example, negative affect has been identified as a common antecedent for binge eating behaviors (Haedt-Matt & Keel, 2011), and evidence suggests that the likelihood of various ED behaviors tends to coincide with elevations in negative affect and anxiety (Crosby et al., 2009; Lavender et al., 2013). Furthermore, studies utilizing mood induction techniques in conjunction with feeding laboratory paradigms also provide evidence for an association between negative affect and ED behavior (Telch & Agras, 1996). Finally, consistent with the Haynos and Fruzzetti model, several studies also suggest that negative affect may decrease (at least temporarily) subsequent to certain ED behaviors (Smyth et al., 2007; Engel et al., 2013), although this remains a source of debate (Haedt-Matt & Keel, 2011).

16.6 EMA Studies of Negative Affect in Eating Disorders

EMA methods have been applied in a variety of ED studies, with many studies focusing on elucidating the associations between negative affect and ED behaviors. As noted previously, EMA methods have the benefit of reducing retrospective recall biases, contrasting with standard assessments that rely on recall of ED behaviors over an extended period of time (e.g., 28 days in eating disorder examination, the gold-standard interview-based assessment of ED psychopathology; Fairburn, 2008). Additionally, the collection of momentary emotion and behavior data across multiple time points within a day, as well as across days, allows for a more precise examination of the temporal nature of the emotion-behavior association in ED. The following is a brief review of findings regarding the association between negative affective states and ED symptoms derived from studies that utilized EMA.

The majority of EMA studies in the ED literature have been conducted with samples of individuals with BN, binge eating disorder (BED), or related groups (e.g., obese individuals who binge eat). In a sample of individuals with subclinical binge eating behavior who completed a 2-week EMA protocol, Wegner and colleagues (2002) found that several indices of negative affect were worse on days during which a binge eating episode occurred versus days in which no eating binges occurred. In a study that included both women with BN and women with BED, Hilbert and Tuschen-Caffier (2007) found that both ED groups reported a more negative mood prior to binge eating than prior to regular eating. Furthermore,

pre-binge mood was worse among those with BN compared to the BED group, and both ED groups were found to display a worse mood subsequent to the binge.

In another study, a sample of women with BN completed a 2-week EMA protocol (Smyth et al., 2007). Results revealed that negative affect was higher on days in which binge eating or self-induced vomiting episodes were reported. Furthermore, a trajectory of increasing negative affect prior to binge eating and vomiting behaviors was observed, while a trajectory of decreasing negative affect subsequent to the behaviors was found. A recent study using data from this study reported on specific facets of negative affect (fear, guilt, hostility, sadness) in relation to the occurrence of binge eating and purging behaviors (Berg et al., 2013). Results revealed a similar pattern of increasing negative affect facets prior to both binge eating and purging, and the facet of guilt in particular was found to increase prior to and decrease subsequent to the behaviors, even when controlling for the other negative affect facets. Finally, Crosby and colleagues (2009) examined types of days characterized by patterns of negative affect in the same sample of women with BN. Results revealed nine distinct types of days that were characterized by different patterns of negative affect (e.g., stable high negative affect across the day, negative affect that increased late in the day, negative affect that decreased late in the day, etc.). The likelihood of binge eating and purging behaviors was found to differ between days characterized by stable low negative affect and several other types of days, providing further evidence for an association between negative affect and ED symptoms.

More recently, studies have also used EMA methods to examine associations between emotion and ED behaviors in AN. In one of the earliest such studies, Engel and colleagues (2005) reported on data collected from 10 women with AN who completed 2 weeks of EMA. Results revealed substantial variables of mood across individuals, as well as across the day within individuals. Mood variability was also found to be positively associated with restrictive and ritualistic eating behaviors. In a larger, more recent study using EMA in a sample of women with AN, Engel and colleagues (2013) examined negative affect antecedent and consequent to a variety of ED behaviors using two methods. Using a method in which multiple data points prior to and following the behaviors were used to model pre- and post-behavior trajectories of negative affect, significant increases in negative affect were observed prior to loss of control eating, purging, and weighing, while significant decreases in negative affect were found following the behaviors. However, using only a single rating immediately before and after the behaviors, results revealed a significant increase in negative affect after loss of control eating, purging, and weighing, while a significant decrease in negative affect was observed after exercise and drinking fluids to curb appetite. These disparate findings thus suggest that further research is necessary to clarify the affective changes that occur in response to ED behaviors.

Finally, in a second study using data from the same EMA study of women with AN, Lavender and colleagues (2013) examined daily patterns of anxiety in relation to the occurrence of various ED behaviors. Results revealed seven distinct types of days characterized by varying patterns of anxiety across the course of the day. Certain ED behaviors (e.g., binge eating, vomiting, dietary restriction) were found

to differ between days characterized by stable low anxiety and other types of days. Furthermore, an examination of the timing of ED behaviors within each type of day revealed that frequently, ED behaviors were more likely to occur during times of elevated anxiety. Taken together, results from the small but growing body of literature comprised of EMA studies with AN samples provide support for a similar association between emotion and ED symptoms that have been found in other ED samples.

Concluding Comments on Affect Regulation in Eating and Substance Use Disorders

We have provided a brief review of affect regulation theory and research in both SUD and ED. Clearly, there has been scientific and clinical growth in this topic in both areas of psychopathology. However, there are also differences between the studies of affect regulation in these two distinct areas of psychopathology. For example, precursors of negative affect in the substance use domain are significantly influenced by withdrawal tied to the drug of use or abuse. As Baker and colleagues (2004) point out, it is the negative affect emanating from the withdrawal experience which appears to serve as the critical antecedent for the behavioral use of the substance. On the other hand, the concepts of withdrawal in ED literature are not well understood or developed. Other than some understanding that starvation may impact affect (Keys, Brozek, Henschel, Mickelson, & Taylor, 1950), the origins of negative emotional experience in the ED seem to reside more in the interface of the individual's environment and their internal propensities and resources (e.g., personality and coping resources). Clearly, a careful consideration of the precipitants of negative affect in both ED and SUD may help to integrate and advance this field of study. For example, it is unclear how an inability to engage in ED behaviors may produce a "withdrawal-like" phenomenon in ED which is similar to SUD. Similarly, the contributions of environmental- and person-based factors in the elicitation of negative affect and its role as a precipitant of episodes of substance abuse remain unclear. Baker and colleagues (2004) acknowledge the role of modulators in their theory, but this aspect of affect generation in the model seems less well developed than negative affectivity associated with withdrawal from substances.

Another factor to consider in the comparison and contrast of SUD and ED in terms of affect regulation is the role of cognitive processes. Much research in ED has focused on cognitions surrounding eating, shape, and weight concerns. This comes out of a significant influence of cognitive-behavioral therapy in ED (Fairburn, 2008) and has resulted in several empirically supported treatments which focus on cognitive factors. An alternative approach to cognition, which is seen in the substance use literature, is the consideration of cognitive constructs, such as expectancy. Specifically, expectancies regarding the possible function of engaging in the use of substances at any given point in time have been shown to be a significant predictor of substance use episodes (Fisher, Smith, Anderson, & Flory, 2003; Fulton, Krank, & Stewart, 2012). For example, the expectation that a certain substance will improve negative mood has been shown to be a robust

predictor of engagement in utilization of that substance (Kuntsche, Knibble, Gmel, & Engels, 2005). Paralleling this literature, there has been an increased consideration of expectancies in ED behavior (Fischer et al., 2012). In particular, there is some evidence that expectations that dieting will result in thinness have a powerful impact on anorexic-like behaviors. Similarly, the expectation that binge eating will reduce negative mood has been found to be associated with bulimic-type presentations (Hohlstein, Smith, & Atlas, 1998). Clearly, in both areas of psychopathology, a consideration of the role of the specific types of cognition needs to be carefully considered to provide a truly integrative and comprehensive affect regulation model.

Finally, affect regulation models in both the ED and SUD have begun to influence treatment. For example, recent empirical work in the SUD suggests that mindfulness-based approaches to treatment (e.g., dialectical behavior therapy, DBT) reduce the use and abuse of substances by patients (Dimeff & Linehan, 2008). Similarly, broad-based cognitive-behavioral strategies which emphasize affect regulation have also been shown to have promise in treating individuals with SUD (Kadden, Carbonari, Litt, Tonigan, Zweben, 1998). Similar observations could be made about ED treatment. For example, both mindfulness-based approaches (Kristeller, Baer, & Quillian-Wolever, 2006) and DBT-oriented approaches have been found to have efficacy in ED treatment (Safer et al., 2009). However, there are also other emerging ED treatments that have evolved more directly from explicit affect regulation models of ED psychopathology. For example, emotional avoidance behavior therapy (EABT; Wildes & Marcus, 2011) and integrative cognitive-affective therapy (ICAT; Wonderlich et al., 2014) are examples of new ED treatments emerging explicitly from affect regulation theories of ED behavior. Future treatments that target factors which increase affective intensities or affect dysregulation, promote greater awareness and tolerance of emotional states, and support inhibition of impulsive or reckless behaviors when affectively aroused hold considerable promise for treatments in both SUD and ED domains.

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The Role of Stress, Trauma, and PTSD in the Etiology and Treatment of Eating Disorders, Addictions, and Substance Use Disorders

17

Timothy D. Brewerton and Kathleen Brady

Abstract

Stress, adversity, and trauma often play important roles in the etiology and treatment of substance use disorders, behavioral addictions, and eating disorders. At the outset, the term "trauma" is defined in terms of events, experiences, and effects, which often include the development of posttraumatic stress disorder (PTSD) and its symptoms. Much has been learned about the psychoneurobiology of stress response, trauma, and PTSD and associated comorbidity over the last several years. Literature illustrating how trauma and in particular PTSD predispose toward both addictions and eating disorders, particularly bulimic-spectrum disorders, is appraised. The emerging field of epigenetics and its relationship to understanding the role of trauma in the development of addictions and eating disorders is also examined. In addition, treatment implications and strategies for traumatized patients who are comorbid with PTSD and addictions and/or eating disorders are reviewed.

Keywords

Abuse • Bulimic-spectrum disorders • Case formulation • Cognitive therapy • Epigenetics • Neurobiology • Prolonged exposure • Psychopharmacology • Posttraumatic stress disorder • Trauma

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"It's not stress that kills us, it is our reaction to it."
-Hans Selye

17.1 Introduction

Data from multiple sources have converged over the past several decades to support the idea that exposure to severe adversity, especially during childhood, can place an individual at increased risk of subsequently developing psychiatric and medical disorders, including substance use disorders (SUD), eating disorders (ED), and all related comorbidity (Brady, Killeen, Brewerton, & Lucerini, 2000; Brewerton, 2004, 2007; Dansky, Brewerton, O'Neil, & Kilpatrick, 1997; Felitti et al., 1998; Kessler, 2000; Kessler, Davis, & Kendler, 1997; Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995; Wonderlich, Brewerton, Jocic, Dansky, & Abbott, 1997). This chapter will focus on the role that stress, adversity, and trauma play in the development, neurobiology, and treatment of SUD, ED, and related conditions, such as posttraumatic stress disorder (PTSD). It is important at the outset of this chapter to define "trauma," a term that is often imprecise. For our purposes, we use the working definition of trauma as recently proposed by Substance Abuse and Mental Health Services Administration (SAMHSA, 2012), which is fairly comprehensive and encompasses traumas of commission, traumas of omission, and the three "E's": Event, Experience, and Effect. These three "E's" are essential in fully understanding the effects of environment on genetic expression, which is captured in the common saying, "Genetics loads the gun, and environment pulls the trigger." As noted by SAMHSA (2012), "Individual trauma results from an event, series of events, or set of circumstances that is experienced by an individual as physically or emotionally harmful or threatening and that has lasting adverse effects on the individual's functioning and physical, social, emotional, or spiritual well-being. *Events and circumstances* may include the actual or extreme threat of physical or psychological harm or the withholding of material or relational resources essential to healthy development. These events and circumstances may occur as a single occurrence or repeatedly over time. The individual's *experience* of these events or circumstances helps to determine whether it is a traumatic event. A particular event may be experienced as traumatic for one individual and not for another." "How the individual labels, assigns meaning to, and is disrupted physically and psychologically by an event will determine whether or not it is experienced as traumatic. In many situations, a sense of humiliation, betrayal, or silencing often shapes the experience of the event. How the event is experienced may be linked to a range of factors including the individual's cultural beliefs (e.g., the subjugation of women and the experience of domestic violence), availability of social supports (e.g., whether isolated or embedded in a supportive family or community structure), or to the developmental stage of the individual (i.e., an individual may understand and experience events differently at age 5, 15, or 50)."

The long-term negative *effects* on any given person result from of that person's experience of the event or situation. These negative effects may occur right away or over a period of time. In some circumstances, the individual may not identify or comprehend the connection between the effects and the events. Negative effects may include a person's failure to manage the normal stresses and strains of everyday life, to value and profit from interpersonal relationships, and to cope with thoughts, emotions, memories, and behaviors. Together with these more observable effects, there may also be alterations in a person's neurobiology, ongoing health, and sense of well-being. Developments in neuroscience and an expanded understanding of the interaction of genetic, neurobiologic, and environmental factors have detailed the consequences of such traumatic events. Traumatic experiences can lead to hypervigilance, or a persistent state of arousal, which then often exhausts mental, emotional, and physical resources. Trauma survivors have also underscored the effect of traumatic events on spiritual beliefs and the quest for meaning. In summary, "trauma is the sum of the event, the experience, and the effect" (SAMHSA, 2012).

It is also pertinent to this discussion to point out that PTSD is no longer classified as an anxiety disorder in DSM-5 but is now under a separate and new category called "Trauma- and Stressor-Related Disorders" (American Psychiatric Association, 2013). Clinical features of PTSD have been expanded from three to four types of symptoms, which now include intrusive symptoms, avoidance symptoms, alterations in arousal and reactivity symptoms, and negative alterations in cognitions and mood associated with the traumatic event. Several published reports over the past few years have recognized the clinical importance of subthreshold or partial PTSD (pPTSD) (Brewerton, 2007; Mitchell, Mazzeo, Schlesinger, Brewerton, & Smith, 2012), which could be subsumed under the new diagnosis "Other Specified Trauma- and Stressor-Related Disorder" (309.89) or "Unspecified Trauma- and Stressor-Related Disorder" (309.9). Importantly, pPTSD has been identified as an important entity to be considered in understanding and treating both SUD and ED that are associated with trauma (Brewerton, 2007; Lipschitz et al., 2003; Mitchell, Mazzeo et al., 2012).

17.2 Trauma and Substance-Related Disorders

Although it is impossible to establish direct causal relationships, compelling findings suggest a positive relationship between exposure to early life trauma and substance-related problems later in life. It has been hypothesized that individuals may use drugs and alcohol to help mitigate trauma-related symptoms and other negative consequences of early life trauma. Individuals with a positive history of early childhood trauma and co-occurring drug or alcohol dependence have a more severe clinical profile, including poorer treatment outcomes, as compared to individuals with either early trauma or addiction alone (Greenfield et al., 2002; Hyman et al., 2008). Recent investigations highlight the importance of assessing trauma among individuals with SUD and the positive benefits associated with the

application of integrative psychosocial interventions that target both trauma-related symptoms and addictive disorder. In this chapter, the prevalence of early childhood trauma and its robust association with the development of SUD, as well as post-traumatic stress disorder (PTSD), will be reviewed. Potential biological mechanisms by which early adverse experiences can result in long-lasting changes in neurobiology, which might underlie the vulnerability for future development of PTSD and SUD, will be discussed. Potential pharmacologic and behavioral interventions that might decrease this vulnerability or be particularly effective in individuals with early life trauma will also be reviewed.

There is little doubt that severe childhood adversity may place an individual at lifelong risk for a variety of problems, including mental health, physical health, employment, and legal difficulties (Putnam, 2006). In an important study conducted by the Centers for Disease Control and Kaiser Permanente (Felitti et al., 1998; ACE study), a sample of 17,337 adults recruited from a large HMO were surveyed concerning a range of adverse events which might occur during childhood (e.g., physical or sexual abuse, incarcerated household member, emotional neglect) and adult risk behaviors, health status, and disease. The investigators found a positive relationship between the number of adverse childhood events (i.e., ACE score), risk behaviors during adulthood, and leading causes of morbidity and mortality in the USA, including heart disease, diabetes, liver disease, emphysema, and obesity. It is possible that these increased rates of adverse health consequences are not a direct result of childhood experiences, but rather the result of dysfunctional and unhealthy behaviors, including substance abuse and disordered eating, in which many victims of childhood adversity engage.

A number of studies also report that child maltreatment places an individual at enhanced risk for emotional difficulties and psychiatric disorders. One of the most consistent results across these studies is the finding that childhood maltreatment is associated with an increased risk for SUD (Enoch, 2011). In a population-based sample of 1,411 female adult twins, self-reported childhood sexual abuse was positively associated with a number of psychiatric disorders and obesity, but the strongest associations were with SUD (Kendler et al., 2000). In the ACE study, the risk of alcohol dependence increased 7.2-fold and illicit drug use increased 4.5-fold for persons with $\geq$4 ACEs (Anda et al., 2006). An investigation by Widom, White, Czaja, and Marmorstein (2007) demonstrated that the increased risk of excessive alcohol use among victims of childhood abuse or neglect is consistent and stable into middle adulthood (e.g., 40 years old). Furthermore, alcohol-dependent patients with a history of sexual abuse were more likely than non-abused patients to have a relapse to alcohol use (87.5 % vs. 63.3 %) and to relapse more quickly (median time to first drink = 60 vs. 115 days) in the one year following inpatient treatment for alcohol dependence as compared to those patients without a sexual abuse history (Greenfield et al., 2002). Of interest, Hyman and colleagues (2008) found that severity of childhood trauma was predictive of relapse and poor treatment outcome in cocaine-dependent women.

In addition to SUD, childhood adversity is associated with increased risk for the development of PTSD (Widom, 1999). Data from a number of studies over the last

20 years have emphasized the high co-occurrence of PTSD and addictive disorders. For example, among 3,768 female twins participating in the longitudinal Missouri Adolescent Female Twin Study (MOAFTS), Sartor and colleagues (2010) found that women exposed to trauma were nearly twice as likely to develop alcohol dependence (hazard ratio = 1.85) as compared to nontraumatized women, and women exposed to trauma who also had PTSD were even more likely to develop alcohol dependence (hazard ratio = 3.54, significantly higher than women with trauma exposure alone, $p < 0.01$). Studies of samples of individuals seeking treatment for SUD also find a high prevalence of reported childhood adversity and PTSD. In a study of men and women in treatment for addictions, 62 % reported having been victims of childhood physical or sexual abuse (Grice, Brady, Dustan, Malcolm, & Kilpatrick, 1995). A review of a number of studies of individuals seeking treatment for addictions reveals rates of PTSD as high as 50 % or greater (Dansky, Brady, & Roberts, 1994). In the majority of cases, the development of PTSD precedes the development of the SUD.

These high rates of childhood victimization in individuals with PTSD and alcohol- and other substance-related problems suggest that there is a link between the childhood adversity and the development of these disorders, although it is impossible to establish a direct causal relationship. However, even when studies control for demographic differences, family discord, and parental pathology, the specific relationship between childhood abuse and the development of SUD holds true. Several theoretical connections have been postulated (Miller, Downs, & Testa, 1993). Childhood victimization may lead to low self-esteem and the subsequent use of alcohol to deal with negative cognitions. It is also possible that victims of childhood abuse feel that their experiences make them "different" from other children and lead them to withdraw from healthier social circles toward "fringe" groups where alcohol use is more accepted. In any case, victims of child abuse are more likely to develop SUD as adults, so early intervention, prevention, and training in parenting are all important to interrupting this cycle of violence and alcohol problems.

Gambling disorders, like SUD, are characterized by a lack of impulse control, and gambling and SUDs commonly co-occur (Taber, McCormick, & Ramirez, 1987) (see Chap. 18). Several studies have found a high prevalence of early life trauma in individuals with gambling disorders (Kausch, Rugle, & Rowland, 2006; Petry & Steinberg, 2005; Taber et al., 1987). However, it is not clear whether this is mediated by the high prevalence of SUD in individuals with gambling disorders or if there is an independent relationship between early life traumas, PTSD, and gambling.

17.3 Trauma and Eating Disorders

A history of traumatic events, especially those involving interpersonal violence, is the rule rather than the exception in individuals with ED, particularly those characterized by bulimic symptoms, i.e., binge eating and/or purging (Brewerton,

2004, 2007; Dansky et al., 1997; Jacobi, Hayward, de Zwaan, Kraemer, & Agras, 2004; Jacobi, Morris, & de Zwaan, 2004; Johnson, Cohen, Kasen, & Brook, 2002; Mitchell, Mazzeo et al., 2012; Wonderlich et al., 2000). Such adverse events have been found to be significant causative risk factors for the development of bulimic ED. In addition, one of the most important mediators between having had traumatic experiences and the development of ED and other related psychiatric problems is the presence of PTSD or its symptoms (pPTSD) (Brewerton, 2004, 2007; Dubosc et al., 2012; Mitchell, Mazzeo et al., 2012).

In much the same way as alcohol and drugs are employed to self-medicate negative affect, binge eating and purging are also behaviors that facilitate (1) decreasing trauma-related hyperarousal or anxiety and (2) the numbing, avoidance, and even forgetting of traumatic experiences (Brewerton, 2004; Brewerton, Dansky, Kilpatrick, & O'Neil, 1999). In this way, these behaviors are reinforcing and hence perpetuated. As a result, traumatic experiences and their disabling effects are not effectively processed and therefore continue to cause problems. It is well known that PTSD tends to be a chronic condition, especially when not adequately addressed and treated (Kessler, 2000; Kessler et al., 1995, 1997).

As noted previously, not everyone who experiences trauma or adversity reacts in the same way. Available evidence suggests that individuals with ED may be particularly sensitive, reactive, or vulnerable to stress and its consequences. It is well established that the majority of individuals with anorexia nervosa (AN) and/or bulimia nervosa (BN) have a primary anxiety disorder, i.e., one that begins prior to the ED (Brewerton et al., 1995; Kaye, Bulik, Thornton, Barbarich, & Masters, 2004). Recent research indicates that individuals with ED are more likely to perceive threat or hostile intent from others, exhibit high levels of anxiety sensitivity characterized by fear or loss of control, are often overconcerned with consequences, and have exaggerated inhibition and anticipatory anxiety, high punishment sensitivity, and impaired flexibility to changing circumstances (Brewerton, 2014). As a consequence, what may seem to be of little or no consequence to one person can be very traumatic to another, particularly to one with or predisposed to an ED.

A related matter is coping style or ability, which can play an important role in whether stressful experiences become traumatic or not. Individuals with an avoidant coping style will not fare as well as those with an active, problem-solving style. Avoidant coping and emotional coping styles appear to increase the odds of developing an ED, whereas an active or task coping style does not. Avoidant coping is associated with intropunitive (self-punishing) thoughts and beliefs, which can be self-defeating and contribute to a poor prognosis, while active coping style is associated with a better prognostic outcome (McFillin et al., 2012; Pamies & Quiles, 2012; Smith-Jackson, Reel, & Thackeray, 2011). On the other hand, Leiner, Kearns, Jackson, Astin, and Rothbaum (2012) reported that prolonged exposure (PE) and EMDR were beneficial for women with PTSD following rape who frequently engaged in avoidant coping responses.

Taken together, these findings have important implications for assessment and treatment of an individual with an ED, with or without an SUD. Identifying all

comorbid or coexisting psychiatric disorders and identifying major stressful or traumatic events in the individual's life are essential to fully understanding possible predisposing, precipitating, and/or perpetuating factors in the course of the individual's struggle with an ED. Clinical experience and some research suggest that trauma and subsequent PTSD or pPTSD predict a worse prognosis. In conclusion, the trauma and resultant PTSD or its symptoms must be satisfactorily processed and abated in order to facilitate *full* recovery from the ED and its associated comorbidity.

17.4 Patients with Comorbid Substance Use Disorders and Eating Disorders

As noted in several other chapters in this volume, SUD and ED are highly comorbid. Multiple studies have reported higher rates of ED and eating pathology in substance users when compared to non-substance users (Bonfa et al., 2008; Cohen et al., 2010; Greenfield, Back, Lawson, & Brady, 2010; Peveler & Fairburn, 1990). In one study of 31 women receiving treatment in an alcohol treatment program (inpatient and outpatient), approximately 25 % were diagnosed with an ED and 33 % reported engaging in binge eating during the previous 4 weeks (Peveler & Fairburn, 1990). Likewise, in a report of 204 women receiving inpatient treatment for an SUD, the lifetime prevalence of an ED was 20 % and the presence of eating pathology predicted a poorer treatment response and a greater probability of relapse (Bonfa et al., 2008). In a review of available studies on ED-SUD comorbidity, Holderness, Brooks-Gunn, and Warren (1994) reported that a substantial number of SUD patients reported histories of BN or other purging-related disorders (1994), with a median prevalence rate of approximately 20 % (Beary, Lacey, & Merry, 1986). Research on AN or the presence of restricting behaviors in substances users is more limited, but suggests that the prevalence is much lower, with most estimates under 10 % (Beary et al., 1986; Hudson, Pope Jr, Jonas, & Yurgelun-Todd, 1983).

Most of the available research studies on the topic suggest that ED patients with bulimic symptoms, i.e., binge eating and/or purging (BN, AN-BP, BED), engage in more substance use and addictive behavior than those who primarily restrict food intake and do not engage in bulimic symptoms (AN-R) (Baker, Mitchell, Neale, & Kendler, 2010; Bulik, 1987; Bulik et al., 2004; Bulik, Sullivan, Fear, & Joyce, 1997; Castros-Fornieles et al., 2010; Holderness et al., 1994; Stock, Goldberg, Corbett, & Katzmann, 2002; Walfish, Stenmark, Sarco, Shealy, & Krone, 1992; Welch & Fairburn, 1996). Although the National Center on Addiction and Substance Abuse (2003) has estimated that about 50 % of women with ED abuse substances, studies of treatment-seeking individuals with BN suggest that approximately 20–50 % also have an SUD (Baker et al., 2010; Bulik, 1987; Bulik et al., 1997; Castros-Fornieles et al., 2010; Holderness et al., 1994; Stock et al., 2002; Walfish et al., 1992; Welch & Fairburn, 1996). Using the National Women's Study, Dansky, Brewerton, and Kilpatrick (2000) found that the lifetime prevalence of

alcohol abuse (AA) in women with BN was 31 %, compared with 18.8 % of those in the control group. Similarly, the prevalence of alcohol dependence (AD) was 13.2 % in the women with BN and 4.3 % in the control women. These differences persisted even when controlling for PTSD and major depression, which also had high rates of AA and AD.

Rorty, Yager, and Rossotto (1994) studied the relationship between childhood sexual, physical, psychological, and "multiple" abuse (more than one type of abuse) and comorbid Axis I and personality psychopathology among 80 women with lifetime BN and 40 control women. Sexual, psychological, and multiple abuse histories were associated with a higher total number of diagnosed Axis I conditions, including SUD. Their results demonstrated that childhood abuse, particularly psychological abuse and abuse in multiple forms, increased the likelihood of lifetime comorbid Axis I disorders, including SUD, and personality pathology among patients with BN. The authors noted that women with an ED and a history of child abuse may thus represent a subgroup of patients requiring especially intensive intervention.

In another study of women with BN, those with childhood sexual abuse reported earlier onset of bulimia, greater depressive symptoms, worse global functioning, and more suicide attempts and were more likely to meet criteria for bipolar II disorder, SUD, conduct disorder, and avoidant personality disorder (Sullivan, Bulik, Carter, & Joyce, 1995). In a study of 712 female ED patients, those who reported having been abused sexually and/or physically had significantly more depression and ED-related psychopathology (Fullerton, Wonderlich, & Gosnell, 1995). In addition, abused subjects were much more likely to report alcohol problems, suicide attempts, and shoplifting.

Lilenfeld and colleagues (1997) found that social phobia, conduct disorder, and clusters B and C personality disorders were significantly more prevalent among women with BN and substance dependence than among women with BN and no substance dependence or control women. Social phobia, panic disorder, SUD, and cluster B personality disorders were significantly more prevalent among the relatives of women with BN and substance dependence than the relatives of the other two groups. Bulik and coworkers (2004) studied a large group of women with ED and healthy controls and reported that alcohol use disorders (AUD) were significantly more prevalent in women with AN-BP and BN than in women with AN-R ($p = 0.0001$). The majority of individuals reported primary onset of the ED, with only one third reporting the onset of the AUD first. After ED subtype was controlled for, AUD were associated with the presence of major depressive disorder, a range of anxiety disorders, and cluster B personality disorder symptoms. Notably, individuals with AUD presented with personality profiles marked by impulsivity and perfectionism.

Women with ED may be at higher risk of developing an SUD over time. Herzog and colleagues (2006) reported that as many as 30 % of women with BN and 18 % of women with AN developed an SUD over a 9-year period. This is compatible with the notion that an ED or the presence of disordered eating behaviors may be a risk factor for subsequent SUD (Krahn, Kurth, Demitrack, & Drewnowski, 1992;

Striegel-Moore & Huydic, 1993; Strober, Freeman, Bower, & Rigali, 1996). On the other hand, it has been claimed that the chronology of symptoms or disorders does not matter since the rates of women who report developing their ED first are about equal to the rates of women who report developing their SUD first (Baker, Mazzeo, & Kendler, 2007; Wiseman et al., 1999).

In the National Comorbidity Survey Replication (Hudson, Hiripi, Pope, & Kessler, 2007), lifetime rates of any SUD in the various ED subgroups were as follows: AN, 27.0 %; BN, 36.8 %; BED, 23.3 %; subclinical BED, 35.5 %; and any binge eating, 28.7 %. Using this same representative sample, Brewerton and Mitchell (2012) found that the rates of PTSD and pPTSD were highest in individuals comorbid for both any bulimic ED and any SUD when compared to either disorder alone or no disorder. This pattern was especially pronounced in men.

Deep, Lilenfeld, Plotnicov, Pollice, and Kaye (1999) found significantly higher rates of prior sexual abuse in women with BN and comorbid substance dependence (67 %) compared to women with BN and no substance dependence (37 %), women with AN (23 %), and control women (7 %). In a recent study of 192 treatment-seeking women with alcohol dependence and 177 healthy controls, Copeland, Magnusson, Goransson, and Heilig (2011) found that the association of sexual abuse with alcohol dependence was limited to the most severe category of sexual abuse involving anal or vaginal penetration. Of the five psychiatric disorders tested, anxiety, AN, and BN met criteria as potential mediators of the abuse-alcohol dependence association. Severe sexual abuse continued to have an independent effect on alcohol dependence status even after accounting for these potential mediators. Of alcohol-dependent participants, those with a history of severe abuse rated higher on alcoholism severity and psychiatric comorbidities.

In a study of 89 women with bulimic-spectrum disorders, Richardson and coresearchers (2008) demonstrated two classes of patients: one with low comorbidity, characterized by high rates of major depressive disorder, and another with high comorbidity, characterized by high rates of major depressive disorder, anxiety disorder, and SUD. The high-comorbidity class displayed significantly higher dieting preoccupations and conduct problems and showed a greater likelihood of childhood abuse and of carrying the 5-HTTLPR S allele than did the low-comorbidity class. These results are consistent with previous findings identifying a subgroup of individuals with bulimia typified by high psychiatric comorbidity and suggest that childhood trauma and the 5-HTTLPR polymorphism may both be relevant to explaining the presence of SUD and anxiety disorders in bulimic-spectrum disorders. A genetic polymorphism is the occurrence together in the same population of more than one allele or genetic marker at the same locus.

Corstorphine, Waller, Lawson, and Ganis (2007) addressed relationships between histories of childhood trauma and multi-impulsivity in 102 individuals with a strictly defined ED. Multiple impulsive behaviors, including substance abuse, self-harming behaviors, stealing, and sexual promiscuity, are common in the ED, especially in those with bulimic symptoms, and multi-impulsive patients appear to do worse in treatment (Lacey, 1993; Lacey & Evans, 1986). They found that any reported history of childhood trauma was associated with a higher number

of impulsive behaviors and with the presence of multi-impulsivity. Childhood sexual abuse was particularly notable, as it was associated with self-cutting, alcohol abuse, and substance abuse, including abuse of amphetamines, cocaine, cannabis, and "other substances," including ketamine and benzodiazepines.

Baker and colleagues (2007) investigated possible influences on the comorbidity between BN and drug use disorders (DUD) in 490 monozygotic and 354 dizygotic female twins and 930 females from opposite sex pairs. Using multiple logistic regression analyses, they found that depression, neuroticism, and childhood sexual abuse (CSA) mediated the association between BN and DUD regardless of which disorder was used as the dependent variable. Results also indicated genetic and nonshared environmental overlap between BN and DUD. The association between BN and DUD is due mostly to overlapping genetic influences with a smaller contribution from nonshared environment. Depression, neuroticism, and CSA are likely important shared correlates.

Matsunaga and associates (1999) sought to clarify the influence of a history of sexual or physical abuse on a variety of psychopathologies in subjects with BN who had been recovered for at least 1 year. Abusive experiences appeared to be associated with some psychopathology of BN, particularly related to PTSD, substance abuse, and more severe core ED pathology.

17.5 Neurobiologic Aspects of Trauma, Substance Use Disorders, Addictions, and Eating Disorders

The recognition of the pervasive and detrimental impact of adverse childhood experiences on quality of life and health outcomes has led to the exploration of potential biological mechanisms by which early experiences can produce long-lasting changes. Evidence from both animal and human research suggests that early stressors can lead to changes in the neurobiologic systems known to be involved in the pathophysiology of depression, anxiety, impulsivity, ED, and addictions (Brewerton, 1995; De Bellis et al., 1999; Heim & Nemeroff, 2001; Steiger, Gauvin, et al., 2001; Steiger, Koerner, et al., 2001; Steiger, Young, et al., 2001) (see Chap. 6). The hypothalamic-pituitary-adrenal (HPA) axis plays a critical role in the stress response and is involved in the pathophysiology of ED and addictive disorders. Early stressors cause long-term increases in the cortisol response to stress (Plotsky & Meaney, 1993) and decreased genetic expression of cortisol receptors and increased expression of corticotrophin-releasing factor in the hypothalamus, both of which may contribute to dysregulation of the HPA axis (Ladd, Owens, & Nemeroff, 1996; Liu et al., 1997). The noradrenergic system also plays a key role in stress (Bremner, 2003) and early stressors can lead to long-term decreases in alpha-2 noradrenergic receptors in the locus coeruleus, which may lead to loss of feedback inhibition of noradrenergic activity with associated increases in the noradrenergic stress responses (Caldji et al., 1998; Sanchez, Ladd, & Plotsky, 2001). This increased noradrenergic activity in response to stress may be one of the

neurobiologic underpinnings of the vulnerability to the development of PTSD, SUDs, and other long-term consequences of early life adversity.

In addition to the long-lasting impact of early trauma on the stress response, a number of studies indicate that early trauma has specific effects on the neurotransmitter systems involved in the positive reinforcing effects of alcohol and drugs, food, exercise, and sex, particularly the mesocorticolimbic dopamine system (Meaney, Brake, & Gratton, 2002; Olsen, 2011). Higley, Hasert, Suomi, and Linnoila (1991) found that adult rhesus monkeys raised in peer groups without maternal care showed increased HPA response to stress and increased alcohol consumption during periods of stress (Higley et al., 1991). In a series of studies, Meaney and colleagues (2002) demonstrated that repeated periods of maternal separation in the early life of rats decreased dopamine transporter expression and increased dopamine and behavioral responses to stress and to the administration of cocaine and amphetamines. Using similar paradigms, other investigators have reported alterations in feeding behavior and food preferences in maternally deprived animals (Mela et al., 2012; Penke, Fernette, Nyakas, Max, & Burlet, 2012). These findings suggest that early life experiences can impact the development of the mesocorticolimbic dopamine system and lead to a vulnerability to addiction and related disorders, including ED and obesity, in later life. Thus, in addition to effects on stress reactivity, early life events might predispose individuals to the development of SUD and/or ED by directly influencing the reinforcing effects of substances of abuse, including certain foods (see Chap. 2). Other neurotransmitter systems involved in the pathophysiology of SUD and ED, such as BDNF, serotonin, and GABA systems, are also impacted by early life trauma in ways that may influence vulnerability to the development of SUD and ED, and the mechanistic connections in these systems are under active investigation (Enoch, 2011). A recent study focused on genetic polymorphisms encoding for GABA-A in alcohol-, cocaine-, and heroin-dependent individuals and controls with and without a history of childhood abuse suggests that variants of the GABA-A receptor play a role in risk and resilience for the development of addictions in individuals with childhood abuse (Enoch et al., 2010).

Serotonergic brain systems are also specifically involved in modulating adaptive responses to aversive events (Corchs, Nutt, Hood, & Bernik, 2009; Graeff, Guimaraes, De Andrade, & Deakin, 1996; Krystal & Neumeister, 2009). It is plausible, based on available evidence that early traumatic events interact in complex neurodevelopmental ways to produce long-term changes in affected brain systems, including the HPA axis, neuropeptides, and the monoamine neurotransmitters, including serotonin. There is evidence for an association between a history of sexual abuse, impulsivity, self-destructiveness, and reduced serotonin function in at least a subset of ED patients (Steiger, Gauvin et al., 2001; Steiger, Koerner et al., 2001; Steiger, Young et al., 2001).

Not all children exposed to early life trauma develop SUD, ED, or other significant pathologies, clearly suggesting that resilience and other mediating factors play a role (Brewerton, 2004, 2007; Enoch, 2011). The genetic risk for SUD and ED involves multiple genes (see Chap. 5). There is emerging evidence to

suggest that variation in some of the genes involved in coding for systems involved in the stress response may determine the risk for psychopathology or resilience in individuals exposed to early life trauma. In particular, there are important variations in the genes encoding corticotrophin-releasing hormone (CRH) that influence the development of substance dependence following an early life trauma in a gene by environment interaction. One study of children at risk found an interaction between CRHR1 SNP variation and sexual trauma in adolescents that predicted an earlier age of onset of drinking and heavy alcohol consumption in the subgroup exposed to trauma (Blomeyer et al., 2008). In the group without trauma exposure, drinking behavior was similar in the group with either polymorphism. This finding is supported by animal studies demonstrating that the CRHR1 genotype and expression interact with environmental stress to reinstate alcohol-seeking in rodents (Hansson et al., 2006), and a functional CRH promoter variant in monkeys confers increased stress reactivity and is associated with increased alcohol consumption in animals reared under stressful conditions (Barr et al., 2009).

Steiger and coinvestigators (2011) documented other gene-environment interactions between the glucocorticoid receptor polymorphism, Bcl1, and childhood maltreatment in women with BN. Compared to normal control women without BN, women with BN were significantly more likely (1) to endorse histories of childhood maltreatment, (2) to have the low-function Bcl1 C allele (CG or CC genotypes), and (3) to have both factors. They interpreted their results as suggesting that traumatic stress, when impacting persons predisposed to lower glucocorticoid receptor modulation, can be etiological for BN. In addition, this same group of investigators (Steiger et al., 2012) has demonstrated that the Bcl1 x child abuse interaction is decreased when levels of depression are accounted for, but is not affected by controlling for affective instability, sensation seeking, or motoric impulsivity. In summary, their findings suggest that stress-induced alterations in glucocorticoid sensitivity contribute to both BN and depressive disturbances but not the behavioral or affective dysregulation seen in many individuals with BN.

These findings suggest that the interaction of genetic susceptibility and environmental exposure can lead to a pathologically activated CRH system which increases the risk for the development of substance dependence and/or ED in some individuals. Individuals with bulimic-spectrum disorders have been shown to exhibit heterogeneous profiles of comorbid psychiatric disorders that are not only in part due to varying degrees of environmental vulnerability but also genetic vulnerability (see Chap. 5). Some interesting research has emerged over the last several years that has explored the possible interaction of two major forces that could affect the manifestation and severity of binge eating: (1) polymorphisms of specific neurotransmitter system genes, such as the serotonin transporter promoter polymorphism (5-HTTLPR), and (2) selected forms of childhood abuse.

Steiger and coworkers (2008) reported that bulimic 5HTTLPR S-allele carriers with histories of prior childhood sexual or physical maltreatment show elevations on personality measures of dissocial behavior, a trait which is overrepresented in those with bulimic-spectrum disorders. These results replicated previous observations concerning phenomenological correlates of traumatic stress in

5HTTLPR S-allele carriers. Richardson and colleagues (2008) found that women with DSM-IV bulimic-spectrum disorders who had high degrees of comorbidity, including SUD, major depression, anxiety disorder, conduct problems, and dieting preoccupations, displayed a greater probability of carrying the 5-HTTLPR S allele and of childhood abuse than did the group of bulimic women with low degrees of comorbidity. These findings suggest that the 5-HTTLPR polymorphism and prior trauma may both be pertinent to explaining the occurrence of greater psychiatric comorbidity in bulimic disorders.

Groleau and colleagues (2012) investigated the interactional effects involving numerous dopamine system gene polymorphisms, including that of the dopamine transporter (DAT1), the dopamine-2 receptor (DRD2), and catechol-o-methyltransferase (COMT) in women with bulimic-spectrum disorders. Sensation seeking was elevated in carriers of the low-function allele of the DRD2 Taq1A polymorphism who also reported CSA, relative to that in individuals showing other combinations of alleles and abuse exposures. This is relevant in that both SUD and bulimic ED patients can have high levels of sensation-seeking or novelty-seeking trait. In addition, the investigators reported that carriers of a low-function allele of COMT scored higher on compulsivity, lower on impulsivity, and marginally lower on frequency of binge eating than did individuals in whom the allele was absent. These findings indicate that genes acting within the dopamine system may contribute, either directly or indirectly (i.e., in interaction with traumatic childhood experiences), to variations in the presentation of comorbid traits and, possibly, bulimic symptoms.

17.6 Trauma and Epigenetics

Epigenetics is the study of inherited changes in gene expression or cellular phenotype caused by mechanisms other than changes in the underlying DNA sequence. It refers to functionally significant modifications to the genome without any changes in the actual ordering of nucleotides. Epigenetic changes typically involve DNA methylation and histone modification, both of which control gene expression without altering the underlying DNA sequencing or structure. Epigenetics heralds a major paradigm shift in understanding the complex relationships between genes and environment, and this certainly includes SUD and ED (Nielsen, Utrankar, Reyes, Simons, & Kosten, 2012; Pjetri, Schmidt, Kas, & Campbell, 2012; Robison & Nestler, 2011; Wong, Mill, & Fernandes, 2011). Epigenetic mechanisms can enable the effects of parents' experiences (including trauma) and behaviors (including dietary practices, substance use, and addictive behavior) to be potentially passed down to subsequent generations. Such environmentally induced changes may remain through cell divisions for the rest of the cell's life and may persist for multiple generations. Thus, nongenetic factors may cause the organism's genes to behave (or "express themselves") differently. Therefore, the effects of traumatic experiences can be passed down to offspring and subsequent generations. This

gives new meaning to the Biblical phrase, "The sins of the father will be visited upon the sons."

Perroud and colleagues (2011) reported increased methylation of the glucocorticoid receptor gene (NR3C1) in adults with a history of childhood maltreatment. Steiger, Labonte, Groleau, Turecki, and Israel (2013) compared the levels of methylation of the glucocorticoid receptor (GR) gene (NR3C1) promoter between women with BN and women with no ED and also explored, in women with BN, the extent to which methylation of the GR gene promoter corresponded to childhood abuse, suicidality, or borderline personality disorder (BPD). Compared to non-ED women, women with BN and comorbid BPD (or BN with a history of suicidality) showed significantly more methylation of specific exon 1C sites. There was also greater methylation in some 1C sites among women with BN when compared to women with no ED, but no parallel effects owing to childhood abuse were observed. These findings link BN (when accompanied by BPD or suicidality) with hypermethylation of certain GR exon 1C promoter sites.

Mehta and associates (2013) studied the impact of different early environments on disease-related genome-wide gene expression and DNA methylation in peripheral blood cells in patients with PTSD with and without early childhood abuse. Compared with the same trauma-exposed controls ($n = 108$), gene-expression profiles of PTSD patients with similar clinical symptoms and matched adult trauma exposure but different childhood adverse events ($n = 32$ and 29) were almost completely nonoverlapping (98 %). Gene-expression changes were accompanied and likely mediated by changes in DNA methylation in the same loci to a much larger proportion in the childhood abuse (69 %) vs. the non-child abuse-only group (34 %). This study is unique in providing genome-wide evidence of distinct biological modifications in PTSD in the presence or absence of exposure to childhood abuse. The findings that nonoverlapping biological pathways seem to be affected in the two PTSD groups and that changes in DNA methylation appear to have a much greater impact in the childhood-abuse group might reflect differences in the pathophysiology of PTSD, independent of exposure to childhood maltreatment. These results contribute to a better understanding of the extent of influence of differences in trauma exposure on pathophysiological processes in stress-related psychiatric disorders.

17.7 Treatment Aspects

Both behavioral and pharmacologic interventions are important to consider in the treatment of co-occurring SUD, ED, and trauma/PTSD (Davis, Barad, Otto, & Southwick, 2006; Weiss & Kueppenbender, 2006). To date, most empirical studies of behavioral or pharmacologic treatments have investigated the treatment of either ED, SUD, or PTSD alone. As such, in spite of the frequency of comorbidity and the negative consequences in terms of course of illness, the treatment of co-occurring PTSD with ED and/or SUD is understudied.

17.7.1 Psychosocial Interventions

Cognitive behavioral therapies (CBT) are the most widely studied and empirically valid psychosocial treatments for PTSD, SUD, and ED. Various adaptations of CBT are used to treat PTSD and fall into three main categories: (1) exposure-based therapy, (2) cognitive-focused therapy, and (3) anxiety/stress management therapy. Exposure-based therapies are considered the "gold standard" treatment for PTSD (Institute of Medicine, Committee on Treatment of Posttraumatic Stress, 2008) and involve having patients confront (1) safe but anxiety-provoking situations (i.e., physical location where childhood abuse occurred), known as in vivo exposure, and (2) the memory of the traumatic experience, known as imaginal exposure (Foa, Chrestman, & Riggs, 2006). With prolonged, repeated in vivo and imaginal exposure, the trauma-related anxiety is extinguished. Cognition-focused therapy includes cognitive therapy (CT), which addresses the meaning that people assign to early life trauma, and cognitive processing therapy (CPT), which combines a narrative element of exposure therapy with efforts to identify and modify unhelpful cognitions related to themes of safety, trust, power, esteem, and intimacy (Resick & Schnicke, 1992). Eye Movement Desensitization Reprocessing (EMDR) is another evidence-based approach shown to be effective for PTSD in randomized controlled trials, which includes strong elements of cognitive processing (Powers, Halpern, Ferenschak, Gillihan, & Foa, 2010). EMDR has been shown to be as effective as CBT with prolonged exposure (Rothbaum, Astin, & Marsteller, 2005) as well as fluoxetine (van der Kolk et al., 2007).

Finally, stress inoculation training (SIT) (Meichenbaum & Novaco, 1985), one of the most widely used and empirically investigated forms of anxiety management therapies, aims to provide a sense of mastery over PTSD symptoms by teaching individuals a variety of coping skills. SIT has also been incorporated into CBT for SUD and includes relaxation training, breathing retraining, thought-stopping, self-instruction training, assertiveness training, cognitive restructuring, anger management, and problem-solving.

Recently, integrative psychosocial interventions have been developed to address both trauma/PTSD and SUD simultaneously (Back, 2010; Foa et al., 2013; Mills et al., 2012). It was previously believed that trauma work was inappropriate until after a patient had been abstinent from alcohol or drugs for a sustained period of time (e.g., 3 months). This model, known as the "sequential" model, posits that continued substance use impedes therapeutic efforts to address and process the trauma and that if trauma work is begun before an individual has achieved sustained abstinence, there will be an increased risk of relapse. However, studies conducted by several investigators in the USA and Australia show that substance-dependent patients who engage in integrative CBT interventions typically experience significant improvements in both conditions, and rates of relapse are not increased by the introduction of trauma work (Brady, Dansky, Back, Foa, & Carroll, 2001; Hien, Cohen, Miele, Litt, & Capstick, 2004; McGovern et al., 2009; Najavits, 2002; Triffleman, Carroll, & Kellogg, 1999). Proponents of integrative treatments posit that unprocessed trauma-related memories and PTSD symptoms may, at least in

part, drive substance use. Thus, attending to and treating the trauma-related symptoms early in the process of therapy may improve the chances of long-term recovery (Back, Brady, Sonne, & Verduin, 2006; Hien et al., 2010). While more randomized, controlled trials of integrative treatments are needed, the studies to date clearly demonstrate that for the majority of substance-dependent patients with trauma/PTSD, the inclusion of trauma work can confer substantial therapeutic benefits (Foa et al., 2013; Mills et al., 2012).

The extent to which this type of integrated approach is appropriate for ED patients with trauma/PTSD and comorbid ED-SUD patients with trauma/PTSD remains unexplored. However, Brewerton (2004) noted the importance of integrated treatment in which the functional connection between trauma history/PTSD and subsequent ED is identified and processed cognitively (e.g., bingeing and purging serve to avoid and numb).

Persons has described an evidence-based case formulation strategy (Persons, 2005; Persons & Tompkins, 2007), which enables the organization of complex clinical material into a theoretically comprehensible scheme that then directs logical prioritization of evidence-based treatment methods, including those used in the treatment of PTSD (Zayfert & Becker, 2007). At the outset, the clinician identifies predisposing, precipitating, and maintaining factors, which is followed by the generation of hypotheses about how comorbid problems are interrelated. These individualized hypotheses are tested using ongoing feedback from the patient, which is used to guide treatment and which utilizes established empirical findings as much as possible. (See Chap. 25 for further information about this approach to treatment.)

In a study by Mitchell, Wells, Mendes, and Resick (2012), 65 women with PTSD following a rape participated in a randomized study of cognitive processing therapy (CPT) with and without a written trauma narrative. Although the patients were not identified as having ED, they completed the Eating Disorder Inventory-2 (EDI-2) before and after treatment. Decreases in PTSD symptom scores were significantly associated with reductions in the following EDI-2 subscales: impulse regulation, interoceptive awareness, interpersonal distrust, ineffectiveness, and maturity fears. Notably, there was no worsening of ED symptoms. Therefore, given that there are no clear treatment guidelines for comorbid ED and PTSD, an initial focus on PTSD symptoms using CPT may positively impact symptoms in traumatized ED patients. The authors observed that further therapy may be required for certain ED symptoms.

17.7.2 Pharmacologic Interventions

There are several general issues to consider when treating co-occurring substance dependence, ED, and trauma/PTSD. It is important to bear in mind that relapse for all of these disorders is common, and consideration of potential toxic interactions that may occur between the prescribed medication, substances of abuse, and ED behaviors is critical. The pharmacologic agent with the least abuse liability

potential should be chosen for this population. Although benzodiazepines are effective in providing immediate relief of anxiety symptoms, they are generally not considered a "first line" of treatment for patients with substance dependence or ED given their abuse potential. During the initial phase of treatment when latency of onset of antidepressants is an issue, benzodiazepines may be considered as adjunctive medication. The amount of benzodiazepines prescribed to the patient should be limited and the patient should be closely monitored for relapse or nonmedical use of benzodiazepines or other medications.

The use of pharmacologic agents specifically targeting substance dependence and PTSD or ED and PTSD is under-explored. There are FDA-approved medications for the treatment of alcohol, nicotine, and opiate dependence, as well as BN (but not AN or BED). Most studies to date have focused on alcohol dependence and suggest that patients with co-occurring alcohol dependence and trauma/PTSD respond well to standard PTSD pharmacotherapies. Sertraline, a serotonin-specific reuptake inhibitor (SSRI) which is approved for the treatment of PTSD, was examined in a 12-week, placebo-controlled trial in 94 individuals with alcohol dependence and PTSD (Brady et al., 2005). The primary outcome analysis indicated no significant impact of sertraline on alcohol-related outcomes and trend level findings for a positive effect on PTSD outcomes. Statistical trends for greater improvement in intrusion and hyperarousal PTSD symptoms were observed in the sertraline-treated group. Follow-up cluster analyses suggested that individuals with primary PTSD, as compared to primary alcohol dependence, derived more benefit from sertraline treatment as evidenced by significantly less severe alcohol use. There was some suggestion that individuals with early-onset alcohol dependence actually had worse alcohol-related outcomes with sertraline treatment as compared to placebo (Brady et al., 2005).

In another study of 254 veterans with alcohol dependence and a variety of co-occurring mood and anxiety disorders (Petrakis et al., 2005), naltrexone, disulfiram, or the combination was added to treatment-as-usual. A high percentage (42.9 %) of these subjects had PTSD, although data analysis for specific disorders was not conducted. There was a significant improvement in alcohol-related outcomes for individuals treated either with medication alone or with combination therapy as compared to placebo, but no added improvement with combination therapy as compared to mo. In a recent trial exploring naltrexone (100 mg) plus prolonged exposure-based CBT in individuals with PTSD and alcohol dependence (Foa et al., 2013), participants in all four groups demonstrated reductions in drinking days, but these reductions were significantly greater in those who received naltrexone with or without prolonged exposure. These studies strongly suggest that medications targeting alcohol consumption should be prescribed for alcohol-dependent individuals with co-occurring PTSD. Trials of treatments targeting opiate and nicotine dependence in individuals with co-occurring PTSD are needed.

There is good rationale for the exploration of a number of other compounds in the treatment of co-occurring PTSD with substance dependence and/or ED. Prazosin is a specific α_1 adrenergic receptor antagonist that has shown promise in several well-controlled trials for the treatment of PTSD, particularly in

decreasing PTSD-related sleep disturbance and nightmares (Raskind et al., 2007). In a preliminary study, prazosin decreased alcohol consumption in an alcohol-dependent population (Simpson et al., 2009). This inexpensive and relatively safe drug warrants investigation in the treatment of co-occurring PTSD and substance dependence. In addition, several anticonvulsant agents, such as topiramate, have shown promise in the treatment of alcohol and cocaine dependence (Johnson et al., 2003). Notably, topiramate has been reported to be effective in the treatment of BN, BED, and obesity (Kramer et al., 2011; McElroy et al., 2007; McElroy, Guerdjikova, Mori, & O'Melia, 2012). It is hypothesized that actions on the glutamatergic systems might be responsible for their therapeutic actions. PTSD has also been associated with glutamatergic dysregulation and topiramate has shown promise in one RCT (Yeh et al., 2011). More research is clearly needed to help advance the behavioral and pharmacologic treatment of co-occurring trauma/PTSD and SUD/ED.

17.8 Summary

Epidemiologic studies as well as studies in treatment-seeking populations converge to support the finding that early life trauma is common in individuals with substance dependence as well as those with bulimic ED. This is particularly true for those with comorbid SUD and ED. There are a number of potential mechanistic explanations for the connection between early life trauma and the development of ED and SUD. These include psychological and developmental issues that are impacted by trauma as well as neurobiologic effects of early trauma that can lead to increased vulnerability to the development of alcohol, other SUD, and/or ED. These explanatory hypotheses are not mutually exclusive. There is a growing literature on efficacious psychotherapeutic and pharmacotherapeutic treatments for individuals with co-occurring PTSD and substance dependence, but much work remains to be done for traumatized individuals with an ED. Integrative psychosocial interventions combining efficacious interventions from the addiction and PTSD fields have shown promise. Evidence suggests that using agents targeting alcohol consumption (disulfiram, naltrexone) can be useful in individuals with co-occurring PTSD and alcohol dependence. Further investigation in this area as well as investigation of drugs that are used in the treatment of other addictions is clearly needed.

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Relationship of Behavioral Addictions to Eating Disorders and Substance Use Disorders

18

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Abstract

There is growing evidence for the similarity between eating disorders and substance use disorders with respect to etiology, neurobiology, clinical presentation, and effective treatments. In addition, they each have features commonly seen in impulse control disorders, which are also frequently characterized as behavioral addictions. Therefore, it is not surprising that eating disorders and substance use disorders frequently are comorbid not only with each other but with pathological gambling and/or other behavioral addictions. This chapter will examine what is known about the clinical presentation, epidemiology, etiology, and treatment of behavioral addictions commonly comorbid with eating disorders and substance use disorders. Identifying and successfully managing these comorbid behavioral addictions are essential in order to achieve positive outcomes in individuals suffering from eating disorders and substance use disorders due to the increased illness severity and poor treatment response associated with this comorbidity.

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18.1 Introduction

As has been well documented throughout this book, eating disorders (ED) and substance use disorders (SUD) are frequently comorbid and share many similarities with respect to clinical presentation, neurobiology, and psychosocial risk factors (see Chaps. 11 and 12). Each has features seen in individuals with impulse control disorders (ICD) often referred to as "behavioral addictions" (BA) and process addictions. Given the growing evidence that SUD and at least some behavioral addictions, such as disordered gambling, may be more similar than different (Karim & Chaudhri, 2012; Leeman & Potenza, 2012), it should be no surprise that high rates of ICD are seen in individuals with both ED and SUD. For clinicians, knowing the clinical manifestations and treatments of ICD is essential because the severity and clinical course of ED and SUD are much worse when both disorders are comorbid with BA (Fernández-Aranda, Pinheiro, et al., 2008). In addition, ICD can be chronic, relapsing conditions with high rates of morbidity and mortality if not treated adequately (Grant, Levine, Kim, & Potenza, 2005; Grant, Potenza, Weinstein, & Gorelick, 2010).

In this chapter, the terms ICD and BA will be employed since both are used by mental health clinicians, experts, the literature we cite, and in the Diagnostic and Statistical Manual of Mental Disorders (DSM-5: American Psychiatric Association, 2013) itself to characterize the disorders being reviewed in this chapter (Karim & Chaudhri, 2012). In addition, a growing body of scientific evidence supports considering these terms as similar and overlapping because individuals suffering from these disorders have been shown to exhibit clinically, in neurotransmitter systems (American Psychiatric Association, 2013, p. 478), and on neuropsychological measures both impulsivity and the compulsive behavior characteristic of addictions.

We will first give an overview of the concept of BA and then describe some changes to them in DSM-5. After discussing the epidemiology of BA in the general population and among individuals with psychiatric illness, we will discuss some of the BA most commonly seen in individuals with ED and SUD. We initially examine one syndrome of clinical relevance in ED and for which there is good data on diagnostic validity and/or treatment: kleptomania (Schreiber, Odlaug, & Grant, 2011). Another BA that is frequently observed in ED patients is compulsive buying disorder, which is discussed in detail in Chap. 19. We then focus on two BA frequently seen in and with important implications for treatment of ED but for which there is much more controversy regarding their status as disorders and, as a result, for which there are virtually no evidence-based treatments: hypersexual

behavior or sexual promiscuity (Fichter, Quadflieg, & Rief, 1994), and Internet addiction (Weintraub, Dunn, Yager, & Taintor, 2011). We then review gambling disorder, a DSM-5 disorder which is highly comorbid with and very similar to SUD (American Psychiatric Association, 2013). We go on to summarize how BA impact the presentation and clinical course of ED and SUD. Intermittent explosive disorder, which will not be as thoroughly reviewed, is also commonly seen in SUD (American Psychiatric Association, 2013, p. 469). We then discuss how treatments of individuals with comorbid ED, SUD, and ICD need to be tailored to address the severe psychopathology seen in these patients. We conclude with future directions in research and treatment for this population.

Although most of the BA to be reviewed are not DSM-5 disorders, they are of tremendous clinical significance in individuals with ED and/or SUD and have the same type of clinical features seen in individuals with DSM-5 BA. They have also been shown to be very prevalent in other medical disorders such as Parkinson's disease (Weintraub, Koester, et al., 2010; Weintraub, Siderowf, et al., 2006), and a growing body of research has shown that they may involve the same type of dysregulation in brain dopamine circuits underlying all addictions. As an example, in Parkinson's disease, treatment with dopamine agonists has been associated with the development of sexual addiction, compulsive buying, and compulsive gambling, all of which are quite common in ED and/or SUD (Weintraub, Siderowf, et al., 2006). Because of this new knowledge gained from the recent increased interest and research in BA, it will likely be possible in the future to offer more evidence-based statements regarding their diagnostic validity, epidemiology, etiology, and treatment as they pertain to the management of ED and SUD.

18.2 What Are Behavioral Addictions?

A major change in the conceptualization of mental disorders has been ushered in with the release of DSM-5, in which gambling disorder (GD) (formally known as pathological gambling) has been placed into the Substance-Related and Addictive Disorders section (American Psychiatric Association, 2013). This new classification represents the formal validation of the view, long held by many experts in the field and now supported by considerable research data, that compulsive behavior not involving exogenous psychoactive substances can be characterized as an addiction (Clark & Limbrick-Oldfield, 2013). A major implication of including this BA in the same diagnostic category as substance-related disorders is that similar approaches to assessment and treatment may be clinically indicated for both GD and SUD (Fong, Reid, & Parhami, 2012; Goodman, 2008; Karim & Chaudhri, 2012; Potenza, Sofuoglu, Carroll, & Rounsaville, 2011). In this context, a unifying feature of BA is the repeated engagement of a particular behavior that once produced a pleasurable feeling but that has become compulsively irresistible and disrupts social, educational, or vocational functioning (Grant et al., 2010). In most respects, such patterns of behavior are quite similar to those seen in individuals with SUD.

Coincident with this change in DSM-5 are the creation of a peer-reviewed arena for the publication of research in this domain, the *Journal of Behavioral Addictions* (Demetrovics & Griffiths, 2012), and the redefining of the term "addiction" by the American Society of Addiction Medicine to include non-substance addictions (Smith, 2012). Other major shifts and changes in the classification of a number of disorders in which impulsivity is a prominent feature are described and discussed below.

18.2.1 Clinical Characteristics of Behavioral Addictions

Poor impulse control occurs in many psychiatric disorders not classified as ICD, including some subtypes of ED and most SUD. Moreover, impulsivity is often an explicit diagnostic criterion in these disorders or is included in the syndromal name as in the hyperactive-impulsive subtype of attention-deficit/hyperactivity disorder (ADHD). Most pointedly, the DSM-IV-TR section on ICD is called "Impulse-Control Disorders Not Elsewhere Classified." The introduction to that section states "This section includes disorders of impulse control that are not presented as part of the presentation of disorders in other sections of the manual (e.g., Substance-Related Disorders, Paraphilias, Antisocial Personality Disorder, Conduct Disorder, Schizophrenia, and Mood Disorders may have features the involve problems of impulse control)" (American Psychiatric Association, 2000, p. 663). With regard to personality disorders, in both DSM-IV-TR and DSM-5, one of the criteria for borderline personality disorder is "impulsivity in at least two areas that are potentially self-damaging (e.g., spending, sex, substance abuse, reckless driving, binge eating)." Similarly, one of the criteria for antisocial personality is "impulsivity or failure to plan ahead."

There is clearly a continuum of impulsivity in psychiatric disorders. In individuals with disordered eating, for example, impulsivity is much greater in those meeting criteria for disorders involving binging and/or purging (i.e., anorexia nervosa binge-purge subtype [ANBP], bulimia nervosa [BN], and binge eating disorder [BED]) than for the restrictor subtype of anorexia nervosa (ANR) (Brewerton, Hand, & Bishop, 1993; Cassin & von Ranson, 2005; Fernández-Aranda, Pinheiro, et al., 2008).

The ICD or BA can be conceptualized as disorders in which impulsivity is the predominant psychopathology in affected individuals. As defined in the DSM-IV-TR, "The essential feature of Impulse-Control Disorders is the failure to resist an impulse, drive, or temptation to perform an act that is harmful to the person or others." In addition, "the individual feels an increasing sense of tension or arousal before committing the act and then experiences pleasure, gratification, or relief at the time of committing the act" (American Psychiatric Association, 2000, p. 663).

18.2.2 Changes in DSM-5

In the DSM-5, a new section is introduced, "Disruptive, Impulse-Control, and Conduct Disorders" (American Psychiatric Association, 2013). Although individuals suffering from them all exhibit deficits in controlling their emotions and behavior, what distinguishes these from other DSM-5 disorders in which there are problems with impulsivity is that "they are manifested in behaviors that violate the rights of others (e.g., aggression, destruction of property) and/or bring the individual into significant conflict with societal norms or authority figures" (American Psychiatric Association, 2013, p. 461). They include most of the disorders in the DSM-IV-TR category "Impulse Control Disorders Not Elsewhere Classified (NOS)": intermittent explosive disorder, kleptomania, and pyromania. Other disorders in this new category include antisocial personality disorder and the following three DSM-IV-TR childhood disorders: oppositional defiant disorder, conduct disorder, and disruptive behavior (NOS). The other two disorders from the DSM-IV-TR impulse control NOS section have been moved elsewhere, the new DSM-5 gambling disorder to the section on SUD, as mentioned above, and trichotillomania to "Obsessive-Compulsive and Related Disorders." But as a further illustration of the complexities and challenges involved in categorizing disorders, a decision was made to place ADHD, one of whose most prominent features is impulsivity, into a new category called "Neurodevelopmental Disorders."

18.2.3 Epidemiology and Comorbidity of Behavioral Addictions

ICD are very common in the general population. In the National Comorbidity Survey Replication (NCS-R), 12-month and lifetime prevalence of any disorder with prominent impulsivity were 9 % and 12 %, respectively (Kessler, Berglund, et al., 2005; Kessler, Chiu, Demler, & Walters, 2005). A much larger and more recent study of 35,000 Americans found that 17 % of the sample was impulsive (Chamorro et al., 2012). This impulsive subgroup had significantly higher rates of psychopathology, functional impairment, dangerous behavior, and a history of trauma. About 80 % had a psychiatric disorder and were more likely than non-impulsive subjects to have attempted suicide, engaged in domestic violence, started fights, or driven recklessly.

In addition, multiple studies in the USA and abroad have shown high rates of ICD in individuals with psychiatric illness. Prevalence rates range from 20 to 30 % with the BA commonly seen in ED and SUD among the most common (Grant et al., 2005; Müller et al., 2011). These include compulsive buying (6–10 %), kleptomania (8 %), pathological gambling (7 %), pathological Internet use (3–5 %), and nonparaphilic compulsive sexual behavior (2–3 %). These studies also found that despite being frequently comorbid, BA are often underdiagnosed by clinicians.

18.2.4 Prevalence and Clinical Significance of Behavioral Addictions in Eating Disorders

In the DSM-5, BED has been officially recognized as a distinct major ED, along with AN and BN. For each of these conditions, different patterns of comorbidity with BA are seen. In general, individuals with ANBP, BN, and BED have higher rates of not only SUD but also impulsivity and ICD compared to those with the ANR (Brewerton et al., 1993; Cassin & von Ranson, 2005; Fernández-Aranda, Pinheiro, et al., 2008).

Multiple studies have shown high rates of ICD in ED. The earlier cited NCS-R found all the major ED to be highly comorbid with ICD with rates of 64 %, 44 %, and 31 % for BN, BED, and AN, respectively (Hudson, Hiripi, Pope, & Kessler, 2007). In the past decade, two large international studies have found that individuals with ED have high rates of ICD (Fernández-Aranda, Jiménez-Murcia, et al., 2006; Fernández-Aranda, Pinheiro, et al., 2008). The prevalence of ICD in the BN subgroup in the 2006 study ranged between 20 and 25 %, and in the 2008 study, one out of six subjects with any ED met criteria for an ICD. In the 2006 study, the BN + ICD group had significantly higher rates of psychopathology, including SUD, than the BN-only group and a comparison cohort with pathological gambling. In the 2008 study, the ED + ICD group, compared to the ED-only group, engaged in significantly greater use of laxatives, diuretics, and fasting to control weight. On psychological assessment, this comorbid group had significantly greater body image disturbance, cognitive impulsivity, and other comorbid psychiatric disorders, including borderline personality. The authors concluded that if untreated, comorbid ICD could lead to a more difficult clinical course with less likelihood of recovery in individuals with ED (Fernández-Aranda, Pinheiro, et al., 2008).

Of particular concern, high levels of impulsivity are associated with increased risk for self-harm in individuals with ED, which have rates of attempted suicide comparable to those seen in major depressive disorder (Bulik, Sullivan, & Joyce, 1999). Multiple studies have found that this already elevated risk for suicide attempts and self-harm is increased further in ED patients with high levels of impulsivity (Corcos et al., 2002; Stein, Lilenfeld, Wildman, & Marcus, 2004).

18.3 Behavioral Addictions Frequently Comorbid with Eating Disorders

18.3.1 Kleptomania

Shoplifting is an extremely common behavior with US prevalence rates of about 14 % in men and 8 % in women (Hoertel, Dubertret, Schuster, & Le Start, 2012). Some experts report that about 5 % of shoplifters suffer from kleptomania, a DSM ICD characterized by recurrent inability to resist impulses to steal (Dell'Osso, Altamura, Allen, Marazziti, & Hollander, 2006), while more recent data indicates the range of shoplifters with kleptomania to be 4–24 % (American Psychiatric

Association, 2013). Individuals with kleptomania are not driven by any type of financial incentive when engaging in this behavior and often feel ashamed of what they have done. Affected individuals experience enormous tension before engaging in stealing and a sense of relief after it is over.

Although there is limited data on kleptomania's prevalence and clinical course, it is believed to be rare with prevalence estimates below 1 % (Dell'Osso et al., 2006; Schreiber et al., 2011), and it is thought that some patients experience problems only intermittently, whereas others show a chronic course (American Psychiatric Association, 2013). About two-thirds to three-fourths of affected individuals are female (American Psychiatric Association, 2013; Grant, Odlaug, Davis, & Kim, 2009). There are high rates of comorbidity with mood, anxiety, and SUD, which are also common in first-degree relatives (Schreiber et al., 2011). It is also commonly comorbid with ED (American Psychiatric Association, 2013; McElroy, Pope, Hudson, Keck, & White, 1991).

Affected individuals can suffer significant functional impairment including being arrested and spending time in jail (Grant & Kim, 2005). One study found that about two-thirds of individuals with kleptomania had been arrested and 20 % incarcerated over a 6-year period (Grant, Odlaug, et al., 2009).

No evidence-based pharmacological or psychosocial treatments for kleptomania are currently available due to a paucity of research in this area (Grant, Kim, & Odlaug, 2009). The first double-blind controlled medication trial to demonstrate efficacy found that administration of naltrexone resulted in significant reductions in stealing urges and behavior (Grant, Kim, et al., 2009).

18.3.2 Hypersexual Behavior (Sexual Promiscuity)

Although not felt to justify inclusion as a new, stand-alone disorder in the DSM-5, hypersexual behavior is common in the general population and associated with significant distress and functional impairment. Present in many psychiatric disorders, sexual promiscuity is also commonly seen in individuals with the binging and purging subtypes of ED. In a Japanese study of more than 230 ED patients, 22 % of those with BN, and 16 % of those with ANBP reported having sex with a person they did not know well compared to 5 % of a comparison control group. Individuals with ANR reported the lowest rates of any group (2 %) (Nagata, Kawarda, Kiriike, & Iketani, 2000).

Sexual promiscuity in patients with BN often denotes more severe psychopathology. Two lines of distinct but overlapping research have examined this issue, one involving a highly impulsive subgroup of bulimics called "multi-impulsive bulimics" and the other exploring the role of comorbid borderline personality as a potential mediator or contributor to sexual promiscuity in women with BN (Wonderlich, Myers, Norton, & Crosby, 2002).

Multi-impulsive bulimia (MIB), named by Lacey (1993), refers to patients with severe impulsivity in many domains, more severe psychopathology, and poorer response to treatment. According to one of the most widely used diagnostic systems

for this syndrome, affected individuals must have exhibited at least three of the following six impulsive behaviors: (1) suicide attempts; (2) severe self-harm; (3) shoplifting, excluding food; (4) alcohol abuse; (5) drug abuse; and/or (6) sexual promiscuity (Fichter et al., 1994). A more recent study validated these criteria in a study of 125 women with BN (Myers et al., 2006). The MIB group had a higher incidence of anxiety disorders, child abuse, and daily self-damaging behavior (including sexual promiscuity) than the non-MIB group. A high proportion of the sample, 44 %, met criteria for MIB.

Borderline personality disorder (BPD), in which there is frequently impulsive sexual behavior and many other features of MIB, is common in individuals with ED, particularly those with ANBP and BN. In a meta-analysis of studies examining the relationship between personality and ED, the rate of comorbid BPD in ED was at least 20 % and associated with greater overall psychopathology and poorer response to treatment although not increased severity of ED pathology (Cassin & von Ranson, 2005). MIB and BPD are also frequently comorbid and appear to overlap. In an earlier cited study, ED patients with comorbid ICD and other evidence of impulsivity were significantly more likely than those without ICD to have BPD (23 % versus 11 %) (Fernandez-Aranda et al., 2008). Conversely, other research has shown that comorbid BPD increases the risk for MIB (Nagata et al., 2000).

18.3.3 Internet Addiction

Although not a stand-alone diagnosis in the DSM-5, problematic and/or excessive Internet use is a major clinical problem worldwide that causes enormous suffering, functional impairment, and lost productivity (Weintraub et al., 2011). The lack of empirical support for this syndrome as a disorder in its own right has resulted in the creation of a subtype of Internet addiction called "Internet gaming disorder" and listed in Section III of the DSM-5 as a condition requiring further study.

What is indisputable, however, is that the Internet serves as a vehicle for many psychiatrically ill patients to engage in unhealthy behaviors that exacerbate their disease. For patients with ED, the availability of the Internet has had mixed effects. A concerning example is that in contrast to websites that provide positive help and psychoeducation about ED ("pro-recovery sites"), a larger number of pro-eating disorder websites ("pro-ANA") actually support eating disordered lifestyles and values and provide information and group discussion on how to maintain unhealthy low weight and pathological behaviors (Wilson, Peebles, Hardy, & Litt, 2006) through the use of virtual communities.

Wilson and colleagues (2006) surveyed 700 females with ED (ages 10–22) and their parents about their knowledge and use of ED-related websites. Approximately 50 % of parents were unaware of pro-ANA websites and that their children were visiting them (Wilson et al., 2006). More than one-third of the patients visited pro-ANA sites, and about 40 % visited both pro-ANA and pro-recovery sites. Of note, 96 % of those who visited pro-ANA sites reported learning new techniques for

losing weight or purging, and, even more astonishing, almost 50 % of those who visited pro-recovery sites indicated that they also learned new weight loss techniques. Visiting these websites resulted in other adverse consequences: spending less time on schoolwork, longer duration of illness, more frequent hospitalizations, and spending significantly more time on the Internet each day.

Another study of 60 females with ED found positive associations between compulsive Internet use and compulsive Internet buying, both of which showed significant positive correlations with emotional lability and measures of impulsivity (Claes et al., 2012). Conversely, individuals with Internet addiction may have elevated rates of disordered eating. In a survey of more than 2,000 Chinese college students, those with Internet addiction reported significantly elevated binge eating, concerns about weight, and weight change compared to those without problematic Internet use (Tao, 2013).

18.4 Behavioral Addictions That Are Often Comorbid with Substance Use Disorders

SUD and BA are united by the core feature of an individual's recurring failures to resist the temptations, impulses, or drive to engage in particular behaviors known to have harmful consequences (Grant et al., 2010). Similarly, both SUD and BA tend to have similar ages of onset, typically emerging during adolescence or early adulthood, and higher rates of both classes of disorders among younger people (Goodman, 2008). These conditions tend to be chronic with high rates of remittance and relapse, as well as relatively high rates of recovery without formal treatment (Grant et al., 2010). While well known in SUD, those suffering from BA also tend to report physiological symptoms including craving; marked tolerance with repeated exposure; an increase in the behavior is necessary to sustain pleasurable effects; and symptoms of discomfort occurring with abrupt cessation of the behavior (Goodman, 2008). Finally, Goodman (2008) has noted that as individuals begin to effectively manage symptoms of SUD or BA, novel or dormant addictive behaviors tend to materialize or intensify.

These overlaps between SUD and BA are likely due to shared neurobiological underpinnings that are strongly associated with reward and reinforcement. Olsen (2011) notes that stimuli that are inherently reinforcing, such as seeing delicious food on a plate, or sexual behavior, activate the same neural regions as gambling, shopping, and playing video games. These regions are believed to include the dopaminergic rich mesocorticolimbic system and extended amygdala (Volkow, Fowler, & Wang, 2004). Further, these neural areas are also activated by drugs of abuse, in particular, cocaine, amphetamine, alcohol, nicotine, and tetrahydrocannabinol (Koob, 1999). A predominant theory regarding the mechanisms driving drug addiction entails the manner in which particular substances that users initially take voluntarily because of their reinforcing properties transition into substances that ultimately drive compulsory behaviors commonly seen in substance dependence. Everitt and Robbins (2005) argue that the switch of a substance from having

reinforcing properties to one that users must habitually consume indicates a corresponding neuroplasticity of cortical and striatal structures, specifically, loss of prefrontal cortical control of the behavior to striatal command. It is now believed that BA induce similar neuroadaptations of the same structures (Olsen, 2011), in particular pathological gambling (Potenza, 2013). These observations suggest the utility of considering SUD and BA together.

18.4.1 Gambling Disorder

With the first major revision of the DSM in nearly 20 years, the disorder formerly known as pathological gambling (PG) has undergone several changes for its new appearance in DSM-5 (American Psychiatric Association, 2013). In addition to its being moved into the section containing SUD, its name has been changed to gambling disorder (GD) as the term "pathological" was seen to carry a negative valence and has become antiquated (Petry et al., 2013). In this chapter, PG and GD will be used interchangeably. Conceptually, we will follow the DSM-5 criteria for the condition and refer to it as GD; however, the vast majority of the research in this area refers to the condition as PG. More importantly, the diagnostic criteria of GD have changed; chief among them is the removal of the criterion regarding the committing of illegal acts, such as fraud, theft, or embezzlement in order to finance gambling. Individuals whose gambling habits require illegal acts to fund their gambling rarely fail to endorse other diagnostic criteria of the condition (Zimmerman, Chelminski, & Young, 2006). It is unlikely that removing this criterion will result in a loss of diagnostic sensitivity (Petry et al., 2013). The threshold for diagnosis of this disorder has also changed (American Psychiatric Association, 2013). Out of the nine criteria, a diagnosis is permissible if four are endorsed, instead of the DSM-IV-TR threshold of five items. Several studies have demonstrated superior diagnostic sensitivity when four items are used (Jimenez-Murcia et al., 2009; Stinchfield, Govoni, & Frisch, 2005). Obviously, increased detection of GD will allow more sufferers to get into treatment.

The DSM-5 criteria for GD include two major categories: one regarding recurring maladaptive gambling and a second noting that such behavior cannot be exclusively accounted for during a manic episode (American Psychiatric Association, 2013). Several of these criteria are very similar to SUD, including a preoccupation with the behavior, the need to invest larger and larger amounts of money to achieve the same thrill, repeated and unsuccessful attempts to reduce or abstain from the behavior, feelings of irritability or restlessness during attempts to cut back, gambling during times of stress or as a means to escape problems, and attempts to conceal the extent of the behavior from others (American Psychiatric Association, 2013). The remaining features include significant disruption in social, vocational, or educational functioning because of gambling, a tendency to spend more money than intended to compensate for losses, and/or the reliance on a monetary bailout from others because of a financial catastrophe created by gambling (American Psychiatric Association, 2013). It should also be noted that the term "problem

gambling" is frequently seen in the literature. This label tends to be applied to gambling behavior that causes problems for an individual, but which is below diagnostic threshold (Petry et al., 2013). Typically, any individual that endorses a single criterion listed in the DSM could be exhibiting problem gambling.

Unlike patients suffering from SUD, who often present with physical manifestations of their addictions, those suffering from GD have no outward signs of their addiction. For example, individuals intoxicated by alcohol show slurred speech and ataxia; inhibitions are lowered; behavior may become erratic. Individuals under the influence of methamphetamine may show akathisia. Intravenous drug users have scar tissue or distinct skin lesions. Metabolites in the urine and/or serum blood levels of most drugs of abuse can be detected. Gambling disorder, however, has no outward signs and has been called the "hidden addiction," making it harder to detect than SUD (Phillips, 2005). And rarely do these patients present to treatment; one study found that only roughly one in ten with the disorder will seek help (Slutske, 2006).

Interestingly, almost half of the respondents with PG in the National Comorbidity Survey Replication reported being under the care of a mental health professional at some point for a mental health disorder, yet none reported receiving treatment for the gambling problem (Kessler et al., 2008). Detection of GD may also be difficult since nonproblem gambling has such high base rates in the USA. One national survey of almost 5,000 individuals found that 75 % participated in various activities where wagers were placed on an unknown outcome (Welte, Barnes, Tidwell, & Hoffman, 2011). Such bets ranged from church raffles and card games in basements to wagers placed on sporting events and attending casinos (Welte et al., 2011). Even though mere endorsement of intravenous drug use by a patient may signal to a clinician the presence of a possible opioid addictive disorder, the same is not true with respect to GD for individuals presenting for treatment who report betting money on the outcome of the Super Bowl, for example.

While identification of GD is likely to improve with the DSM-5 criteria, it is not thought that the prevalence of the condition will rise (Petry et al., 2013). Research regarding prevalence rates are based on the earlier DSM-IV-TR PG criteria are variable. Kessler et al. (2008) estimated the adult 12-month PG prevalence at 0.3 % and lifetime prevalence at 0.6 %. Blinn-Pike, Worthy, and Jonkman (2007), however, determined a nearly 8 % lifetime prevalence when studying college students. Barnes, Welte, Hoffman, and Tidwell (2009) found that 10 % of adolescents and young adults reported having three or more gambling problems in the past year. Ultimately, a thoughtful meta-analysis by Sussman, Lisha, and Griffiths (2011) estimates the adult 12-month prevalence of PG to be 2 % in the USA. Generally, more men suffer from PG than women (Desai & Potenza, 2008; Petry, Stinson, & Grant, 2005).

The finding of higher rates of PG in younger adults is a consistent observation. This is not surprising as some studies report that as many as 90 % of adolescents have engaged in gambling (Gupta & Derevensky, 2000), with an average age of first wager being 12 years old (Wilber & Potenza, 2006). In one study of more than 1,600 adolescents in Connecticut, 69 % reported gambling before their 12th

birthday (Rahman et al., 2012). Children engage in a wide range of gambling behaviors, including buying lottery tickets, playing dice and card games, betting on sporting events, as well as entering casinos despite being under age (Rahman et al., 2012; Wilber & Potenza, 2006). Given that gambling is quite prevalent, is more socially acceptable than substance use in minors, and does not cause intoxication, it is no surprise that most children will have an experience with gambling before they initially use nicotine, alcohol, or other drugs of abuse (Wilber & Potenza, 2006). Experiences with gambling earlier in life may account for the finding that a majority of individuals develop PG earlier in their lives than other addictions. Jimenez-Murcia and her colleagues (2009) found that 45 % of their 904 PG patients reported age of onset of this disorder before the end of their 20s. Kessler et al. (2008) found similar results, with a median age of onset of the disorder at age 23, also noting that individuals with PG tended to start gambling at significantly earlier ages than those who do not have PG.

Blaszczynski and Nower (2002) constructed a pathways model of problem and pathological gambling that is useful when conceptualizing PG. Their model captures the complexity of this disorder and acknowledges its heterogeneous and multidimensional nature, proposing that there are clinically distinct subgroups of sufferers. While all share core symptoms, Blaszczynski and Nower (2002) argue that Pathway 1 of PG consists of "behaviorally conditioned gamblers" who engage in pathological gambling, but are otherwise free from premorbid psychopathology and SUD and are easily treatable. This group tends to have the least severe gambling habits and may even manage reasonable gambling behavior following treatment. Pathway 2 is a second group of "emotionally vulnerable" individuals, characterized by those with preexisting mood disorders and who possess limited problem-solving abilities and poor coping strategies, often coming from disordered families. This group is believed to engage in gambling as a means of emotional escape and is more difficult to treat, as premorbid conditions must also be addressed. Finally, Pathway 3 is the group with the most severe form of PG. It includes "antisocial impulsivist" problem gamblers, who have high rates of impulsivity, drug and alcohol use, criminal behavior, and poor interpersonal abilities. They tend to be the most difficult to treat, it is suspected, because of underlying neurological dysfunction. In summary, this model suggests that some gamblers develop PG due to exogenous factors (Pathway 1), some as a maladaptive coping strategy (Pathway 2), and some, a more severe group, due to endogenous factors (Pathway 3).

Regardless of the pathway, PG can be quite impairing. Individuals with PG have higher rates than nonproblem gamblers of bankruptcy, contacts with law enforcement, divorce, incarceration, and a lower quality of life (Gerstein et al., 1999; Grant & Kim, 2005). Those suffering from PG have been known to have high rates of attempted suicide (Kausch, 2003; Potenza, Kosten, & Rounsaville, 2001), although several authors caution against a causal link between the two (e.g., see Newman & Thompson, 2003). Problem gambling may be a risk factor for intimate partner violence (Muelleman, DenOtter, Wadman, Tran, & Anderson, 2002). Further, those with problem gambling are far more likely to have deleterious health conditions,

particularly cardiac and hepatic conditions, than those without a gambling addiction (Morasco et al., 2006). Additionally, its rate of comorbidity with SUD is high. Lorains, Cowlishaw, and Thomas (2011) recently completed a meta-analysis based on general population studies of conditions that are frequently comorbid with GD and found the highest rates to be for nicotine dependence, followed by alcohol use disorder, and then other SUD. Additionally, GD is also highly comorbid with major depression, anxiety disorder, and antisocial personality disorder (Lorains et al., 2011).

18.5 Gambling Disorder and Specific Comorbid Substance Use Disorders

18.5.1 Nicotine Dependence

Nicotine dependence co-occurs with PG in very high rates, ranging from 76 % (Cunningham-Williams, Cottler, Compton, & Spitznagel, 1998) in a US sample to about 35 % in a South Korean study (Park et al. 2010). Grant, Desai, and Potenza (2009) speculate that high rates of nicotine dependence and PG co-occur for a variety of reasons, including a genetic predisposition for impulsivity, a tendency for nicotine to facilitate the reinforcing effects of dopamine in the presence of stimuli eliciting such effects, the cognitive-enhancing effects of smoking enhancing experiences involving wagering, and social factors involved in gambling and smoking in groups. Potenza and his colleagues (2004) note that problem gamblers reporting daily tobacco use are more likely to endorse depression and suicidal ideation because of their gambling habit, as well as higher rates of gambling-related arrests, SUD, and engagement in mental health treatment. Grant, Kim, et al. (2009), however, note that even those who are not dependent on nicotine, but who have problematic gambling, still show high rates of SUD and other psychopathology.

18.5.2 Alcohol Use Disorders

Alcohol abuse and dependence also co-occur with PG at very high rates. Petry et al. (2005) examined a sample in excess of 43,000 people living in the USA and found that of those meeting criteria for PG, an astonishing 73 % also met criteria for alcohol abuse or alcohol dependence (collectively known as "any alcohol use disorder"). Other groups have found lower but still very elevated rates of 44 % (Cunningham-Williams et al., 1998) and 18 % (Welte, Barnes, Wieczorek, Tidwell, & Parker, 2001). As a result, clinicians treating GD are obligated to also assess for alcohol use disorders (AUD). This may be particularly true for patients from higher socioeconomic statuses, as Welte et al. (2001) found that sufferers of PG who fell into higher SES strata were more likely to be dependent on alcohol. It should also be of concern to those working with GD patients in clinical settings that comorbid

AUD and PG have been associated with increased rates of intimate partner violence (Brasfield et al., 2012).

Given the concerning frequent co-occurrence of problematic alcohol use and gambling, it is desirable to understand what links these conditions. However, testable models regarding *why* GD and AUD co-occur remain elusive (Grant, Kushner, & Kim, 2002). Potenza (2008) makes a convincing argument for overlapping cortical systems that predispose individuals to develop gambling problems and excessive alcohol use. Additionally, Fischer and Smith (2008) note that a specific personality type may represent the expression of possible overlapping brain systems; individuals who are high in "trait urgency" (a form of impulsivity) tend to act rashly when distressed. This tendency is strongly associated with problem drinking in both genders, as well as problem gambling in men and binge eating in women.

18.5.3 Illicit Drug Use

As with AUD, GD is frequently comorbid with illicit drug use. Petry et al. (2005) found that 38 % of those with PG also endorsed drug abuse or dependence. Very high rates of nicotine dependence, AUD, and cannabis use occur in those meeting criteria for GD. PG and problem gambling also occur with relatively high frequency in samples of those with drug use disorders, ranging from 8 to 15 % of those using cocaine; similar rates are found in opioid-dependent individuals engaged in methadone maintenance (Petry & Champine, 2012). Interestingly, in a study measuring PG in treatment-seeking substance abusers, those scoring highest on a pathological gambling measure were those abusing cannabis (Toneatto & Brennan, 2002); 24 % of cannabis abusers scored in the critical range on a gambling screen, a far higher rate than seen in those seeking treatment for alcohol (4 %), cocaine (12 %), or opioid abuse (5 %). Leeman and Potenza (2012) argue that commonalities between those with PG and SUD on neuropsychological tasks (particularly those involving impulsivity, compulsivity, and risk/reward decision making) suggest that both groups share dysfunction in the ventromedial prefrontal cortex and striatum, as well as in monoamine, glutamate, and opioid neurotransmitter systems.

Those with both GD and a SUD are likely to have more severe symptoms of disordered gambling, with onset of problem gambling earlier, and tend to be resistant to treatment. As GD is an "invisible addiction" and is not typically the presenting problem when seeking treatment, clinicians are advised to screen for GD in all of their SUD patients. Patients who are first known to have GD are in turn best served by also being screened for co-occurring drug and alcohol problems.

18.6 Gambling Disorder and Comorbid Eating Disorders

PG and ED are rarely comorbid, despite both being highly comorbid with SUDs. In a 2013 study, Jiménez-Murcia et al. examined 1,681 ED patients and found only 25 had co-occurring gambling disorder. In an investigation of 94 individuals with BED, only a single patient met criteria for both an ED and PG (Yip, White, Grilo, & Potenza, 2011). However, it should be noted that individuals who report symptoms of GD and who do not meet full clinical criteria may have increased lifetime rates for ED and disordered eating (von Ranson, Wallace, Holub, & Hodgins, 2013).

18.7 Treatment of Gambling Disorder

Many PG patients recover without formal intervention (Slutske, 2006). Those with both PG and a SUD, however, have worse outcomes (Ladd & Petry, 2003). Treating GD is similar to treating other addictions, and many therapies for PG have been adapted from SUD treatment. For example, the most common treatment for PG is Gamblers Anonymous (GA), closely modeled on the abstinence model of Alcoholics Anonymous (AA) (Petry, 2007) (see Chap. 27). CBT has been shown to be effective in treating PG (Okuda, Balán, Petry, Oquendo, & Blanco, 2009; Petry et al., 2006), although the best results seem to result when CBT is combined with GA (Petry, 2005). Brief therapy, such as motivational enhancement therapy (MET), based on existing treatments for alcohol abuse, has also been shown to be effective (Hodgins, Currie, Currie, & Fick, 2009) (see Chap. 22).

There has been much research on drug treatments of GD, but, as of the writing of this book, none have an FDA indication. A 2007 meta-analysis concluded that antidepressants, mood stabilizers, and opiate antagonists are superior to placebo (Pallesen et al., 2007). Additionally, N-acetylcysteine (NAC), a glutamate modulator, has been shown to lessen the symptoms of PG in a small ($n = 12$) sample (Grant, Kim, & Odlaug, 2007). In a case report, disulfiram was shown to curb gambling urges in a person with PG (Mutschler et al., 2010). Outcomes with the atypical antipsychotic olanzapine have been equivocal (Fong, Kalechstein, Bernhard, Rosenthal, & Rugle, 2008). Pharmacotherapy is a viable option for GD co-occurring with a mood disorder, although no agent has emerged as a frontline treatment (Hodgins, Stea, & Grant, 2011).

18.8 Treatment of Comorbid Eating Disorders, Substance Use Disorders, and Behavioral Addictions

Little empirical data exists to inform treatment planning in individuals with ED, SUD, and BA. In fact, scant published literature exists on the treatment of individuals presenting with only the "dual diagnoses" of ED and SUD, with little agreement on or guidelines for "best practices" with this population (Brandt, Crawford, & Halmi, 2011). Reasons for this lack of research relate to the fact that

until recently most experts have argued that treatments for these disorders should be sequential rather than concurrent (Brandt et al., 2011; Courbasson, Nishikawa, & Dixon, 2012). In addition, controlled treatment studies of ED and SUD typically exclude patients showing comorbidity with the other disorder (Courbasson et al., 2012).

Remarkably, no pharmacologic treatment studies of individuals with both ED and SUD have been published, and only one psychosocial treatment study of patients with both disorders has been published (Courbasson et al., 2012). In this small outpatient study, 25 women with ED and SUD were randomized to dialectical behavioral therapy (DBT) or treatment as usual (TAU) (Courbasson et al., 2012). The treatment trial lasted 1 year with subsequent follow-ups at 3 and 6 months. The DBT group had a much higher rate of retention posttreatment (80 % to 20 %) and at follow-up (60 % versus 20 %). DBT resulted in positive changes in multiple domains, including improvements in abnormal behaviors and attitudes related to the ED, substance use severity, regulation of negative mood, and depressive symptoms. Subjects' perceived ability to manage and cope with negative emotions was significantly associated with reductions in emotional eating and increases in confidence to resist cravings for substances. For ethical reasons, the TAU arm had to be terminated prematurely because of worsening symptoms, preventing a head-to-head comparison of the two treatment groups. The authors noted that the huge dropout rate in the TAU arm confirms that retention of this patient group in treatment is very challenging.

Currently, expert clinical experience and the extant limited treatment literature have converged on recommending that comorbid ED and SUD should be treated concurrently (see Chap. 21). Based on what we currently know about these disorders and BA, a recommended treatment program for individuals with all three disorders should be multimodal using a chronic disease model in a patient population with a high degree of morbidity, mortality, relapse, and recurrence. Given the multiple comorbidities and needs of these patients, frequent contacts and coordination of care with multiple providers with expertise in each of the specific comorbid disorders are often necessary to ensure optimal clinical outcomes.

The initial component of successful intervention requires thorough assessment that includes systematically asking about specific BA including gambling disorder, kleptomania, problematic Internet use, and excessive/inappropriate sexual behavior. These conditions often go unrecognized because clinicians working with patients with ED and/or SUD do not think to ask about them.

Since all these conditions are associated with unsafe and potentially dangerous behaviors that cause enormous functional impairment and place individuals at much higher risk for self-harm and harm to others, assessment of safety and level of functional impairment is particularly important in this population. For GD patients, financial and legal problems and their impact on family relationships need to be investigated. Has the person declared bankruptcy or been incarcerated? For the person with hypersexual behavior, assessment for sexually transmitted diseases and unwanted pregnancies is crucial. For the individual with problematic Internet use, the impact of these activities on vocational functioning and

relationships must be assessed to determine if excessive use has been associated with academic failure or poor work performance. In addition, has the person used the Internet to fuel another addiction such as going to pro-eating disorder web sites, gambling on-line, or accessing pornography?

The elevated rates of trauma in the backgrounds of patients with ED, SUD, and BA mandate an inquiry about a history of childhood physical, sexual, and emotional abuse (See Chap. 17). The assessment should also include questions about exposure to traumatic experiences in adulthood such as rape and domestic violence. Finally, there should be evaluation for the psychiatric sequelae of trauma, such as PTSD and borderline personality.

A flexible biopsychosocial approach to case formulation and treatment is optimal to address the myriad of problems these patients present. Treatment selection should be guided by the recognition that individuals with these comorbid disorders have problems with *global dysregulation* including affect regulation difficulties, impulsivity, addiction, and compulsive and habitual self-destructive behaviors, which some experts argue should be the major focus of intervention rather than a specific syndrome (Dennis & Sansone, 1997). Therefore, combinations of treatments shown to work for these deficits should be considered. For example, DBT and CBT are potentially helpful for these disorders. In addition, preliminary studies suggest that SSRIs and naltrexone may show promise as potential pharmacological treatments for BA, providing indirect support that these illnesses may overlap in part with mood disorders, anxiety disorders, obsessive–compulsive illnesses, and alcohol and drug use disorders.

In addition to interventions effective in treating all BA, consideration should be given to treatments specific for particular disorders such as Gamblers Anonymous for those with GD. Treating the major comorbid mood, anxiety, and personality disorders seen in ED and SUD must also be factored into treatment plans for addressing these complex and challenging conditions.

Conclusions

In summary, illness severity is greater and treatment response worse for ED and SUD when comorbid with BA at least in part because there are currently no evidence-based treatments for individuals with both ED and SUD with and without the accompanying BA discussed in this chapter. Moreover, with the exception of GD, there continues to be a lack of consensus regarding the validity of the other BA reviewed despite their obvious clinical importance. Accordingly, it is difficult to devise well-designed treatment studies for conditions whose existence and boundaries continue to be debated.

Nevertheless, future research directions for understanding the etiology of these comorbid conditions and developing novel interventions for them have been suggested by our growing understanding of the natural history of ED, SUD, and BA. For example, there is increasing evidence that impulsivity may be a risk factor for the development of many of the syndromes reviewed in this chapter. It has been shown, for instance, that impulsivity in adolescence actually predicts, often within less than a year, the development of signs and symptoms of ED

(Wonderlich, Connolly, & Stice, 2004). In addition, in one of the largest cross-sectional studies of adult ED and ICD cited earlier, in more than 60 % of the subjects ICD preceded BA, and in almost half the sample, the onset of both illnesses was within a 3-year period (Fernández-Aranda, Pinheiro, et al., 2008).

As Fernández-Aranda, Pinheiro, et al. (2008) point out, these findings raise questions about the etiology of this comorbidity. Is the development of ED symptoms after the onset of ICD one of many manifestations of a core problem with impulsivity as seen in multi-impulsive bulimia (Lacey, 1993), or do those with comorbid ICD have more severe variants of ED related to genetic or environmental factors (Fernández-Aranda, Pinheiro, et al., 2008)? Better understanding of the nature of this comorbid relationship is essential in order to devise effective treatments for patients with ED, SUD, and ICD.

Certainly, one possible environmental factor that may be a leading candidate for the development of these disorders of impulse control is trauma, such as sexual abuse, which, with the advances of neuroimaging, has been shown to cause structural brain changes (Heim, Mayberg, Mletzko, Nemeroff, & Pruessner, 2013). In this context, psychosocial adversity may also increase the risk for the development of comorbid ED and ICD. Multiple studies have shown an association between a history of childhood abuse and multi-impulsive bulimia (Fichter et al., 1994; Wonderlich et al., 2001). In the earlier cited Myers et al. study (2006), women with MIB were significantly more likely than those with BN alone to have had a history of physical, sexual, and emotional abuse. They also were significantly more likely to suffer from post-traumatic stress disorder (PTSD) (34 % versus 14 %).

And, as cited earlier, the characteristic of "trait urgency," which may represent deficits in specific brain systems, is associated with BA but sometimes with gender differences, suggesting that different phenotypes of BA may be related to the same underlying pathophysiology (Fischer & Smith, 2008). In other words, as the authors point out, gambling disorder in men and binge eating in women may actually be related to the same core deficits.

As we await further research, and a better understanding of the nature of non-DSM BA like Internet addiction and hypersexual behavior, additional controlled studies of treatments for comorbid ED and SUD should be conducted, as discovery of evidence-based treatments for these two disorders with prominent impulsivity may also be useful as interventions in BA. More controlled trials of both psychosocial and drug treatments of BA should also be performed given the preliminary promising results of studies cited in this chapter. The key to reducing the devastating effects of these comorbid disorders is for a greater collaboration among experts and researchers in ED, SUD, and BA in developing more effective treatments that alleviate the tremendous suffering of affected individuals.

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Compulsive Buying: Relationship to Eating Disorders, Substance Use Disorders, and Other Impulse Control Disorders

19

Astrid Müller and James E. Mitchell

Abstract

Compulsive buying remains a relatively neglected problem, although research on this disorder has increased over the last decade. This chapter will briefly review the literature on compulsive buying, define the disorder as it is currently understood, suggest how it might be classified, and present the available data on prevalence. Although compulsive buying disorder is not included in DSM-5, working diagnostic criteria are available. Compulsive buying is defined as the preoccupation with buying and shopping, generally characterized by frequent buying episodes and the overpowering urge to buy. The maladaptive buying behaviors are associated with significant social, occupational, or financial problems. The available prevalence data suggest this disorder occurs in approximately 6–7 % of individuals. In this chapter, we also focus on the relationship between compulsive buying disorder and certain comorbidities. We will examine the relationship between compulsive buying and eating disorders, as there does appear to be an association between these two types of problems. We will then turn to the relationship between compulsive buying disorder and substance use disorders. Although the data here are less clear, there also may well be an association. Third, we will discuss compulsive buying and other impulse control disorders. Treatment studies for those with compulsive buying disorder thus far have been very limited. Of note, antidepressant medications have not been shown to be a particularly effective strategy. There is some evidence that

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cognitive behavioral therapy may be an appropriate intervention, but this has not been adequately studied.

Keywords
Compulsive buying disorder • Oniomania • Eating disorders • Substance use disorders • Impulse control disorders • Cognitive behavioral therapy • Pharmacotherapy

19.1 Introduction

Although compulsive buying (CB) has only been the focus of research in recent years, the disorder was actually described more than 100 years ago. Kraepelin (1909), in his classic psychiatric textbook, described "oniomania," which can be translated to mean madness involving something for sale. Following his description, the concept CB retreated into relative obscurity with only occasional references in the literature until recent years. Now, there is a growing interest in understanding this disorder including its prevalence, comorbidities, and treatment. However, a major problem remains in that CB tends to be rarely identified in clinical practice, and most therapists tend not to inquire about difficulties in this area unless there is some obvious suggestion of a problem. In this chapter, we will review this literature briefly and highlight some of the problems with our current understanding. We will close by focusing on the issue of treatment, which has received surprisingly little attention.

19.2 Compulsive Buying: Phenomenology, Prevalence, Etiology, and Classification

CB is actually referred to by several different terms, including "compulsive shopping," "pathological buying," and "shopping addiction." The latter regards CB as a form of a maladaptive "process addiction" similarly to other addictions to certain activities such as gambling, binge eating, or compulsive Internet gaming. The most commonly used term, however, remains "compulsive buying" that was used in the 1990s to delineate CB from "impulsive buying." The word "impulsive buying" is used in consumer research and describes spontaneous, immediate shopping sprees that are mostly externally driven and often occur in ordinary consumers. In contrast to impulsive buying, CB refers to chronic maladaptive buying behavior that leads to marked negative consequences.

The diagnostic criteria usually employed in research settings as well as clinically were proposed by McElroy, Keck, Pope, Smith, and Strakowski in 1994. These define CB as a preoccupation with buying and shopping, characterized by frequent buying episodes or overpowering urges to buy that are experienced as irresistible or senseless. The episodes are accompanied by a sense of relief and pleasure, which

are followed by feelings of remorse and guilt, and are accompanied by significant social, occupational, or financial problems. Despite considerable research on the topic, no diagnostic criteria are included in either the American Psychiatric Association DSM-5 (American Psychiatric Association, 2013) or the International Classification of Diseases ICD-10 (World Health Organization, 1993).

The clinical picture seen in CB has been well characterized by studies using descriptive samples (Christenson et al., 1994; Müller et al., 2012; Schlosser, Black, Repertinger, & Freet, 1994). Many patients with CB experience a sense of relief or pleasure while engaging in the behavior, but afterwards experience remorse, and develop the negative consequences of their behaviors often including social and economic problems. Of note, individuals who engage in CB rarely use the items they buy; they may store them, take them back, or give them away as gifts. Many individuals with CB will focus on specific items or groups of items (e.g., clothes, shoes, electrical equipment). Most report that their primary interest is in the buying behavior itself rather than the items purchased. For some, the interaction with sales staff is important, while others prefer to shop alone, including via the Internet or television. CB usually develops between the ages of 20 and 30 but is recognized as problematic only later (Christenson et al., 1994).

Relative to the epidemiology of CB, the available studies have generally found rates of CB among adults to be around 6–7 % (Koran, Faber, Aboujaoude, Large, & Serpe, 2006; Mueller, Mitchell, Crosby, et al., 2010; Raab et al., 2012), although a rate of 11 % was found in a sample of adolescents in a study in Italy (Villella et al., 2011). Overall, younger people seem to be more prone to develop CB than older individuals. Treatment-seeking patients indicate that the onset of the disorder is often in their early twenties (Schlosser et al., 1994). With regard to a possible gender effect, research has produced mixed results. While some studies reported on a higher prevalence of CB among women, others did not find gender differences.

In considering possible etiologies and predisposing factors, a number of issues that may be involved have been identified, some of which remain rather speculative. Some research has focused on the construct that CB represents a failure of self-regulation and that it frequently serves as a means to escape negative affects, such as anxiety, depression, or boredom (Claes et al., 2010; Miltenberger et al., 2003; Müller et al., 2012). There is also some evidence that those who strongly endorse materialistic values may be at higher risk (Dittmar, 2005; Rose, 2007). However, several authors have stressed the heterogeneity among those who have this disorder (DeSarbo & Edwards, 1996; Yi, 2013).

Biological factors have been examined, including activity in certain brain areas using functional magnetic resonance imaging. Brain regions associated with decision making have been implicated that resemble earlier findings regarding drug addiction or pathological gambling (Knutson, Rick, Wimmer, Prelec, & Loewenstein, 2007; Raab, Elger, Neuner, & Weber, 2011). Abnormalities of neurotransmission have also been suggested, particularly involving the serotonin transporter system (Devor, Magee, Dill-Devor, Gabel, & Black, 1999). However, nothing definitive has been identified, and these studies must be regarded as preliminary.

As mentioned above, CB is not included in the DSM-5. Exactly how CB should be classified is unclear. There is currently a lack of clinical and neurobiological studies on CB that contributes to the confusion. Various suggestions have been made in the literature, including classifying CB as an obsessive–compulsive-related disorder. Others have suggested that CB is an impulse control disorder, and more recently, the model of CB as a behavioral addiction ("shopping addiction") has been advocated (Grant, Levine, Kim, & Potenza, 2005; also please see Chap. 18). Given the overpowering urges to buy, the repetitive loss of control over shopping and spending, and the lack of discontinuation of buying despite marked negative consequences, CB indeed could be classified as a non-substance addiction. Again, there is a strong need for more research on this topic in order to classify this phenomenon appropriately and to develop treatment as well as prevention strategies.

CB rarely occurs as a problem in isolation, but instead occurs in individuals who have other comorbid psychiatric disorders, with anxiety and depressive disorders being particularly common (Black, Repertinger, Gaffney, & Gabel, 1998; Christenson et al., 1994; Mueller, Mitchell, Black, et al., 2010). The most prevalent lifetime anxiety disorders are social phobia and panic disorder, followed by obsessive–compulsive disorder. Although preliminary data suggest a link between childhood trauma, in particular witnessing violence and emotional abuse, and CB symptoms (Sansone, Chang, Jewell, & Rock, 2013), the prevalence rates for posttraumatic stress disorders apparently are not increased among individuals with CB. A subgroup also developed problems with hoarding, and the overlap between these two conditions is substantial (Frost, Steketee, & Tolin, 2011; Mueller, Mueller, Albert, et al., 2007). In addition, many persons with CB suffer from a personality disorder, most frequently avoidant, depressive, obsessive–compulsive, or borderline personality disorder (Schlosser et al., 1994).

Only a few studies have investigated the prevalence of eating disorders (ED), substance use disorders (SUD), and impulse control disorders (ICD) in individuals with CB compared to healthy controls.

19.3 Compulsive Buying and Eating Disorders

The few controlled interview-based comorbidity studies have produced mixed results with regard to the occurrence of ED in individuals with CB (Table 19.1). While some studies found higher lifetime prevalence rates in women with CB compared to healthy controls (Christenson et al., 1994; Mueller et al., 2009), others did not (Black et al., 1998; Mitchell, Redlin, Wonderlich, et al., 2002). This might be explained by the fact that the latter studies did not include binge eating disorder (BED).

As shown in earlier studies, BED is quite common at least among treatment-seeking patients with CB. Vice versa, individuals with BED are more likely to suffer from CB (Faber, Christenson, de Zwaan, & Mitchell, 1995; Marcinko, Bolanka, & Rudan, 2006). According to reports by patients with CB we have

Table 19.1 Lifetime prevalence rates (percent) of eating disorders, substance use disorders, and impulse control disorders in individuals with compulsive buying disorder (controlled studies)

	Christenson et al. (1994)	Black et al. (1998)	Mitchell et al. (2002)	Mueller et al. (2009)
	$N = 24$	$N = 33$	$N = 19$	$N = 30$
Any eating disorder	21[a]	15	5	33[a]
Anorexia nervosa	0	12	5	10
Bulimia nervosa	12	9	0 (0.0)	3
Binge eating disorder	17	n/a	n/a	27
Any substance use disorder	46[a]	21	53[a]	23
Alcohol use/dependence	46	18	47	23
Cannabis use/dependence	21	n/a	21	3
Any impulse control disorder	21[a]	n/a	n/a	23[a]
Intermittent explosive disorder	4			17
Kleptomania	4			7
Pathological gambling	8			3
Trichotillomania	4			3
Pyromania	0			0

[a]Significant differences to healthy control groups

treated, the decrease of CB behaviors during treatment can occasionally be accompanied by increasing BED symptomatology. This is in line with the findings of Christenson et al. (1994) and Mueller et al. (2009) who found relatively high lifetime prevalence rates of BED in their CB samples. In some individuals, CB and BED might represent alternative responses to similar underlying problems such as low self-esteem, depression, high impulsivity, decision-making deficits, and heightened reward sensitivity. Also, the substantial availability and a variety of goods and food may contribute to overspending and overeating.

With regard to controlled studies, it appears that anorexia nervosa and bulimia nervosa are less commonly linked to CB. However, other studies reported prevalence rates for anorexia nervosa up to 20 % and for bulimia nervosa up to 25 % among patients with CB (McElroy et al., 1994).

The prevalence of CB among those with other ICD was investigated in 227 women with bulimia nervosa (Fernández-Aranda et al., 2006). In this sample, CB was the most frequently diagnosed ICD with a lifetime prevalence of 18 %. Also, CB was the most prevalent ICD (prevalence rate 12 %) in another large study including 709 women with a lifetime ED (Fernández-Aranda et al., 2008). The findings suggested that ICD including CB occurred more often among persons with the binge eating subtype of ED. Taken together, it appears that patients with CB are

at higher risk to develop a BED and vice versa. Therefore, one may assume a close link between these two "consumption" disorders.

19.4 Compulsive Buying and Substance Use Disorders

As shown in Table 19.1, two controlled studies have suggested an increased occurrence of SUD during the life span of individuals with CB compared to healthy controls, with nearly every second person with CB reporting this association (Christenson et al., 1994; Mitchell et al., 2002). In contrast, two other studies found prevalence rates that did not differ from the control samples (Black et al., 1998; Mueller et al., 2009). However, the latter studies had investigated patients with CB that were included in treatment studies and did not suffer from any SUD in the past 6 months. Hence, the lower lifetime prevalence of SUD could be explained by the exclusion of individuals with current SUD.

The mixed results could also be explained by the heterogeneity of individuals with CB. There exists some evidence that more severe CB is associated with higher prevalence rate of SUD. The results of a latent profile analysis in a sample of 171 individuals with CB revealed two clusters (Mueller, Mitchell, Black, et al., 2010). The first cluster ($N = 64$) consisted of individuals with more severe CB and higher psychiatric comorbidity than the second cluster, in particular in regard to anxiety and affective disorders but also in regard to SUDs (30 % vs. 15 %, $p < 0.05$, respectively). This is primarily due to higher rates of alcohol and cannabis use. A suggestion of increased alcohol use was also reported in earlier studies investigating individuals with CB (Christenson et al., 1994; McElroy et al., 1994; Mitchell et al., 2002). Overall, one can assume that at least in a subgroup of persons, CB seems related to any SUD.

19.5 Compulsive Buying and Other Impulse Control Disorders

Previous data suggest a high comorbidity between CB and other ICD. This is not surprising given the assumption that CB can be classified as an ICD. Table 19.1 presents the findings of the few studies conducted thus far that found elevated rates of other lifetime ICD in individuals with CB compared to healthy controls. The most common ICD reported were intermittent explosive disorder, gambling, and kleptomania (Christenson et al., 1994; McElroy et al., 1994; Mueller et al., 2009; Schlosser et al., 1994).

Other literature also suggests that CB is more prevalent among individuals with other ICD. For example, in a sample of 40 persons with pathological gambling, 25 % suffered from comorbid CB (Specker, Carlson, Christenson, & Marcotte, 1995). Another study suggested that 63 % of costumers of a fitness room tended to CB (Lejoyeux, Avril, Richoux, Embouazza, & Nivoli, 2008).

19.6 Treatment of Compulsive Buying

The development of treatments for CB must be viewed as in its infancy. Very few controlled trials have been done, and those that have been completed have small sample sizes.

Case reports have suggested the efficacy of SSRIs (Koran, Aboujaoude, Solvason, Gamel, & Smith, 2007), the narcotic antagonist naltrexone (Grant, 2003), and the NDMA receptor antagonist memantine (Grant, Odlaug, Mooney, O'Brien, & Kim, 2012). However, randomized controlled trials have been fairly consistent in showing a surprising and generally unequivocal lack of efficacy for medications (Steffen & Mitchell, 2011).

Relative to psychotherapy approaches, although various strategies have been suggested in the literature, controlled psychotherapy studies have included only three small trials (Mitchell, Burgard, Faber, Crosby, & de Zwaan, 2006; Mueller, Mueller, Silbermann, et al., 2008; Müller, Arikian, de Zwaan, & Mitchell, 2013). These trials all used the same cognitive behavioral approach, delivered in a group format. The therapy is a fairly didactic manual-based approach with frequent homework assignments. The manual focuses on problems with failure of self-regulation, as well as low self-esteem and depressed mood. Stimulus control techniques are used, as well as the development of alternative behaviors and cognitive restructuring. Although small in sample size, the three published studies have consistently shown significant improvement in CB on all major outcome measures. Of particular noteworthiness, in all three trials, improvement was well maintained at 6 months' follow-up. One of these trials also included a telephone-based guided self-help approach as one of the treatment arms, which appeared to be superior on some variables to the waiting list control, but overall not as successful as the group CBT approach (Müller, Arikian, de Zwaan, & Mitchell, 2013). As CB is typically associated with increased psychiatric comorbidity, patients with comorbid disorders were not excluded from the studies. The disorder-specific treatment did not lead to a significant improvement in compulsive hoarding, but had a positive effect on depressive symptoms.

Conclusion

It is generally acknowledged that CB is a significant psychiatric problem that causes marked distress and is associated with substantial psychiatric comorbidity. CB is not only relevant to mood and anxiety disorders and compulsive hoarding but also to binge eating-type disorders, SUD, and ICD. Due to small sample sizes, putative sampling bias, and the different methodologies used in the comorbidity trials, the available data should be viewed as preliminary and interpreted with caution. CBT appears to be the most promising treatment approach thus far. Given the high psychiatric comorbidity and social impairment seen with CB, there is a need for the development of specific psychotherapy approaches that take the comorbid disorders into account.

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Muscle Dysmorphia: Where Body Image Obsession, Compulsive Exercise, Disordered Eating, and Substance Abuse Intersect in Susceptible Males

20

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Abstract

A growing number of adolescent and adult males are dissatisfied, preoccupied - even impaired by concerns about their physical appearance. Feelings of discontent and insecurity can lead to disordered eating and substance abuse when males compare themselves against popularized cultural standards of attractiveness and see themselves falling short. When body image dissatisfaction, compulsive exercise, and food intake dysregulation combine and intensify, risk increases for a psychiatric condition identified within the past 20 years known as muscle dysmorphia, a disturbance of self-perception in which individuals are obsessively preoccupied with the belief they are insufficiently large or muscular. Muscle dysmorphia shares characteristics with eating disorders (such as persistent attention to intake and compensatory behaviors focused on control of weight/shape), obsessive–compulsive disorder (excessive body monitoring and physical activity), and body dysmorphic disorder (including extreme attention to outward appearance). The condition is frequently associated with anabolic androgenic steroid abuse, which may exacerbate an observed exercise dependence or compulsive drive for muscularity. Therapeutic approaches are guided by the individual symptom picture with respect to issues such as obsessional thoughts/compulsive behaviors, mood dysregulation, and anxiety, and have

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principally been derived from clinical studies examining body dysmorphic disorder, for which both psychotropic medications and cognitive behavioral therapy are considered first-line treatment. Nutrition education and support is also warranted. More research is needed to increase understanding regarding the unique pressures and profound subjective distress males can experience with respect to accepting their bodies, as well as to develop better approaches to assessment and treatment of this complex condition.

Keywords
Anabolic-androgenic steroids • Bigorexia • Binge-eating disorder • Body dysmorphic disorder • Eating disorder • Exercise • Males • Muscle dysmorphia • Obsessive-compulsive disorder • Substance use disorder

20.1 Eating Disorders in Males

The diagnosis of eating disorders (ED) in males is on the rise. In clinical settings, estimates currently range from 5 to 10 % of all patients with anorexia nervosa (AN), 10–15 % of patients with bulimia nervosa (BN), and 40 % of binge-eating disorder (BED) cases (Nunez-Navarro et al., 2012), while one US population-based study reported prevalence in males may be as high as 25 % of all ED cases (Hudson, Hiripi, Pope, & Kessler, 2007). This may reflect both an increased recognition of food-related behavioral dysfunction as well as an increased willingness of men to seek help. Prior to the recent release of the 5th edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) (American Psychiatric Association, 2013), 90 % of men diagnosed with EDs were classified as eating disorder not otherwise specified, suggesting female gender-biased criteria for the two principal diagnoses (Stanford & Lemberg, 2012). Specifically, absence or delay of menses was a requirement for identification of AN, while compensatory behaviors described for BN, including abuse of laxatives, are more likely to be employed by women (Button, Aldridge, & Palmer, 2008; Nunez-Navarro et al., 2012), with no mention of the muscle-building or thermogenic agents often abused by men preoccupied with body size and shape.

Since relatively fewer individuals who seek treatment are male, there are significantly more ED facilities for female patients, and programs usually tailor their approach to meet the needs and concerns of this population. Limited treatment access and less-specialized attention may lead many young and adult men to defer or evade treatment entirely. The possibility that adolescent and adult males postpone seeking help until they are experiencing significant psychological distress or functional impairment is consistent with a study by Hackler, Vogel, and Wade (2010) in which males reported lower expectations of anticipated benefits from ED treatment than their female counterparts. According to Tanofsky et al. (1997), men diagnosed with binge-eating disorder present for treatment with a greater history of comorbid Axis I psychopathology, including mood dysregulation and anxiety. And

in a recent study from the UK, males in a therapeutic setting had difficulty admitting their disorder due to fear of a negative reaction, consistent with the suggestion that food-related difficulties are perceived as a female issue (Robinson, Mountford, & Sperlinger, 2013).

In addition to social stigma, males face all the common obstacles to ED recovery: co-occurring mood, anxiety, or substance use disorders (SUD), compulsive exercise, past adverse treatment experiences, and a history of trauma, including sexual abuse and weight-based victimization (Weltzin et al., 2012; Woodside et al. 2001). Evidence suggests men can be as preoccupied with outward appearance as women (Edwards & Launder, 2000), and their level of impairment from dysregulated eating similar (Striegal, Bedrosian, Wang, & Schwartz, 2012); however, differences in ED presentation have also been reported, such as the nature of body image concerns (e.g., overvaluation of muscle mass) and practices related to their involvement in sports (Muise, Stein, & Arbess, 2003). In a study conducted at a major treatment center in Brazil (Scagliusi et al., 2009), male patients had fewer negative attitudes (i.e., feelings) about food and eating, suggesting they consider the act of eating more natural and positive than their female peers. Anorexic men had lower scores than bulimic men for eating attitudes, while the opposite was observed for women. The authors posited an explanatory role for societal values regarding thinness, speculating that women who restrain intake successfully are more likely to feel they have overcome food and report feeling less, rather than more, controlled by it. While greater value is placed on dietary discipline for women in many cultures, consistent with reported feelings of satisfaction and success by some anorexic females (Scagliusi et al., 2009), dietary restraint for men has also become increasingly popular in the past several decades through the establishment of the bodybuilding culture. Feelings of shame can occur when individuals of either sex compare themselves against present-day cultural standards of attractiveness and perceive themselves as falling short of the ideal, exacerbating ED symptoms (-Wiseman & Moradi, 2010).

Gender-based differences have been reported for gastrointestinal hormones associated with hunger (Beasley et al., 2009), brain structures involved in hedonic (pleasure-based) and homeostatic (maintenance of physiological equilibrium) appetite control systems (Horstmann et al., 2011), and food-related social and environmental cues (Rolls, Fedoroff, & Guthrie, 1991), suggesting there may be certain fundamental differences in neurobiological drivers of eating behavior in men vs. women. Investigators examining differences in ED behavior and prevalence among males vs. females have also suggested gender-specific associations with risk factors related to education, nationality, age (Alfano, Hildebrandt, Bannon, Walker, & Walton, 2011), and body-checking behavior (Forrester-Knauss & Stutz, 2012). With regard to psychopathology, Nunez-Navarro et al. (2012) found male study subjects with an ED had lower scores for depression, anxiety, interpersonal sensitivity, and somatization than their female counterparts.

Disordered eating behaviors often present differently in males and females. Men are less likely to engage in laxative abuse (Nunez-Navarro et al., 2012) or to resort to fasting or self-induced emesis (Striegel-Moore et al., 2009) and have been

described as engaging in half as many compensatory behaviors overall (Jackson & Grilo, 2002). De Young, Lavender, and Anderson (2010) reported that binge eating in males was associated with exercise-related behavior, regardless of whether they were trying to control their weight. For these men, physical activity tended to be aimed at caloric expenditure, muscle development, or other alterations of body composition. For both genders, perceptions of body image and weight are often more important than reality for those engaging in disordered eating (Eichen, Conner, Daly, & Fauber, 2012). Dissatisfaction with or misperception of body image is a common characteristic of most EDs and is of particular importance for men with muscularity concerns, suggesting that mental health professionals are likely to see more men with disordered eating as the standard of attractiveness for the male body is increasingly centered on muscular physique.

Sexual orientation has also received significant attention as a correlate of ED in males. In one report, homosexual subjects showed greater body dissatisfaction and ED symptomatology compared with their heterosexual counterparts (Kaminski, Chapman, Haynes, & Own, 2005). These authors found gay men diet more, were more fearful of becoming fat, and were less satisfied with their degree of muscularity, but did not differ in the degree to which they exercised or experienced guilt about missing a workout. The authors hypothesized this group may experience more body dissatisfaction because they engage in more social comparisons than heterosexual men, while other researchers have suggested gay men are more vulnerable to media influences regarding body image (Carper, Negy, & Tantleff-Dunn, 2010). Although the drive for muscularity appears to be a prevalent theme across all subgroups of males, there are data which suggest the drive for thinness may be more common among gay men (Carper et al., 2010). The aggregate of these reports suggests a need for further study regarding potential metabolic, hormonal, psychiatric, and psychosocial contributions to intake behaviors associated with each gender, as well as for developing and evaluating gender-specific approaches to ED treatment and related comorbidities.

20.2 Eating Disorders and Co-occurring Substance Abuse in Males

A gender-based interaction between eating behavior and substance abuse has also received attention. In an early examination of male ED subjects, Tanofsky et al. (1997) reported men with BED have a greater frequency of substance abuse. Barry, Grilo, and Masheb (2002) likewise found men who engage in binge eating have a greater concurrence of SUD (and were more likely to be obese), while women were more likely to report using overeating, rather than substance abuse, as a coping strategy for negative emotions. Barry and Petry (2009) also found an increased risk for lifetime alcohol abuse and dependence in overweight men, but not women. The link between food intake behaviors and substance use in normal weight vs. overweight adolescents was recently examined by Eichen et al. (2012). Using data from the 2007 National Youth Risk Behavior Survey, they found the use

of tobacco, cocaine, or binge drinking was strongly associated with disordered eating behavior. With regard to purging behavior in the overweight group, smoking and binge drinking significantly predicted purging in female subjects, while cocaine use was significantly associated with purging in males. Overall, these findings suggest a need for different weight and behavioral management practices across BMI categories and gender, highlighting the importance of targeted education for individual subgroups.

Stanford and Lemberg (2012) found many men uncovered symptoms of EDs during the process of addiction treatment, consistent with previously documented high rates of chemical dependency in ED populations (Root et al., 2010; Weltzin et al., 2012). Certain populations may show different prevalence; for example, Feldman and Meyer (2010) reported gay and bisexual men with an ED were more likely to have comorbid SUD than gay and bisexual men without a similar diagnosis. Co-occurring SUD is not limited to street drugs and may include supplements such as so-called fat burners, anabolic-androgenic steroids (steroids), and other performance-enhancing drugs (Eisenberg, Wall, & Neumark-Sztainer, 2012). Further investigation into the interaction of food and substance intake patterns may inform new gender-specific treatment approaches for disordered eating, particularly for men who use substances to enhance muscle mass.

20.3 Muscle Dysmorphia: Overview

Male dieters typically think of themselves as dieting for legitimate reasons, such as improved health (Souza & Ciclitira, 2005). However, adolescent and adult males are not immune to out-of-control behaviors stemming from their pursuit of a physical ideal. When body image dissatisfaction, compulsive exercise, and disordered eating combine and intensify, risk increases for a psychiatric condition first described by Pope and Katz in 1994 in a report on male bodybuilders known as muscle dysmorphia. The authors originally used the term "reverse anorexia," referring to their observation of a single-minded desire to gain, rather than lose, weight. Other authors have referred to the drive to have a more robust and athletic body as "bigorexia" (Mosley, 2008), a term more often seen informally in popular press reports.

Muscle dysmorphia is a disturbance of body image perception in which individuals become obsessively preoccupied with the belief they are too small, too thin, or insufficiently muscular, although these individuals are actually more likely to have a higher proportion of lean body mass than the average person. Additional concerns about the appearance of individual body parts are not uncommon. Associated behaviors typically include a rigorous weight-lifting regimen, a special diet focused on high protein and energy intake spread across six or more meals daily, compulsive mirror-checking, comparison with others, and, perhaps somewhat paradoxically, often going to great lengths to avoid public body display. Some individuals demonstrate insight regarding their distorted beliefs, but are

nevertheless not reassured by objective recognition of their muscularity, while others are convinced they look much smaller than others of similar size, leading to considerable impairment and distress (Pope, Gruber, Choi, Olivardia, & Phillips, 1997).

Muscle dysmorphia resembles obsessive–compulsive disorder, where the obsessional thoughts center on muscularity and the compulsive behaviors include rigorous dietary rituals, excessive exercise, self-inspection, and reassurance seeking. Since the focus is on body image, muscle dysmorphia is considered to be a specific form of body dysmorphic disorder (BDD). Nearly 30 years ago, Veale (1987) suggested the term "exercise dependence" to describe the related phenomenon of compulsive physical activity, proposing diagnostic criteria with biomedical (e.g., symptoms of tolerance and withdrawal) and psychosocial (interference with social/occupational function) features (See Chap. 7). The supporting studies did not involve muscle development, focusing only on aerobic exercise. In the most recent (10th) revision of the International Statistical Classification of Diseases and Related Health Problems (ICD-10-CM), BDD remains classified within the somatoform disorders category, where it was also found in the DSM-IV-TR. In the DSM-5, muscle dysmorphia is included in the section on obsessive–compulsive and related disorders within the description of BDD, consistent with suggested links between these two disorders in recent brain morphology research (Atmaca et al., 2010).

One of the earliest links between muscle dysmorphia and EDs was described in a report by Pope et al. (1997) in which 22 % of male subjects with characteristics of muscle dysmorphia also formerly met diagnostic criteria for AN, having "replaced their earlier preoccupation with being too fat with being too small." The same authors also described another group of male subjects with muscle dysmorphia in which 13 % had a history of AN and 13 % formerly met criteria for BN, and among a sample of 32 female competitive bodybuilders with features of muscle dysmorphia, 47 % reported a history of either AN, BN, or both (Pope et al., 1997). The EDs resemble muscle dysmorphia in a number of important ways: both involve a compulsive preoccupation with perceived physical inadequacies and abnormal intake habits, as well as characteristic compensatory behaviors such as attempts to hide or cover the defects and excessive exercise. Not uncommonly these individuals avoid activities involving eating due to fear of disrupting their diet, or forgo personal relationships as well as occupational opportunities which might interfere with the time needed to focus on working out or food preparation.

Since many of the presenting characteristics of muscle dysmorphia overlap with AN and BN, including body dissatisfaction and low self-esteem, some authors have suggested similar approaches to patient assessment be used, with close scrutiny to uncover rules governing eating behaviors, black-and-white thinking, and frequent body checking (Grieve, Truba, & Bowersox, 2009). The suggestion was made to classify muscle dysmorphia within the eating disorder unspecified category in the DSM-5, since the two share many features and some individuals with muscle dysmorphia have a comorbid ED, but this lacked sufficient scientific support and was ultimately dropped. According to a study by Goldfield, Blouin, and Woodside (2006) examining competitive male bodybuilders (a related population) and males

with BN, while the two groups share many disordered behaviors, including excessive weight/shape preoccupation, extreme body modification practices, and binge eating, they share relatively few general psychological similarities. The authors also pointed out that excessive exercise in the form of strength training is typically the exclusive form of compensatory behavior in the former group, since bodybuilders often avoid cardiovascular training due to a fear of muscle atrophy.

Following a carefully planned diet, taking supplements, and engaging in rigorous resistance-training is a common strategy for men to build mass, but has also been observed in women who want to be more muscular and toned (Robert, Munroe-Chandler, & Gammage, 2009). However, while both men and women can struggle with muscularity concerns (Greenberg & Schoen, 2008), muscle dysmorphia is observed almost exclusively in males, as noted in the DSM-5 entry (American Psychiatric Association, 2013), although exceptions have been found for subsets of the female bodybuilding population (Hitzeroth, Wessels, Zungu-Dirwayi, Oosthuizen, & Stein, 2001; Pope et al., 1997). Early signs can be identified in some adolescent males, with physique-enhancing behaviors such as altered eating and methodical/strenuous exercise, as well as use of protein powders, steroids, and other muscle-building agents. In this age group, high prevalence has been observed in male high school students of Asian background, in overweight/obese adolescents, and in individuals involved in competitive athletics (Eisenberg et al., 2012). Preoccupation with dietary intake in males is especially common in sports where participants are divided by weight class, such as wrestling, horse racing, and mixed martial arts. Where weight is not a determinant for eligibility, but aesthetics and agility remain important, such as in marathon running, gymnastics, and diving, the emphasis often shifts from body weight to percentage body fat (Kollei, Schieber, de Zwaan, Svitak, & Martin, 2013).

Moving to a lower weight class can create a competitive edge. Athletes in sports with strict weight classes may drastically modify their eating patterns to accomplish this, restricting intake before weigh-in, followed by compensatory eating immediately after in an effort to improve strength and endurance before competing (Lambert & Jones, 2010). Aggressive dieting that restricts specific foods is a well-known risk factor for craving and rebound binge eating, and athletes are not immune from this outcome (De Bruin, Woertman, Bakker, & Oudejans, 2009). Nutrients targeted for restriction typically include high-starch carbohydrates/sugars, sodium, and water, which can lead to reduced glycogen stores and extracellular water retention. Some diets may even include a planned binge episode for muscle anabolism, or simply to relieve stress. While many of these athletes would have been considered subclinical for an ED based on DSM-IV-TR criteria, the new DSM-5-based diagnosis for BED may capture a larger percentage of this population. Other well-known strategies for weight loss in athletes include cardiovascular training with special gear or garments to promote diaphoresis as well as use of diuretics, laxatives, and saunas prior to weigh-in. Compromised fluid volume is a significant health risk and has been fatal in extreme cases (Franchini, Brito, & Artioli, 2012). When clinicians are aware of these aggressive weight management

strategies, they may be more effective in screening for muscle dysmorphia and intervening before a clinically significant ED develops.

Some researchers have also reported observing an increase in relatively extreme dietary practices among nonathletic males seeking to modify their physical appearance to achieve a desired body image (McCreary, Hildebrandt, Heinberg, Boroughs, & Thompson, 2007). In attempting to mimic the behaviors of professional athletes, many of these individuals receive nutritional guidance from muscle magazines, online bodybuilding forums, and the sports supplement industry, investing in fitness products, ergogenic nutrients, and gym memberships with the goal of achieving an ideal body. Similar to the fashion industry's controversial use of underweight models, the fitness industry may rely on unrealistic imagery to engender body insecurity in its customer base.

20.4 Muscle Dysmorphia and Steroid Abuse

Muscle dysmorphia is frequently associated with various types of substance abuse—most notably, appearance and performance-enhancing drugs (APED) (Hildebrandt, Langenbucher, Lai, Loeb, & Hollander, 2011). Steroids, classified as schedule III controlled substances by the Anabolic Steroid Control Acts of 1990 and 2004, are the most widely sought after and abused APED. Dependence syndromes and progression to other recreational drugs are potential long-term consequences, consistent with a report by Kanayama, Hudson, and Pope (2008), which found that 35 % of male steroid users met lifetime criteria for an SUD. In a more recent Internet-based study of study of male steroid users ($n = 508$) vs. nonusers ($n = 771$), 23.4 % of the former group met criteria for SUD vs. 11.2 % for nonusers (Ip, Barnett, Tenerowicz, & Perry, 2011). The use of narcotics in steroid users was examined by Arvary and Pope (2000) who found a significant percentage of male heroin addicts living at a treatment facility ($n = 227$) used opioids to counteract associated depression and withdrawal following steroid use. Other studies have also suggested a link between steroids and opioids (Kanayama et al., 2008). In view of the estimation that subclinical levels of muscle dysmorphia may affect a significant number of men (Grieve et al., 2009), one benefit of early symptom detection in this population may be to reduce escalation to abuse of steroids and other substances.

Medical uses for steroids include treatment of disease states involving muscle wasting, such as HIV-AIDS and cancer, osteoporosis, and, most commonly, to increase low testosterone levels in men secondary to hypogonadism. Individuals seeking to improve athletic performance may turn to steroids to increase fat-free mass, strength, and endurance, as well as reduce body fat. Recreational bodybuilders are less likely to use steroids than competitive bodybuilders (Blouin & Goldfield, 1995; Goldfield et al., 2006), but interest and use have been increasing in the former population. Steroids are often used in conjunction with thyroid

hormones, pain medications, and other drugs such as fertility medications designed to counter their negative side effects (McCreary et al., 2007). There are also reports of combining steroids with sports supplements such as creatine or legal and illegal prohormones which enter the marketplace regularly with little or no regulation by the FDA, often not even listed on the product label (Cafri et al., 2005).

Adverse effects of supraphysiological doses of steroids include acne, impaired reproductive function, gynecomastia, (Casavant, Blake, Griffith, Yates, & Copley, 2007; McCreary et al., 2007), and increased risk for cardiovascular disease secondary to atherosclerosis, thrombus formation, and hypertension (Kanayama et al., 2008). Psychiatric complications include mood dysregulation, anxiety, and aggression (Casavant et al., 2007; McCreary et al., 2007), as well as withdrawal symptoms such as variable energy level (fatigue or restlessness/insomnia), reduced libido, and depression (Rohman, 2009), which may include suicidal ideation (Wong, Zhou, Goebert, & Hishinuma, 2013). Beaver, Vaughn, DeLisi, and Wright (2008) reported that young adult males who use steroids have a high frequency of violent behaviors, suggesting steroid use may complicate treatment for individuals who already have a history of struggling with anger, trauma, and post-traumatic stress (Mitchell, Mazzeo, Schlesinger, Brewerton, & Smith, 2012). Steroid users often find it difficult to discontinue once their athletic or appearance goals are met, in some cases accelerating use as new goals are set or progressing to other substances, perpetuating the cycle of body dissatisfaction.

Although steroid use can reach the level of dependence seen with many recreational drugs (Kanayama, Brower, Wood, Hudson, & Pope, 2010), the behavioral effects and neurobiological drivers remain poorly understood. One working hypothesis is that steroids may amplify the exercise dependence seen in everyday fitness seekers and, under a certain circumstances, be an integral part of a dysfunctional and perfectionistic obsession with muscularity (Hale, Roth, DeLong, & Briggs, 2010; Szabo & Griffiths, 2007). In individuals who meet criteria for muscle dysmorphia, there is a unique pathological intersection of distorted self-perception, exercise compulsivity, disordered eating, and abuse of performance-enhancing agents. When the psychiatric picture is complicated by habitual and entrenched substance abuse, addiction treatment may be the necessary first step (Rohman, 2009). An overview of muscle dysmorphia, from specifics regarding the behavioral presentation to diagnostic symptoms to proposed therapeutic strategies, is outlined in Fig. 20.1.

20.5 Treatment Approaches for Muscle Dysmorphia

20.5.1 Nutrition Guidelines

Achieving lean muscularity has become something of an ideal to attain for both sexes, although females tend to focus on thinness, while males are more likely to place greater value on body mass (Edwards & Launder, 2000). With respect to medical nutrition therapy, gender differences in the Dietary Reference Intakes, especially with regard to protein intake, are not considered significant enough to warrant

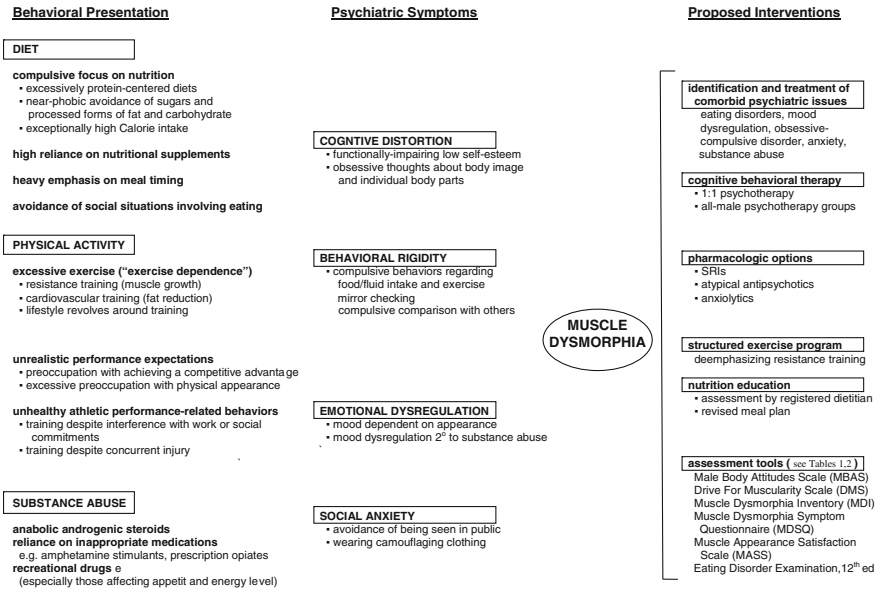

Fig. 20.1 Overview of muscle dysmorphia, from the behavioral presentation to diagnostic symptoms to proposed therapeutic strategies

separate guidelines for nutritional intervention (Food and Nutrition Board. Institute of Medicine of the National Academies, 2005). Dietary needs are best assessed individually, based on eating behavior, physical activity, lab tests, and other indices of physiological status. For individuals who meet criteria for muscle dysmorphia, in addition to cessation of hypertrophy training, reduction or elimination of excessive sports supplements, including protein/amino acids, creatine, and pre-workout formulas is an important goal. Reducing overall protein intake, which reportedly can be as high as 3 g/kg bodyweight (Mosley, 2008), may encounter resistance from many bodybuilders who believe this will lead to muscle atrophy. Arguably the most critical objective is to avoid the diet-related extremes driven by the constant preoccupation regarding body image that characterizes these individuals.

Assessment is also guided by the psychological symptom picture with respect to food and eating behavior-related fears and misconceptions. Since women represent the majority of Registered Dietitians in the USA, some males may, rightly or wrongly, perceive communication barriers in diet interventions during treatment. Nutrition education including gender-specific components can begin to integrate a psychology-of-men perspective into the predominantly female ED therapeutic approach (Greenberg & Schoen, 2008). Following the model of other recovery communities, creating an alumni base to incorporate the insight and support of ex-bodybuilders and steroid users who are uniquely able to describe their previously distorted thinking in a manner that resonates with newly diagnosed patients could also prove helpful.

20.5.2 Physical Activity Recommendations

An important aspect of treatment for men with muscle dysmorphia is the work to educate about healthy body composition as well as normalize levels of body fat and muscle, shifting exercise goals away from an emphasis on extreme muscle mass towards more sustainable fitness. A period of abstinence from exercise may be important in the initial stages of recovery, after which activity can be added back slowly. Research supports the benefit of a guided exercise program during residential ED treatment (Calogero & Pedrotty, 2004; Hausenblas, Cook, & Chittester, 2008), and incorporation of graded exercise has been shown to increase short-term treatment compliance (Thien, Thomas, Markin, & Birmingaham, 2000). With knowledge about EDs and muscle dysmorphia, fitness trainers can be an important component of the treatment team by monitoring/evaluating goals and encouraging new activities that do not involve extreme weight lifting. With respect to curtailing dysregulated eating and excessive activity, getting patients to alter their rigid and familiar diet plan and abstain from compulsive exercise can prove to be a difficult process and may require inpatient hospitalization or residential treatment.

20.5.3 Psychiatric Considerations

The first barrier to treatment in muscle dysmorphia is usually identifying the disorder, since patients often look healthy from an outward perspective. As with AN, the disorder tends to be ego-syntonic; individuals see themselves as healthy and are not inclined to seek help. For males with disordered eating and an excessive preoccupation with body image, there is an additional social stigma associated with acknowledging the presence of a disorder. The biopsychosocial formulation is guided in part by the individual presentation with respect to issues such as obsessional thoughts/compulsive behaviors, mood dysregulation, substance abuse, and anxiety spectrum illness, including social phobia, each of which can complicate the treatment approach (Phillips, Gunderson, Mallya, & Carter, 1998). Also important to address are the potential impact of cultural pressures for adolescent and adult males to conform to advertising and media representations of the ideal body type (Baird & Grieve, 2006). Exploratory discussions may be effective in a group setting, where individuals can share long-held, and typically unhelpful, beliefs about gender stereotypes.

Despite the growing attention to muscle dysmorphia in recent years in comparison with other psychiatric conditions, such as EDs, where appearance preoccupation is central, a limited number of intervention strategies have been systematically studied (Pope, Phillips, & Olivardia, 2000). Consistent with its inclusion in the DSM-5 as a form of BDD, suggested therapeutic approaches for muscle dysmorphia have principally been derived from clinical studies examining body dysmorphia, for which both psychotropic medications and behavioral therapy are considered first-line treatment. Although no drugs are FDA approved for treatment of BDD absent sufficient empirical data, another justification for medication is the

frequent presence of multiple psychiatric comorbidities for which the efficacy of pharmacotherapy is well established. Pharmacologic treatment is also considered highly advisable for more severely ill or suicidal patients (Phillips, 2005a), given reported lifetime rates of suicidal ideation in individuals with BDD are approximately 80 %, and suicide attempt rates range from 24 to 28 % (Phillips & Diaz, 1997; Phillips & Hollander, 2008).

Originally targeted for treatment of depression and anxiety, the serotonin reuptake inhibitors (SRIs) have been found to lessen obsessional thinking and compulsive behaviors. No studies have compared different SRI doses for BDD, but higher doses are typically needed to treat obsessive–compulsive disorder (Allen & Hollander, 2005) and would likely be considered appropriate for muscle dysmorphia. In a review by Phillips and Hollander (2008), clinical practice recommendations for mean doses (in mg/day) were as follows: fluoxetine 67 ± 24, sertraline 202 ± 46, citalopram 66 ± 36, escitalopram 29 ± 12, fluvoxamine 308 ± 49, and clomipramine 203 ± 53, the authors underscoring patients often benefit from SRI doses exceeding the maximum recommended dose.

Pope et al. (2005) reported that when muscle dysmorphia occurs with more general BDD symptoms, including delusionality, subjects were more likely to engage in multiple compulsive behaviors and exhibit significantly greater psychopathology, most notably suicidality and substance abuse, including steroids. The SRIs remain the medication of choice whether patients demonstrate insight regarding perceived appearance defects or have delusional beliefs related to body image (Hollander et al., 1999; Somashekar, Jainer, & Wuntakal, 2013). Although delusional symptoms are typically treated with antipsychotics, multiple studies have found delusional BDD patients respond well to SRI monotherapy (Phillips, McElroy, Dwight, Eisen, & Rasmussen, 2001; Phillips, McElroy, Keck, Pope, & Hudson, 1994).

An SRI tried for 12–16 weeks with limited effectiveness, having been raised to the highest approved or tolerated dose for at least 3 weeks, should be switched to a different SRI or augmented with another medication (Phillips, 2005a). In one study, 43 % of patients who did not adequately respond to an initial adequate SRI trial responded to at least one subsequent SRI trial (Phillips, Albertini, Siniscalchi, Khan, & Robinson, 2001). Only a limited number of trials have examined SRI augmentation in BDD, and no methodologically rigorous studies have compared one agent to another or established an optimal time frame for an augmentation trial. Positive results have been reported with addition of both clomipramine (Phillips, Albertini et al., 2001) and buspirone (Phillips, 1996), but poor results were seen in a randomized trial with pimozide (Phillips, 2005b), suggesting first-generation antipsychotics may not be effective. Although well-recognized to modulate cognitive rigidity and delusional thinking, there are limited data describing addition of atypical antipsychotics, although one study showed a promising result with aripiprazole (Uzun & Ozdemir, 2010), suggesting further examination of these agents is needed.

Cognitive behavioral psychotherapy (CBT) is another viable treatment approach (Ipser, Sander, & Stein, 2009). Males tend to externalize emotional distress (Gjerde, Block, & Block, 1988) and are often less comfortable discussing their emotions and negative experiences. CBT can provide a framework for identifying and

Table 20.1 Instruments designed to evaluate how males evaluate satisfaction with their own body image

Male Body Attitudes Scale (MBAS) Tylka, Bergeron, and Schwartz (2005)
Masculine Body Ideal Distress Scale Kimmel and Mahalik (2004)
Drive for Muscularity Scale (DMS) McCreary and Sasse (2000)
The Appearance Anxiety Inventory Veale et al. (2013)
Yale-Brown Obsessive Compulsive Scale for BDD (BDD-YBOCS) Phillips et al. (1997)
Body Parts Satisfaction Scale for Men McFarland and Petrie (2012)
The Upper Body Strength Subscale of the Body Esteem Scale Franzoi and Shields (1984)
Shape and Weight Based Self-Esteem Inventory (SAWBS) Geller, Johnston, and Madsen (1997)
Eating Disorder Examination The Eating Disorder Examination (12th Ed.), (1993)
Eating Disorder Examination Questionnaire (EDEQ) Fairburn and Beglin (1994)

challenging misconceptions in thinking regarding food, weight, body image, and the compulsion to exercise, as well as to neutralize triggers, which perpetuate dysfunctional behaviors. No studies have directly compared the efficacy of CBT to pharmacotherapy, but the relative success of a psychotherapeutic approach would likely depend on a number of factors, including patient treatment preference, motivation, history of medication tolerance, and availability of qualified psychotherapists. Whatever the psychotherapeutic approach, addressing shame, anxiety, social avoidance, and body image concerns is arguably vital to the process of recovery. Concurrent medication treatment can make it possible for severely distressed, delusional, or suicidal patients to engage in and tolerate CBT. Under favorable circumstances, the treatments are complementary and mutually enhancing.

As prevalence rises and the presentation of body image disturbance in males becomes increasingly complex, there is a need for more sophisticated assessment tools. A summary of gender-specific measures for assessing self-image conflict and disordered eating in males is presented in Table 20.1. In the eating disorder assessment for men, for example, Stanford and Lemberg (2012) focus on the following core diagnostic issues: binge eating, disordered intake behaviors, body dissatisfaction, and muscularity concerns. While the majority of the scales highlight the importance of assessing the level of anxiety and distress that accompanies the process of striving to achieve a specific body type, a critical objective with each of these instruments is to distinguish a healthy focus on weight training, athleticism, and body image from the collection of obsessive thoughts and compulsive

Table 20.2 Instruments developed to assess symptoms for potential diagnosis of muscle dysmorphia

Muscle Dysmorphia Symptom Questionnaire (MSDQ) Olivardia, Pope, and Hudson (2000)
Muscle Appearance Satisfaction Scale (MASS) Mayville, Williamson, White, Netemeyer, and Drab (2002)
Muscle Dysmorphia Inventory (MDI) Rhea, Lantz, and Cornelius (2004)
Muscle Dysmorphic Disorder Inventory Hildebrandt, Langenbucher, and Schlundt (2004)

behaviors which may characterize pathological extremes of body dissatisfaction in BDD. Although scales and inventories designed to detect BDD are likely to provide useful information, caution should be used since these instruments may not necessarily be sensitive enough to detect the presence of muscle dysmorphia. A number of instruments that were developed to more directly evaluate muscle dysmorphia are included in Table 20.2.

Concluding Perspectives

Many studies in the past decade reveal a surprisingly high proportion of adolescent and adult males are dissatisfied and preoccupied—even impaired by concerns about their appearance (Knoesen, Thai Vo, & Castle, 2009). In muscle dysmorphia, the associated thoughts and behaviors are persistent, pathologically amplified, and associated with profound subjective distress. The condition shares many characteristics with EDs (including persistent and rigid attention to behavioral habits focused on control of body weight/shape/image), obsessive–compulsive disorder (e.g., compulsive body monitoring and physical activity), and body dysmorphic disorder (such as extreme preoccupation/dissatisfaction with outward appearance). Formerly a rare, or rarely recognized, occurrence, muscle dysmorphia may be an increasingly prominent psychiatric condition—a predilection for fitness resulting from cultural trends and attitudes regarding appearance which evolves into pathological behaviors among certain subpopulations. The condition is almost wholly exclusive to men, presumably given the greater cultural pressures for men to be muscular, as well as their greater genetic potential to achieve this objective. When the clinical picture includes other psychiatric comorbidities or involves abuse of anabolic steroids and other drugs, the potential for disrupting social and occupational functioning is significantly increased. More research is needed to uncover neurobiological and psychosocial drivers that may underpin the unique pressures males experience with respect to accepting or trying to perfect their physical appearance. Earlier recognition and acceptance of treatment by affected individuals will start with better tools for initial assessment, with sustainable recovery based on therapeutic strategies aimed at normalizing the self-destructive thoughts, emotions, and behaviors that characterize this complex and incompletely understood condition.

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Part III

Treatment Perspectives

Integrated Treatment Principles and Strategies for Patients with Eating Disorders, Substance Use Disorder, and Addictions

21

Amy Baker Dennis, Tamara Pryor, and Timothy D. Brewerton

Abstract

Currently, there are no evidence-based treatments or established treatment protocols for patients that present with both eating disorders and substance use disorders/addictions. The lack of available integrated treatment programs, at all levels of care, has left the dually diagnosed patient vacillating between these two disorders. Eating disorder treatment programs frequently exclude patients with active substance use disorders, and addiction programs regularly exclude or do not effectively treat patients with eating disorders. Often, these patients are referred to addiction treatment programs prior to entering into eating disorder treatment. This approach is problematic, as both disorders are associated with high rates of relapse following treatment. Sequential treatments focus on the most acute disorder first, often utilizing multiple providers in different locations, with different theoretical orientations, staff training, and treatment protocols, which can make continuity of care quite difficult. Developing a comprehensive integrated approach to the treatment of comorbid patients will improve treatment delivery, reduce time in treatment, lower overall treatment costs, improve treatment outcome, and lessen consumer confusion. This chapter will provide

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a definition, rationale, and the basic principles of an integrated treatment model with applicability to those working to develop and provide integrated services for these dually diagnosed patients.

Keywords
Addictions • Comorbidity • Dual diagnosis • Eating disorders • Integrated treatment • Levels of care • Substance use disorders

21.1 Introduction

Eating disorders (ED) and substance use disorders (SUD) co-occur at alarmingly high rates. Prevalence data suggest that upward of 50 % of individuals with an ED are also abusing prescription or over-the-counter medications, illicit drugs, and/or alcohol, which is five times the abuse rates seen in the general population (The National Center on Addiction and Substance Abuse (CASA) (CASA), 2003). Similarly, approximately 35 % of individuals seeking SUD treatment report eating pathology. Unfortunately, to date, there are no empirically supported treatments or established protocols for the integrated treatment of these two disorders. There are very few comprehensive treatment programs in either field that provide integrated services. A majority of treatments are provided either sequentially (i.e., treatment focuses on the most acute disorder first and then the secondary disorder is treated, often at different locations with different providers) or concurrently (i.e., different treatment providers and/or different programs provide each specialized service).

This chapter will begin by defining the general principles of integrated treatment and then outlines the potential advantages of providing this type of service to ED patients with SUD. Facility needs, staffing, and training recommendations will be followed by a discussion of how an ED partial hospital program (PHP) or intensive outpatient program (IOP) can modify their treatment protocol to more effectively treat patients with SUD. Likewise, a model for how current residential or PHP substance abuse programs could be modified to effectively treat ED patients will be described. The chapter will conclude with a discussion of the most common adaptive functions of substance use and abuse in eating disorder patients, and a case example will be presented.

21.2 What Is Integrated Treatment?

Based on several discussions in the literature on integrated treatments for patients with comorbid psychiatric conditions (Drake et al., 2001; Horsfall, Cleary, Hunt, & Walter, 2009; Kelly, Daley, & Douaihy, 2012; Mills et al., 2012; National Institute on Drug Abuse, 2012; Substance Abuse and Mental Health Service Administration, 2009), a working definition of integrated treatment for patients with ED and SUD will be presented. Services for patients with comorbid ED and SUD should be organized in a comprehensive and integrated manner which includes the following

elements (1) comprehensive screening (i.e., scientific, evidence-based screening, and assessment instruments) for ED, SUD, and any other co-occurring psychiatric disorders or medical conditions and relevant drug screenings and lab tests; (2) comprehensive, individualized treatment plans that encompass both the ED and SUD; (3) individual therapists and treatment teams that are highly trained in evidence-based treatments (EBT) for both disorders; (4) services that are provided in the same location by the same providers in a stepwise, integrated fashion; (5) if only one level of care is provided at the facility, then a plan for patient movement through the levels of care should be established with similar facilities that provide integrated services at different levels of care; and (5) if a facility is unable to provide a specific intervention "on-site," then a plan or contract is made with a specific provider to manage that service elsewhere (e.g., methadone maintenance, AA/NA meetings).

21.3 Why Are Integrated Services for Comorbid ED/SUD Patients Needed?

As mentioned previously, there are high rates of SUD in individuals with ED. It is argued that developing a comprehensive integrated approach to the treatment of comorbid patients will improve treatment delivery, reduce time in treatment, lower overall treatment costs, improve treatment outcome, and lessen consumer confusion. Sequential treatments (i.e., treatments that focus on the most acute disorder first) that often utilize multiple providers in different locations with different theoretical orientations, staff training, and treatment protocols can make continuity of care difficult (Helfman & Dennis, 2010).

Unfortunately, these disorders have the highest mortality rates across all mental illnesses. In a meta-analysis of 249 reports of mortality in individuals with mental illness, Harris and Barraclough (1997) found that individuals with anorexia nervosa (AN) and bulimia nervosa (BN) had rates of suicide that were higher than any other psychiatric disorders and 23 times higher than seen in the general population. In a multisite study, Bulik and colleagues (2008) reported that in all ED, anorexia nervosa, binge-purge subtype (ANBP), is associated with the highest risk of death. Likewise, women with alcohol use disorders (AUD) are 20 times more likely to commit suicide than the general population (Kessler, Borges, & Walters, 1999). When these two disorders co-occur, the risk for death can increase exponentially. Papadopoulos and colleagues (2009) conducted a study of 6,000 women in Sweden and found that AN subjects were 19 times more likely than the general population to have died from a substance use disorder, primarily AUD.

Recent studies have highlighted the lack of available services for comorbid patients. Two prominent studies in the addiction field have looked at assessment and treatment of co-occurring ED in publicly funded and privately funded programs. In a study by Gordon and colleagues (2008), 351 face-to-face interviews were conducted with program directors of publicly funded addiction treatment programs in the USA. Approximately 50 % of the programs screened for ED

upon intake. Of these, 29 % admitted all people with ED and 48 % admitted individuals with ED of low severity. Of the programs that did admit ED patients, treatment most often emphasized the medical-psychiatric model of addiction as opposed to standard EBT for ED (see Chap. 11). In 2011, Killeen and coinvestigators completed a similar study of 345 privately funded addiction treatment programs in the USA and found similar results. A majority of programs (74 %) screened for ED, and 67 % reported admitting cases of low severity. However, only 21 % of the programs attempted to treat the ED. Of these, researchers found that 17 programs (5 %) identified having some ED protocol, which included meal planning, supervised meals, bathroom monitoring, weighing, and self-monitoring (food journals). Ten programs (3 %) provided dietary and/or nutritional services, and eight programs (2 %) incorporated medical monitoring of the ED. Both studies concluded that most publicly and privately funded substance abuse treatment facilities neither treat nor provide multidisciplinary EBT for patients with ED.

To date, no studies have been conducted in the ED field assessing the availability of SUD services in ED programs. However, Dennis and Helfman (2010a) conducted a small pilot survey of 20 nationally known, long-standing ED inpatient, residential, and PHP/IOP treatment programs in the USA. The purpose of the survey was to determine how these programs assessed and treated patients with comorbid SUD. Fifty-five percent ($n = 11$) of programs reported providing *integrated* screening, assessment, and treatment of ED and SUD on-site. Of the remaining nine programs, four provided sequential treatment on-site (one disorder treated first and then the other), one program provided sequential treatment off-site (SUD treated first elsewhere followed by ED treatment), and four programs provided parallel treatments (both disorders treated at the same time but by different treatment providers or programs within the facility). However, one of the most interesting findings in the programs that reported providing *integrated* treatment was that only 3 of 11 programs had professionals that were specifically trained, certified, or licensed in the treatment of SUD and addictions. As a result, most programs surveyed were not equipped to treat patients requiring medical detoxification for either alcohol or drugs and did not provide certain psychopharmacological interventions that require additional training and DEA numbers. Although this was a very small, preliminary pilot survey of well-established ED treatment facilities, it suggests that the availability of fully *integrated* treatment for this comorbid group is also lacking in the ED field.

The lack of available integrated treatments leaves patients with both conditions vacillating between ED and SUD (Dennis & Helfman, 2010b). It is common for patients that are being treated for an ED to experience an increase in substance use and abuse or even relapse as they go through recovery. Likewise, patients that are being treated for their SUD can experience an increase in ED symptomology. Therefore, even programs that initially exclude patients that have these comorbid disorders may be confronted with the emergence or reemergence of an ED or SUD during the course of treatment. Effective treatment must address the relationship between the two disorders. For example, do ED behaviors (i.e., restriction, binge

eating, purging) trigger the SUD? Do these disorders occur concurrently? Or do they function in the service of each other (i.e., stimulant abuse to promote weight loss)? (See below for a discussion of the adaptive function of substance abuse in individuals with ED.) Complete recovery is not just abstinence from a substance or the normalization of eating patterns, weight gain, and/or the elimination of compensatory behaviors. Rather, it requires improving self-esteem and the development of coping skills and strategies to move beyond the illnesses to pursue a meaningful life. Integrated treatment providers promote and empower patients to delineate and attain their personal goals (Substance Abuse and Mental Health Service Administration, 2009).

Additionally, the lack of available integrated treatment programs is a problem not only for primary care physicians that encounter these patients but also for ED and SUD specialists from multiple disciplines that need to provide an appropriate referral. Do they send the patient with both disorders to an established SUD program or an ED program? Consumers (families and sufferers) often complain that when researching treatment options, they are confused about how to proceed. It is important that both professionals and consumers understand the complexities of these disorders and have confidence that patients will receive comprehensive and effective treatments.

Finally, patients that have received services in nonintegrated programs have poorer treatment outcomes (Drake et al., 2001). There is evidence in the substance abuse field that when comorbid diagnoses are treated concurrently and integrated on-site, treatment retention and outcome improve (Weisner, Mertens, Tam, & Moore, 2001).

21.4 Basic Guidelines and Principles for Integrated Treatment

In 2009, the Substance Abuse and Mental Health Service Administration (SAMHSA) published guidelines for the development of integrated treatment programs for co-occurring psychiatric disorders and SUD. In this chapter, their recommendations, principles, and guidelines have been adapted to address patients with ED and SUD.

Guidelines for Integrated Program Development

I. ***An integrated approach requires a specialized team of professionals that treat the comorbidities concurrently***. A multidisciplinary team that includes a medical professional (i.e., pediatrician, adolescent medicine physician, internal medicine physician, addiction medicine physician, and/or psychiatrist), a therapist, and a dietitian are the primary members of the team. However, other team members should be added depending on the specific needs of the patient (i.e., group therapist, occupational therapist, gynecologist for high-risk pregnancy patients, endocrinologist for diabetic patients, infectious disease physician for HIV/AIDS patients).

II. ***Integrated treatment specialists should be fully trained in evidence-based practices for both ED and SUD. Formal and informal cross-training between disciplines and clinical specialities is essential***. Unfortunately, there are no known specific training programs currently providing training and ongoing supervision for clinicians interested in this dually diagnosed population. However, treatment providers that are currently specialists in the field of ED can become certified and licensed through their state to become *Certified Addiction Counselors* (CAC). The CAC program trains addiction counselors as generalists who acquire a body of knowledge, skills, training, and supervised work experience to incorporate evidence-based practices currently being utilized in the addiction treatment field. A Level III CAC has passed a national examination and has the authority to practice independently and assume clinical supervision duties. The American Psychological Association also provides specialized training for members, and participants can earn a *Certificate of Proficiency in the Treatment of Alcohol and other Psychoactive Substance Use Disorders*. In addition to state certification or licensure, credentialing programs offered by the Association for Addiction Specialists (i.e., NAADAC), including *The National Certified Addiction Counselor* and the *Master Addictions Counselor*, are available. Several professional organizations (National Association of Addiction Treatment Providers, NAAPT; American Academy of Addiction Psychiatry, AAAP; American Society of Addiction Medicine, ASAM) also provide training programs and continuing education credits at national and regional conferences.

Also recommended for an integrated ED/SUD treatment program is a psychiatrist that is trained to treat ED as well as board certified in addiction psychiatry. The American Board of Medical Specialties (ABMS) has implemented multifaceted Maintenance of Certification (MOC) requirements to verify the competence of physicians to provide best practices for the assessment and treatment of patients with SUD (Duffy et al., 2011).

Another useful training tool for ED treatment providers who do not have a comprehensive understanding of the philosophy or vernacular of AA/NA or do not fully understand the complexities of the 12-step program is to read the 12-Step Facilitation Manual (Nowinski, Baker, & Carroll, 1992) and attend a meeting as a professional guest. Most AA communities have this service available to health and mental health professionals who are interested in becoming more familiar with the meeting process (Ries, Galanter, Tonigan, & Ziegler, 2011). 12-Step Facilitation is an important EBT approach for SUD (see Chaps. 12, 27, and 29). Treatment providers should also familiarize themselves with other resources specific to their community that support SUD recovery (i.e., Women in Sobriety).

For treatment providers that are substance abuse specialists with little training in the treatment of ED, there is only one formal certification process in the ED field. The International Association of Eating Disorder Professionals (iaedp) provides training and certification for therapists (CEDS, Certified Eating Disorder Specialist), dietitians (CEDRD, Certified Eating Disorder Registered Dietitian), and nurses (CEDRN, Certified Eating Disorder Registered Nurse). In

addition, there are numerous educational opportunities including written materials, webinars, and conferences offered through the Academy for Eating Disorders, the National Eating Disorder Association, and other national ED organizations.

Finally, this complex and often difficult comorbid patient population can become extremely overwhelming to treatment providers. Internal cross-training, staff development, and collaborative, routine case consultation and supervision can provide the specialized integrated team members with ongoing learning to ensure effective treatment and continuity of care, provide peer support, and prevent burnout.

III. ***Motivational interventions are used to assist patients at all stages of treatment but particularly during the precontemplation and contemplation stage***. Motivational interviewing is not only a counseling tool but also an interpersonal process designed to help patients explore and resolve ambivalence and strengthen their desire and commitment to change (see Chap. 22). Patients with comorbid ED and SUD often express varying degrees of readiness to change depending on what behaviors are being discussed. For example, the individual may be highly motivated to eliminate cocaine abuse but extremely reluctant to gain weight. Providing a collaborative therapeutic climate infused with motivational interventions helps patients overcome resistance and empowers them to take responsibility for behavior change.

IV. ***Cognitive behavioral therapy is the cornerstone of treatment for ED and SUD during active treatment and relapse prevention***. Although there are no specific EBT for this comorbid population, there is an extensive body of evidence for the use of cognitive behavioral treatment (CBT) for each population separately (see Chaps. 11, 12, and 25). It is also considered an EBT for many other psychiatric conditions that are often present in patients with SUD and ED (i.e., mood disorders, anxiety disorders, obsessive–compulsive disorder (OCD), post-traumatic stress disorder (PTSD), personality disorders).

V. ***Multiple modalities for services are available***, ***including individual***, ***group***, ***family***, ***nutritional***, ***and self-help***. Patients benefit when multiple formats are available to them during the treatment and recovery process.

Finally, in another recently published multicenter randomized controlled efficacy trial in adults with AN (Zipfel et al., 2014), researchers compared two manualized outpatient treatments (CBT-E and focal psychodynamic therapy) to optimised treatment as usual. At the conclusion of 10 months of treatment, body mass index increased in all groups but there were no significant differences between treatments, including at 12 month follow up.

Individual and group behavioral therapies involve addressing the motivation to change and help patients build skills to replace substance abuse and ED behaviors with self-enhancing behaviors and constructive tools to assist in managing negative affect, low self-esteem, and interpersonal problems. Family involvement is often essential for both disorders during the treatment and recovery process. In both the ED and SUD field, EBT for families and couples have been identified (see Chap. 26). Nutritional education and counseling, meal

supervision, and food management are important components of an integrated program (see Chap. 23). Also, participation in peer support groups during and following treatment can help patients maintain abstinence from substance abuse.

VI. ***Medications are integrated with psychosocial interventions and often are an important component of treatment for patients with comorbid ED and SUD***. There are several medications that have been found effective in helping individuals with SUD stabilize their lives and eliminate drug use. Likewise, there are psychotropic medications that have been found effective in the treatment of ED and other comorbid conditions that are often present in patients with ED and SUD (see Chap. 15).

VII. **Principles for effective treatment**

The National Institute on Drug Abuse (2012) has developed principles for the effective treatment of SUD. These have been adapted to the treatment of patients with co-occurring ED and SUD.

(a) ***Eating disorders***, ***SUD***, ***and addictions are complex but treatable conditions that affect brain functioning and behavior***. Both ED and SUD alter the brain's structure and function, resulting in changes that persist even after recovery and may help to explain the high rates of relapse in both conditions (see Chaps. 3, 4, 5, and 6).

(b) ***No single treatment is appropriate for all individuals***. Psychosocial, pharmacological, and nutritional interventions must be tailored to the needs of the individual. It is essential to match treatment settings, interventions, and services to an individual's specific needs and problems.

(c) ***Treatment needs to be readily available***. Individuals with ED and SUD are often reluctant to seek treatment. Treatment needs to be available, readily accessible, and affordable. Early intervention often predicts improved treatment outcomes.

(d) ***Effective treatment attends to multiple needs of the individual***, ***not just the ED/SUD***. There are significantly high rates of other co-occurring medical and psychiatric disorders in patients with ED and SUD. Medical complications from both the ED and SUD should be monitored and treated. Mood and anxiety disorders, OCD, PTSD, and personality disorders frequently co-occur in these individuals and should be treated in an integrated way.

(e) ***Remaining in treatment for an adequate period of time is critical for treatment effectiveness***. Research suggests that most addicted individuals need at least 3 months in treatment to eliminate their substance abuse and that the best outcomes occur with longer durations in treatment. Likewise, treatment of ED, particularly AN, is often protracted and can take from 4 to 7 years to attain full recovery (Strober, Freeman, & Morrell, 1997). Relapse is not uncommon and recovery from an ED and SUD is a long-term process that may require multiple treatment exposures.

(f) ***Counseling (individual, family, group, and nutritional) and other behavioral therapies are critical components of effective treatment for both disorders***.

(g) ***Medications are an important element of treatment for many patients, especially when combined with counseling and other behavioral therapies***.

(h) ***An individual's treatment and services plan must be assessed continually and modified as necessary to ensure that the plan meets the person's changing needs***. Ongoing assessment can provide valuable information to treatment providers and can alert them when the level of care needs to be changed.

(i) ***Medical detoxification is only the first stage of addiction treatment and, by itself, does little to change long-term drug use. Likewise, weight restoration, the normalization of eating patterns, and the elimination of compensatory behaviors is only the first stage of recovery from an ED***. Patients with both disorders remain at risk for relapse long after the amelioration of symptoms. To ensure lasting change, psychosocial interventions that focus on problem solving, coping strategies, affect regulation, interpersonal relationship strategies, and relapse prevention are essential.

(j) ***Treatment does not need to be voluntary to be effective***. SUD and ED affect both the mind and body. Patients that are seriously compromised because of their ED or addiction may refuse help. Family interventions; sanctions by employers, criminal justice system, colleges or universities, or athletic organizations; or civil commitment by a significant other can be effective means of persuading a seriously ill individual into treatment (see Chap. 30).

(k) ***ED-related behaviors and drug use during treatment must be monitored continuously***. Ongoing monitoring of drug use and ED behaviors can be a powerful incentive to help patients resist using drugs or engaging in compensatory behaviors. Integrated programs need to provide ongoing medical monitoring of the ED (i.e., weight, lab tests) as well as the SUD (i.e., breathalyzer tests, urinalysis). Additionally, many patients will require supervised meals and bathroom and exercise monitoring. Self-monitoring (food and personal journals) are also helpful components of a comprehensive intervention.

(l) ***Patients should be tested for the presence of HIV/AIDS, hepatitis B and C, tuberculosis, and other infectious diseases, and targeted risk-reduction counseling should be provided***. These services should be provided on-site instead of being referred for off-site testing to increase the likelihood that results will be received while they are active in treatment and appropriate interventions are provided if they test positive.

21.5 Levels of Care for Integrated Programs

Eating disorders and SUD are complex psychiatric disorders that are heterogeneous in etiology and clinical presentation. As a result, this population varies widely and no single treatment approach or level of care can effectively or efficiently accommodate the diverse clinical needs of these patients. When determining the level of care, it is important to consider several factors including the patient's current physical condition, psychological status, behaviors, substance abuse/addiction, and social circumstances rather than simply rely on weight status (Yager et al., 2010). This continuum of care is fluid and patients can move back and forth through the levels depending on their specific needs. Additionally, the length of stay in each level of care is not static and depends on severity of illness and progress/response to treatment. When patients move between levels of care, it is important to establish continuity of care. Programs that provide integrated ED/SUD services at only one level of care should identify integrated programs at other levels to ensure a smooth transition between treatment locations.

Guidelines for determining the appropriate level of care have been independently outlined for patients with ED (American Psychiatric Association, 2006; Yager et al., 2010) and patients with SUD (American Psychiatric Association, 2007; Gastfriend & Mee-Lee, 2011). Below are some basic recommendations on how an integrated treatment program could optimally utilize these levels of care for patients with comorbid ED and SUD.

A. **Level 0.5**: **Early Intervention**. This is considered a pretreatment level of care for individuals who have risk factors or problems associated with ED/SUD. Interventions are designed to help the individual recognize the negative consequences of their ED/SUD and gain skills and strategies to avoid future problems. School, university, and community evidenced-based prevention programs can be an important component of early intervention if the information disseminated helps individuals develop skills to resist negative social influences, enhances competence, is provided within an ecological context, and is engaging and interactive for all participants (see Chap. 20).
B. **Level I**: **Outpatient Treatment**. This level of care usually consists of one or two weekly sessions of individual, family, nutritional, and/or group sessions for individuals with less severe symptoms of ED/SUD or for individuals who need continued support for ongoing recovery. The focus of treatment is lifestyle, attitudinal, and behavioral changes that are necessary to reduce the negative consequences associated with ED/SUD or issues that could promote relapse. Outpatient family-based treatment (FBT) for adolescents below 85 % (but above 70 %) of their estimated healthy weight has been found effective in producing weight gain for some patients (Lock, Agras, Bryson, & Kraemer, 2005); however, significantly underweight adolescents and adults often require a higher, more structured level of care to gain weight. It is often important to provide ongoing monitoring of drug use (i.e., breathalyzer tests, urinalysis) and ED behaviors (i.e., food journals, weight, compensatory behaviors, medical

status) in this level of care. Attendance at AA/NA or other self-help groups is frequently encouraged.

C. **Level II: Intensive Outpatient/Partial Hospitalization**. Intensive outpatient treatment (IOP, 9–25 h per week) or partial hospitalization programs (PHP, up to 85 h per week) provide extended mental health services during the day, after work, in the evenings, and on weekends; however, patients reside at home or in supervised housing. This level of care is designed to provide a higher intensity of programming, contact with clinical staff, and support for individuals with ED/SUD than traditional outpatient treatments. The basic services provided by this level of care include medical, psychiatric, nutritional, and psychopharmacological consultation, medication management, and 24-h crisis services. Individuals with ED/SUD may enter directly into the IOP/PHP level of care or step down from a residential or inpatient treatment program to continue the recovery process.

IOP/PHP Integrated Treatment Model for ED Programs

Systematic assessments of the quality of general medical and specialty mental health care in the USA indicate that patients with SUD receive the poorest overall quality of care when compared with treatment for other conditions (McGlynn et al., 2003). These findings underscore the fact that clinical assessments and treatment of co-occurring SUD/ED represents a tremendous opportunity to improve clinical practice in disorders associated with high morbidity and mortality. A comprehensive assessment for SUD and ED should begin with an initial telephone screen to determine whether it is appropriate to schedule a full diagnostic assessment. If the person calling (i.e., potential patient, outside mental health professional, or family member) indicates any evidence of medical instability or active suicidality, it is recommended that the identified patient be assessed at the nearest emergency room before proceeding with a full assessment for ongoing treatment. If the patient is determined to be medically stable and motivated to proceed with assessing care, then the patient is sent a medical clearance form to be completed by a licensed practitioner. A comprehensive assessment is scheduled to take place either by phone or in person with the potential patient and an admission clinician. This evaluation includes assessment of the following:

1. Detailed history of past and present eating disordered behaviors and substance use, associated problems, symptoms, and types and amount of substances used, last use, and history of withdrawal
2. Effects of ED, SUD, and related comorbid disorders on cognitive functioning
3. Effects of ED, SUD, and related comorbid disorders on psychological and behavioral functioning (i.e., academic or job performance or interpersonal relationships)
4. Effects of ED, SUD, and related comorbid disorders on physiological functioning
5. History of all psychiatric and nonpsychiatric treatments and outcomes
6. Family, developmental, and social histories

7. Risk to self and others, including aggressive, suicidal, or other self-injurious behaviors
8. Assessment for signs and symptoms that would suggest a need for medically managed withdrawal or detoxification
9. Determine a preliminary diagnoses based on a thorough ED/SUD history
10. Release of information forms signed to obtain records from previous treatment providers

After the assessment has been completed and all records are received and reviewed, the admissions team comprised of the Clinical Director, Medical Director, and Admissions Clinician determine whether the potential patient meets criteria for admission and treatment. Within the first 48 h, the patient receives a history and physical by the in-house primary care physician, a psychiatric evaluation by a certified addiction medicine physician, a psychological assessment, a family therapy assessment, a nutritional assessment, and an initial meal plan. The meal plan is developed to ensure normalization of eating behaviors. Evidence-based psychosocial interventions and pharmacotherapy are described and offered, and the patient and team (which include a certified addiction therapist) create the initial treatment plan. Drug screening is explained, breath alcohol testing frequency is determined, and an order is written for the patient to be assigned to the ED/SUD track of the program.

To promote or maintain sobriety during integrated treatment, it has been indicated that treatments that bolster motivation for change and adherence to therapy and improve coping skills and mood regulation benefit patients with coexisting SUD and ED (Grillo, Sinha, & O'Malley, 2002; Marcus & Levine, 2004). Groups for comorbid patients are often delivered in a specialized addiction track that covers the following content: basic psychoeducational information; a review of treatment models; biopsychosocial risk factors; definitions of recovery; the relationship and interplay between ED and SUD (i.e., symptom substitution); stages of change (i.e., how one may be in a different stage for ED or SUD); decisional balance (looking at the benefits of continuing to engage in ED and SUD behaviors vs. the consequences and risks); identifying triggers/cravings; practicing strategies for symptom interruption and urge management; and building coping, problem solving, assertiveness, affect regulation, distress tolerance, and effective interpersonal communication skills.

D. **Level III**: **Residential/Inpatient Services**. This level of care is provided to individuals that require 24-h, supervised "live-in" care to prevent imminent danger or the negative consequences of continued ED/SUD. Residential services include medication management, counseling, nutritional interventions, psychoeducation, and skills building to help individuals safely transition to less restrictive levels of care.

Residential/Inpatient Integrated Treatment Model for Addiction Programs

In order for an addiction program to treat ED patients at this level of care, several alterations and additions to the existing program would have to be implemented, including the development of a structured behavioral program

geared toward (1) normalization of eating behaviors, (2) weight restoration (in patients with AN), and (3) response prevention of compensatory behaviors (i.e., dieting/restricting, bingeing, purging, and excessive exercising). Three meals and three snacks per day at regular intervals become the cornerstones of supportive behavior therapy, or meal behavior therapy, since at this level of care, patients often require intense structure and empathic supervision at all meals and snacks. Nutritional needs for patients with ED should be managed by a dietitian familiar with incremental weight restoration and normalization of eating pattern. Meals and snacks are calibrated to the patient's individual treatment goals. During meals and snacks, nursing staff check to make sure patients are getting the specific types and amounts of foods prescribed, model healthy eating behaviors and attitudes, offer psychological support when needed, and intervene when necessary to prevent ED behaviors, such as hiding or taking food; cutting food into little pieces; talking negatively about food; talking about calories; using excessive amounts and/or unusual combinations of condiments, drinking water, carbonated, or caffeinated beverages excessively; binge eating; purging; or moving excessively. A major challenge on a unit with both ED and non-ED patients is to have sufficient dually trained staff to adequately meet the needs of all. At this level of care, patients are generally medically stable to the extent that they do not need intravenous fluids, daily laboratory tests, or nasogastric tube feedings. In addition, patients are usually not overtly suicidal, are generally able to cooperate within a structured environment, and are able to ask for support when needed.

E. **Level IV**: **Medically Managed Intensive Inpatient Services**. This level of care is reserved for individuals that have severe ED and/or substance-related problems that require 24-h medical management (metabolic stabilization) and/or medically supervised detoxification services. In addition, patients may have additional severe psychiatric complications including psychosis, delusions, or homicidal and/or suicidal ideations. Staffing consists of addiction medicine physicians, skilled nursing staff, a dietitian, and other mental health clinicians who provide specialized biomedical, psychiatric, and nursing services. Patients at this level of care require intensive medical assessment and treatment, which may include daily laboratory measurements, parenteral fluids and medicines, heart monitoring, and/or nasogastric or nasoduodenal tube feedings.

21.6 Adaptive Function

One extremely useful clinical tool in the treatment of patients with ED and SUD is to explore the adaptive function of the patient's symptoms and behaviors. Understanding the social, psychological, physical, or interpersonal problems that the ED/SUD manages or solves and identifying the secondary gains that result from engaging in these behaviors can provide the clinician with insight into the predisposing, precipitating, and perpetuating factors that influence, initiate, and maintain the disorders, respectively. In other words, these behaviors may serve an

underlying purpose or function: they answer a question, solve a problem (seemingly), temporarily fulfill an unmet need, or alter the environment in a positive way (despite negative consequences). In most cases, the patient is unaware of how these behaviors are related to underlying issues. Often these symptoms are difficult to relinquish, and discovering the adaptive function can guide the patient toward the development of healthier coping strategies and methods of problem solving. A brief review of adaptive functions that are commonly found in ED is detailed in the following section. Understanding the adaptive function can also inform case formulation, team constellation, goals for treatment, and the selection of appropriate levels of care, therapeutic modalities, and treatment approach.

21.6.1 Common Adaptive Functions Found in Eating Disorders

ED symptoms and behaviors are far more complex than just serving as a method to lose weight or improve body image. Underlying psychological problems are frequently displaced onto food, weight, and body issues. Regulating food intake, body size, and weight brings a personal sense of mastery and control into a world that is often perceived as unmanageable. Bruch (1973) was an early clinical investigator to note that ED patients often experienced a "profound sense of ineffectiveness." Rigid control of food intake and weight is often believed to either cause, offset, or prevent some life event or change a mood state (e.g., prevent parental divorce, enhance athletic prowess, attract a romantic partner, delay attendance at college, deter sexual abuse, reduce anxiety or negative affect) (Dennis & Sansone, 1997). (For further information on adaptive function in ED and related conditions, see Brewerton, 2004; Costin, 2007; Dennis & Sansone, 1997; Johnson, Sansone, & Chewning, 1992.) In addition, the work of Persons and others (Persons, 2005; Persons & Tompkins, 2007; Zayfert & Becker, 2007) also emphasizes the importance of understanding the functional links and causation of behavior when developing case formulations and implementing evidenced-based treatment for complex patients.

21.6.1.1 To Manage Maturity Fears

ED most commonly develop in adolescence. One of the primary developmental tasks of adolescence is to separate and individuate from the primary caretakers. If the individual feels unprepared to separate from the family, or is fearful of relying on their ability to navigate the world without assistance, he or she may engage in "practicing" behaviors that promote autonomy and mastery. Taking charge of one's body is one method of demonstrating mastery and asserting control while simultaneously avoiding maturation. Limiting food intake, engaging in strenuous exercise, eliminating the menses, and driving their weight down virtually guarantee not having to separate from the family (see Crisp, 1967, 1980, 1997). Parents are less likely to send their critically ill adolescent off to college if they are unable to manage the basic task of eating. Thus, the adaptive function of the ED successfully allows the individual to manage his or her maturity fears by remaining attached, in a

seemingly safer and more predictable environment, until he or she feels sufficiently ready to tackle the world independently.

21.6.1.2 To Manage Sexual Conflicts

Adolescence is the initiation of both physical and sexual maturation. Changes in the intensity and quality of drive activity, changes in appearance and biological capacity, as well as the sexualization of peer attachments can be overwhelming challenges for some adolescents. Self-induced starvation retards normal growth and development and flattens out hormone profiles, thereby eliminating all of the secondary sexual characteristics of adulthood. Again, by taking control of the body, there is a return to the prepubescent biological state where the adolescent is no longer threatened by either external attention by peers or internal biological drives (see Leon, Lucas, Colligan, Ferdinande, & Kamp, 1985).

This adaptive function is utilized in various forms by patients of all ages and all types of ED. It is not uncommon for individuals that have experienced sexual trauma or those wrestling with gender identity or sexual orientation issues to engage in self-starvation, binge eating, and purging to manage sexual conflicts.

21.6.1.3 To Provide Structure, Predictability, and Control

Today's world is replete with rapid-fire decision making, monthly technological changes, and environmental stresses and demands that challenge even the most adept. Unfortunately, many individuals with ED also suffer from cognitive rigidity and anxiety and are harm avoidant and resistant to change (see Brewerton, Hand, & Bishop, 1993; Holliday, Tchanturia, Landau, Collier, & Treasure, 2005). The ED can provide the individual with a world that is structured and predictable. Dieting is a national pastime that many engage in but few are successful. Being successful requires sacrifice, commitment, and diligence. To obtain a sense of control, individuals with ED take charge of the body and structure their world by counting calories, engaging in regular rigorous physical activity, eliminating previously desired foods, avoiding activities that might interfere with their routines, and making a commitment to a long-range goal. For example, "If I exercise for 2 h every day, eat less than 800 calories a day, avoid anything that contains fat, purge anything that is a 'forbidden food,' I can successfully reach my weight loss goals." These behaviors produce measurable results, which reinforce the belief that "I am powerful, in control, and can achieve something that most people can't." In a world that seems unpredictable and "out of control," engaging in ED behaviors provides a sense of mastery and control.

21.6.1.4 To Consolidate a Self-Identity

Another important developmental task of adolescence is to cultivate a system of values, internalize self-esteem, and develop a stable and cohesive self-identity. Adolescents strive to be unique even within their peer group. AN is a rare illness and girls/women with AN are unique; they "stand out" in a crowd. Their mere presence in a room frequently attracts attention and concern. There is a sense of pride in achieving a goal (weight loss) that is admired by most, even if the ED

behaviors compromise physical and emotional health (see Tan, Hope, & Stewart, 2003). The glorification of thinness and the glamorization of ED among high profile celebrities, musicians, and athletes have contributed to the belief that having an ED is not "so bad." Unfortunately, they are consolidating a self-identity around a serious psychiatric illness.

21.6.1.5 To Get Attention or Help

It is not uncommon to discover that the ED is a "smoke screen" for other issues in the individual or within the family. ED symptoms and behaviors can speak louder than words. One very effective way of drawing attention to systemic problems is to lose a considerable amount of weight or engage in bouts of binge eating and purging. In many cases, this will spark a trip to a health and/or mental health professional that will explore and identify the underlying issues or systemic problems.

21.6.2 Adaptive Function of Substance Use in Eating Disorder Patients

This section will explore the adaptive function of substance use in patients with ED. Several of these adaptive functions are applicable to ED patients without SUD (i.e., to escape, avoid, and numb; to punish the negative self; and to manage mood and anxiety disorders) but are also common in comorbid patients. Understanding the underlying motivation behind the initiation of substance use in ED patients can assist the treatment provider in case formulation and treatment planning.

21.6.2.1 To Control Appetite, Manage Hunger, and/or Promote Weight Loss

Often, the adaptive function of substance use in our patients is in service of the ED. In other words, the problematic use of substances starts after the onset of the ED and is designed to assist the individual in suppressing appetite, restricting intake, and maintaining or promoting weight loss. A variety of substances have been abused for these purposes including OTC laxatives, diet pills, diuretics, emetics, caffeine, and nicotine; prescribed medications including insulin, thyroid hormone, and ADHD medications (i.e., psychostimulants); and illicit drugs, primarily central nervous system stimulants like cocaine and methamphetamine.

When the adaptive function of the SUD is in service of promoting weight loss or controlling appetite, treatment providers need to educate their patients on the relative ineffectiveness of OTC medications and the serious medical complications and potential side effects of abusing prescribed medications or illicit substances. If prescription medications are being abused, it requires consultation with all prescribing physicians, and it is often requested that they become an ongoing member of the treatment team.

21.6.2.2 To Manage Uncomfortable Physical Symptoms

ED patients often complain of feeling fat, bloated, or physically uncomfortable when they attempt to normalize their food intake. Some of these symptoms may be attributed to gastroparesis (delayed gastric emptying). Most frequently seen in AN patients, gastroparesis can cause nausea, vomiting, and gastric fullness. Laxatives, diuretics, emetics, and herbal teas or supplements can provide temporary relief from painful physical symptoms; however, gastroparesis can only be resolved through normalizing eating patterns and weight gain.

Clinicians are encouraged to fully educate their patients about the uncomfortable physical and psychological symptoms associated with the normalization of eating patterns. Predicting these symptoms *before* they encounter them, emphasizing their temporary nature, and assuring the patient that these symptoms will ameliorate with proper hydration and food intake often help the individual tolerate the discomfort.

21.6.2.3 To Decrease Sexual or Physical Shame or Discomfort

Some individuals with ED feel socially and/or sexually awkward, while others have significant body image disturbance or suffer from body dysmorphic disorder (BDD). Several different substances can reduce social anxiety and body consciousness. Opioids which reduce both physical and emotional pain, central nervous system depressants that reduce anxiety and promote relaxation, marijuana and alcohol that decrease inhibitions, stimulants that enhance physical sensations, and hallucinogens that alter conscious reality have all been used in service of reducing sexual or physical shame or discomfort.

If the adaptive function of the substance abuse is to reduce body dissatisfaction in order to increase the individual's ability to engage in interpersonal relationships or participate in social activities, treatment should consist of cognitive restructuring to develop alternative body schemas, exploration of self-identity, and strategies to improve self-care and self-esteem.

21.6.2.4 To Enhance Performance

Individuals with ED also use performance-enhancing drugs such as anabolic steroids. In addition to significant organ damage, these drugs have adverse psychological effects (e.g., aggression and irritability, depression, mood swings, altered libido, addiction, and psychosis). Unfortunately, alcohol or substance use is frequently increased to diminish these negative side effects.

If anabolic steroid abuse is discovered, medical intervention to manage detoxification and severe withdrawal effects is essential. Inpatient treatment may be necessary if the patient is experiencing severe physical problems or becomes psychotic. Recovery from steroid abuse is often long term, and, in addition to individual therapy or milieu treatment, patients are encouraged to attend self-help groups like Narcotics Anonymous (Giannini, Miller, & Kocjan, 1991).

21.6.2.5 To Manage Addiction

The adaptive function of continued substance abuse in some ED patients is to manage their addiction and prevent withdrawal. Once an individual becomes

dependent on alcohol or drugs, they continue using in order to prevent the severe side effects that accompany abstinence. Severe alcohol, sedative-hypnotic, or opioid abuse often requires medically managed detoxification, which should be supervised by trained addiction specialists. If the ED patient is medically stable and not at risk for refeeding syndrome, medically managed withdrawal is recommended prior to treating the ED (see Chap. 15).

21.6.2.6 To Escape, Avoid, or Numb

One of the most common reasons ED patients use substances is to alter their current state of consciousness. Unresolved interpersonal problems, difficulties at work or school, family conflict, domestic violence, sexual abuse, post-traumatic stress, teasing, or bullying, all can lead to the use of drugs/alcohol to help individuals numb themselves from negative feelings and/or to avoid intolerable life circumstances or traumatic memories. Alcohol, marijuana, opioids, central nervous system depressants, and stimulants all have mood-altering effects that can help the individual distance themselves from their current reality.

Obviously, if the adaptive function of the substance abuse is designed to emotionally remove the individual from painful environmental situations, therapy must focus on helping the patient confront and resolve these roadblocks. Cognitive restructuring and strategies to increase self-esteem, distress tolerance, assertiveness, and problem solving can assist the individual in developing less self-destructive methods of coping with and managing difficult life struggles.

21.6.2.7 To Punish the Negative Self

ED and/or substance abuse behaviors can be used as a method of self-punishment or a way of managing excessive guilt (see Berghold & Lock, 2002). This adaptive function is most frequently seen in individuals with severe low self-esteem and trauma histories and individuals with borderline personality features. These behaviors can be used to reaffirm the individual's entrenched belief that they are worthless or unlovable, which reinforces their negative self-concept and low self-esteem (e.g., "Good things don't happen to bad people. Bad people need to be punished."). ED behaviors including severe restriction ("I don't deserve to eat."), binge eating ("Weight gain will allow me to disappear and I will no longer be a target."), self-induced vomiting ("I need to get all the evil out of me."), chronic use of laxatives ("I deserve to experience severe cramping, nausea, and painful diarrhea."), and excessive exercise ("Bad people deserve to be in pain.") can all be used in service of self-punishment. Likewise, any psychoactive substance can be used to punish the negative self.

This patient subgroup frequently has significant psychopathology with dysregulation in multiple areas of their lives (e.g., cognitions, affect, impulses, interpersonal relations, sleep and feeding patterns). They regularly engage in multiple self-destructive behaviors, which can include polysubstance abuse, shoplifting, cutting, burning, self-mutilation, violence, gambling, driving while intoxicated, parasuicidal behaviors, and sexual promiscuity. Treating this population requires a thorough understanding of psychopathology, the ability to manage

and treat multiple diagnoses simultaneously, a long-term professional commitment to the patient, and ongoing professional supervision (Brewerton, 2004; Dennis & Sansone, 1989, 1991, 1997). Antidepressant medications or mood stabilizers may be helpful in reducing self-destructive behaviors in these patients.

21.6.2.8 To Manage Mood Disorders

There are high rates of comorbid unipolar and bipolar affective disorders in both ED and SUD. In some instances, depressive symptoms are evident *prior* to the onset of the ED or SUD (primary mood disorder). Wildman, Lilenfeld and Marcus (2004) found that major depression preceded the onset of an ED in 33 % of their female participants. Abraham and Fava (1999) reported that AUD followed the first depressive episode by 4.7 years and cocaine use occurred 6.8 years after the onset of depression. They conclude that alcohol and cocaine use in depressed individuals is often a form of self-medication. Similarly, researchers have suggested that individuals with bipolar disorder may use alcohol during the manic phase to either prolong pleasurable sensations or reduce irritability and agitation (Sonne & Brady, 2002). Additionally, several licit and illicit drugs can *cause* symptoms of depression (e.g., alcohol, barbiturates, opioids, and stimulants when withdrawn) or mania (e.g., steroids, opioids, amphetamines, and thyroid hormone). Furthermore, an ED can lead to depression as evidenced by the classic semi-starvation study conducted by Ancel Keys (Keys, Brozek, Henschel, Mickelsen, & Longstreet, 1950). In this study, dramatic changes in emotional states including increased depression and anxiety were found in previously mentally healthy subjects as food restriction and weight loss progressed.

It is extremely important, therefore, to screen for comorbid mood disorders during the assessment phase of treatment. If the mood disorder was present *prior* to the onset of the ED, psychotropic medications may be an important consideration when developing the treatment plan. Antidepressant medications for some patients with BN or BED have been found effective in not only reducing depressive symptoms but may also reduce binge eating and purging episodes. It is essential that patients are properly educated about how to take their antidepressants, since purging or the use of other central nervous system depressants (e.g., alcohol, barbiturates, benzodiazepines, opiates) can render the medication ineffective. Stabilizing mood, and the elimination of SUD particularly in individuals with bipolar disorder, can improve the likelihood of recovery from an ED.

Unfortunately, antidepressants have not been found effective in managing depressive symptoms or promoting weight gain in patients with AN. The atypical antipsychotic, olanzapine, is the only psychotropic medication shown in randomized, controlled trials to be effective for weight gain and/or reduction of psychological symptoms, such as depression and obsessional anxiety, in patients with AN (Brewerton, 2012). Other than possibly olanzapine, medications to manage depressive symptoms in AN patients should generally be avoided until acute medical problems have been resolved and significant weight gain has taken place. If the patient continues to have significant depressive symptoms after the normalization of eating patterns and weight restoration, then pharmacologic intervention

should be considered. Depressive symptoms often ameliorate with proper nutrition, the reduction/elimination of purging behaviors, and/or weight gain, unless major depression was a primary disorder.

Empirically supported interventions such as CBT and IPT have been found effective in the treatment of both mood disorders and bulimic ED. Self-monitoring tools such as food diaries can provide valuable information about eating patterns, hunger and satiety, binge-purge episodes, and situations and feelings associated with disordered eating patterns. With substance abusers, self-monitoring sheets can be utilized that have been modified to track drug/alcohol use in addition to ED behaviors. Becoming aware of distressing events, problematic interpersonal interactions, and/or uncomfortable moods states and their relationship to episodes of binge eating/purging and substance abuse can help patients monitor their progress in treatment.

21.6.2.9 To Reduce Stress or Manage Anxiety

It has been well established that anxiety disorders including social anxiety, OCD, PTSD, phobia, and panic disorder are common comorbid conditions in individuals with ED (Hudson, Hiripi, Pope, & Kessler, 2007). In a great majority of cases, anxiety disorders develop before the onset of the ED (Brewerton et al., 1995; Bulik, Sullivan, Fear, & Joyce, 1997; Deep, Nagy, Weltzin, Rao, & Kaye, 1995; Godart, Flament, Lecrubier, & Jeammet, 2000; Kaye, Bulik, Thornton, Barbarich, & Masters, 2004). Likewise, anxiety disorders and SUD co-occur at very high rates. In a national survey of over 43,000 participants, researchers discovered that individuals with anxiety disorders were three times more likely than the general population to develop a substance use disorder and that individuals with SUD were three times more likely to have anxiety disorders at some point in their life (Grant et al., 2004). In some instances, substance use begins as a way to self-medicate symptoms of anxiety (primary anxiety disorders). Central nervous system depressants tend to be the drugs most frequently used to reduce anxiety. In other cases, anxiety and panic attacks can be *caused* by substance abuse (substance-induced anxiety). Long-term alcohol use can cause neurological changes in the brain that increase both anxious and depressive symptomology. In addition, alcohol/drug withdrawal can cause or exacerbate anxiety symptoms thus creating a "vicious cycle" in which the original purpose of the drug (to reduce anxiety) now becomes the cause of the anxiety.

In ED patients with comorbid anxiety disorders, efficacious treatments include CBT, IPT, and behavioral interventions (e.g., exposure with response prevention). In some instances, these interventions can significantly reduce anxiety symptoms without the introduction of psychotropic medications. In other instances, benzodiazepines or antidepressants that have antianxiety effects are needed to reduce severe anxiety.

In summary, primary mood/anxiety and substance-induced disorders are all serious conditions that require attention. Unfortunately, most treatment providers and substance abusers alike have long assumed that comorbid mood and anxiety disorders were usually substance-induced, and once the SUD was treated, the

depression/anxiety would disappear. However, Grant and colleagues (2004) provide extremely persuasive evidence for the high prevalence of *primary* mood and anxiety disorders among individuals with SUD.

This research highlights the importance of a comprehensive assessment in ED patients, not only to determine the extent of the substance use but also to determine if medications could be beneficial to the recovery process. The nonphysician clinician will need to consider the psychiatrist or primary care physician as an integral part of the treatment team when there is comorbid depression or anxiety requiring psychotropic medications.

21.7 Case Example

21.7.1 Presenting Problem

JC is a 20-year-old heterosexual white female. She is a sophomore in college and recently took an academic leave to attend treatment. Her outpatient team referred her to PHP for the treatment of restricting anorexia nervosa and substance abuse. JC has had multiple courses of ED treatment over the past several years. A review of her medical clearance form revealed that she tested positive for opiate and alcohol use, but her tests for the presence of HIV/AIDS, hepatitis B and C, tuberculosis, and other infectious diseases were negative.

21.7.2 History of Presenting Problem

JC stated that her disordered eating began at age 13 with restricting behaviors in response to a negative body image, a negative self-image, and a desire to be more popular. As a child, she was small for her age, socially awkward, and a tomboy. She reported being teased about her appearance.

Commentary

- *Explore with the patient any negative comments he or she has received about body size or shape.*
- *Is there any evidence of teasing or bullying*?
- *Was the patient ever overweight or obese*?

She described herself as a perfectionist and stated that when she did not feel as though she was "succeeding," for example, in classes or as a violinist, restricting made her feel "productive." She also enjoyed the positive feedback she received from her thin appearance. JC also hypothesized that her disordered eating was tied to family problems and her profound sense of responsibility for others, especially

her fraternal twin brother. However, when questioned about her brother, she stated that he is well adjusted, well liked, and "good at everything." She reports that shortly after discovering her father's infidelity, her parents separated and she began to lose weight.

Commentary

- *Explore with the patient all important interpersonal relationships as well as premorbid personality traits.*
- *How do these relationships/traits influence the patients' behaviors*?
- *What is the connection between these relationships/traits and the onset and maintenance of the ED and SUD*?

Regarding active ED behaviors just prior to admission, JC reported consuming only small portions of food such as a yogurt or one quarter of a sandwich. She also reported drinking approximately 64 oz of diet coke per day and would go up to 2 or 3 days without consuming any food. She denied any other current compensatory behaviors including self-induced vomiting, excessive exercise, or laxative use to control appetite or weight; however, she did admit to briefly using (i.e., 2 months) over-the-counter diuretics but denied use in the past year.

21.7.3 Substance Use

JC reported that she started using marijuana at 16 years old and began experimenting with ecstasy, ketamine, benzodiazepines, opiates, cocaine, LSD, and alcohol at "around 18 years old." She noted that she began using heroin at age 19 and that this is her current drug of choice. JC administers heroin intravenously. She reports being socially isolated, lonely and "sad" during middle and high school. In search of an accepting peer group, she began using drugs and spending time with others that did the same. Prior to admission, she used approximately one gram/week and spent $60–80 per week. She endorsed withdrawal symptoms including physical weakness, nausea, akathesia, and sleep disturbance. She reported that she has been able to reach sobriety lasting 4 months but that she repeatedly relapses. She noted that her problematic substance use "plagues" her, and she experiences strong guilt and shame as a result. She was able to briefly discuss a few of the negative consequences of her substance use including a rape when she was intoxicated, stealing money from, and lying to her parents. Additionally, she had to withdraw from college due to poor performance and went from being a straight "A" student to an average student.

Commentary

- *Explore with the patient the "adaptive function" of the substance abuse.*
- *What precipitated the use of drugs?*
- *What was her family's reaction to her substance use?*
- *What were the consequences of the substance use? Entanglements with the law? Academic, social, interpersonal, or family consequences?*
- *What happens to the frequency and intensity of ED symptoms during abstinence from substances?*
- *What happens to the frequency and intensity of the substance use with the reduction of ED symptoms?*

21.7.4 Diagnosis at Intake

A complete family assessment was conducted, and a history of OCD, mood disorders (including major depression and bipolar disorder), and alcoholism was identified in JC's relatives. Her initial diagnosis was anorexia nervosa—restricting subtype, opiate use disorder, and mood disorder—not otherwise specified.

21.7.5 Course of Treatment

JC was admitted to a PHP with additional nighttime supervised housing. Following medically supervised detoxification, she was able to participate in all program activities. Overall, JC made progress in normalizing her eating patterns and was able to interrupt restrictive behaviors. She restored from 68 % of expected weight to 92 % of expected weight and restored her menses. She was able to develop insight into the adaptive function of ED/SUD symptoms and verbalize connections between uncomfortable emotions, cognitive distortions, and behaviors. While JC was progressing in ED recovery, she was not forthcoming about increased urges for opiates. JC was able to obtain heroin and overdosed, which resulted in a brief hospitalization (i.e., 48 h) off-site. Prior to this, all drug screens were negative. JC denied suicidal intent and demonstrated awareness for how this jeopardized her recovery. She took personal responsibility and was compliant with a strict behavioral contract throughout the remainder of her treatment. She participated in daily urine analyses and agreed to the use of Suboxone to interrupt urges for heroin use.

21.7.6 Treatment Contract

- Patient will attend all groups and meals as scheduled, arriving on time.
- Patient will stay in program, during program hours, unless permission is granted to leave the site.

- Patient will demonstrate accountability regarding medications and substances.
  - Store all medications in a lock box and take as prescribed under supervision of staff.
  - Abstain from asking any patient or outside party for any substance that is not prescribed, including any OTC medications.
  - Abstain from giving any substance to a peer or any outside party, including OTC medications.
- Patient will demonstrate accountability regarding any substance use.
  - Report to staff the use of any prescribed or illegal substance.
  - Submit to all drug screens.
- Patient will demonstrate respect to therapists during groups, meals, and individual sessions.
  - If behavior is experienced as inappropriate, patient may be asked to leave the session and can discuss it with that staff member at a scheduled time.
- Patient will comply with supportive housing structure and rules as described in the handbook.
  - Comply with random room searches.
  - Must stay with staff on all scheduled outings until further notice.
  - May not leave the house on non-outing nights until further notice.
  - Must stay in smoking area only during smoke breaks.
- Demonstrate appropriate language and boundaries with others in the program.
  - Abstain from speaking in terms that glorify substance use.
  - Verbalize emotional discomfort to staff and peers through "I" statements.
- Participate in the specialized treatment track that integrates targeted treatment for both the ED and the SUD.
  - Identify and attend 3 outside AA meetings per week after program hours.
  - Obtain an AA sponsor and invite this sponsor to attend multifamily group to learn about and support treatment of the ED and SUD.

Commentary

- *The use of 12-Step Facilitation in individual therapy can increase the likelihood that patients will attend meetings at the completion of formal treatment.*
- *Individuals with other SUD besides a drinking problem may find NA meetings to be more relevant and to meet their needs better than AA groups.*
- *Programs should also have other recovery resources available to patients that are not comfortable with the 12-step approach.*

21.7.7 Treatment Goals

JC participated in the addiction track with the following specific treatment goals (1) develop an understanding of her personal pattern of relapse in order to sustain long-term recovery, (2) develop an understanding of the relationship between her SUD and ED, (3) create a relapse prevention plan outlining components of a drug-free lifestyle while in recovery from the ED, (4) create a plan to develop new peer relationships, and (5) develop an academic and/or career plan for the future.

Her central treatment team consisted of a primary therapist (i.e., a licensed psychologist with CAC certification and substantial training in ED), a psychiatrist (i.e., certified in addiction medicine), a registered dietitian, and a family therapist. JC actively participated in individual CBT treatment, DBT skills group, trauma group, mindful meditation group, art therapy, and psychoeducational groups for substance users and met regularly with the dietitian for nutritional counseling and caloric adjustments to promote weight restoration.

21.7.8 Discharge

JC remained in treatment for 10 weeks and was discharged on the following medications: sertraline 200 mg daily, trazodone 100 mg nightly, clonazepam 1 mg BID, buprenorphine/naloxone (Suboxone) ½ strip daily, and gabapentin 1,200 mg daily in divided doses. JC's treatment disposition once she was weight restored was to admit herself to a 30-day transitional sober living house to begin her effort to step down from Suboxone and become fully abstinent from all drug use while maintaining her ED recovery. The discharge coordinator discussed this plan with her former outpatient team who resumed counseling, drug testing, and medication management during and after her 30-day stay in a sober living house.

21.7.9 Summary

This case illustrates a number of principles associated with successful integrated care, including the following:

1. The availability of a SUD track within an existing ED program.
2. The availability of therapists and staff dually trained in both ED and SUD.
3. The availability of an addiction certified psychiatrist as a member of the treatment team.
4. The incorporation of the concepts of adaptive function into the case formulation and treatment of dually diagnosed patients.
5. The use of nonconflicting, integrated therapies delivered in various formats by various staff.
6. The use of CBT as a cornerstone of treatment and the use of other EBT.
7. The inclusion of 12-step programs and philosophy or other recovery self-help programs.

8. The ongoing monitoring of eating disorder behaviors and drug use.
9. The use of testing for the presence of HIV/AIDS, hepatitis B and C, tuberculosis, and other infectious diseases.
10. Multiple needs of the individual (not just the ED/SUD) were met, including the treatment of other comorbid conditions and the development of a plan for the future.
11. The patient remained in treatment for an adequate period of time for treatment effectiveness.

Conclusions

In this chapter, the case for an integrated treatment approach for patients with comorbid ED and SUD and related disorders has been developed and discussed. Basic guidelines and principles of effective treatment for this population are presented, which are modifications of recommendations made by the Substance Abuse and Mental Health Service Administration and the National Institute on Drug Abuse. Guidelines are also proposed for the development of integrated treatment programs at various levels of care, including pretreatment or early intervention, as well as outpatient, intensive outpatient, partial hospitalization, residential, and inpatient treatment settings. The concept of adaptive function is described as an important and potentially unifying strategy for case formulation and treatment. The chapter concludes with a case example that illustrates some of the complexities of implementing integrated treatment for patients with both ED and SUD.

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Motivational Interviewing in the Treatment of Substance Use Disorders, Addictions, and Eating Disorders

22

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Abstract

Motivational interviewing is an evidence-based approach for helping clients resolve ambivalence about change. An accumulation of research supports the efficacy of motivational interviewing in engaging and retaining clients in treatment across a variety of healthcare settings and with diverse populations. Core components and proposed mechanism of action will be reviewed. This chapter will further examine how motivational interviewing is used in the treatment of addictive and eating disorders and explore research that has advanced the field for these disorders, with implications for future study.

Keywords

Motivational interviewing • Substance use disorders • Eating disorders • Comorbidity • Treatment

22.1 Introduction

Motivational interviewing (MI), an evidence-based client-centered approach aimed at facilitating behavior change, has been used in the addiction field for over 25 years. The effectiveness of MI in changing the way clinicians approach addictive disorders has permeated other healthcare areas that involve lifestyle change. MI is a departure from the traditional confrontational approaches used in the treatment of

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substance use disorders (SUD). Inherent in MI is maintaining a spirit that embraces client autonomy and collaboration, which allows clients the freedom to more fully explore their own behavior. Ambivalence is a normal process on the path to change, and MI helps clients resolve ambivalence. MI empowers clients to make decisions that are consistent with important life values and/or goals. Change is often considered when clients perceive a discrepancy between their current behavior and what they value or what is important in life.

Client change talk, elicited by clinicians using evocative approaches and reinforcement, has been identified as an important predictor of behavior change (Amrhein, Miller, Yahne, Palmer, & Fulcher, 2003; Moyers et al., 2007). **Preparatory change talk** often precedes a more robust type of change talk called **mobilizing talk** in which commitment to take action is stated. Linguistic analysis of MI session recordings has demonstrated that the active ingredient for behavior change outcomes in MI is client change talk. Specifically, **commitment talk**, the more robust form of change talk, is strongly associated with behavior change (Amrhein et al., 2003). Within session mechanisms of change associated with client behavior and clinician MI technique were explored in a meta-analysis of 19 studies. Better outcomes were associated with client change talk/intention and client experience of discrepancy. Clinician MI-inconsistent behavior was related to worse outcomes. Clinicians' use of specific techniques such as a decisional balance exercise showed the strongest association to better outcomes (Apodaca & Longabaugh, 2009).

Clinicians use open-ended questions, affirmations, reflections, and summaries to evoke change talk and explore goals/values that are important. Having the self-efficacy to make major life changes is essential in order for change to occur. Clinicians use MI to empower clients to increase confidence in their ability to make behavior change. For example, a clinician may ask a client "what did you do the last time you were successful in reducing your alcohol use (*or other problem behavior*)?" Allowing clients the autonomy to make decisions is pertinent to both SUD and eating disorders (ED) where clients experience a loss of control in so many areas of their life. Now in its third edition, *Motivational Interviewing: Helping People Change*, Miller and Rollnick (2012) have revised the practice of MI in several areas as a result of ongoing empirical research.

22.1.1 Four Processes of MI

Miller and Rollnick (2012) introduce the four processes of MI in the third edition of their book. These four processes provide more direction to the flow of the MI conversation. In sequentially moving through these processes, clinicians are better able to guide clients through a path toward change. The four sequential but overlapping processes include engaging, focusing, evoking change, and planning for change. MI was initially developed with phase 1 and phase 2 components. MI principles and skills addressed client ambivalence about change (phase 1), with less emphasis on moving the conversation to discussions about "how to" or exploring

options for change once clients were ready to change (phase 2). Thus, clinicians move clients beyond resolution of ambivalence into more action-oriented conversation with the flexibility to cycle back through processes with fluctuating ambivalence and/or sustain talk. Establishing a trusting, therapeutic relationship through **engaging** the client builds the foundation for future work and has been shown to impact retention and substance use outcomes (Crits-Christoph, Gibbons, Hamilton, Ring-Kurtz, & Gallop, 2011). **Focusing** the conversation on a mutually established agenda often involves exploring and clarifying values/goals that may lead to behavior change consideration. **Evoking** the client's own motivation for change is critical to MI. Clients, not clinicians, argue for change. Clinicians elicit change talk through MI-specific skills and reinforce it when it does occur. Clinicians determine the level of readiness for clients to move to more action-oriented MI. There are several indicators of readiness such as envisioning what change would be like or asking for help or information about change options. **Planning for** change involves utilizing the same MI spirit and style as used in the other processes. It is important that the clinician is in tune with the client and moves through these processes at the client's pace (Miller & Rollnick, 2012).

Clients demonstrating readiness to change are often ready to mobilize a plan of action. There is a shift from preparatory to mobilizing language. Clinicians are evoking and supporting client statements that express intentions and commitment. Clinicians take care to approach change plans in a collaborative, respectful manner, allowing clients the autonomy to decide what is in their best interest. The "righting reflex" or the helpful desire on the part of the clinician to "fix" the problem is a common urge that clinicians should be aware of and monitor. The skillful MI clinician will exchange information and provide advice with permission in an MI-consistent manner. It is not uncommon for the clinician to offer the client a menu of options. Clients with SUD often feel out of control in their lives and having choices can empower them. Change plans can be verbal or written, but often specific goals need to be broken into small achievable steps so that patients may experience early success. The MI change plan may also identify expected benefits of change, barriers that may get in the way of change, and success criteria. It is important that patients have the confidence and self-efficacy to implement the plan (Miller & Rollnick, 2012).

22.2 MI Applied to Substance Use Disorders and Addictive Disorders

22.2.1 Outcome Research

Motivational interviewing has been one of the most widely researched interventions. To date, there have been several meta-analyses exploring the effectiveness of MI across various health behaviors. The most recent meta-analysis of MI effectiveness across a variety of problem behaviors showed that 50 % of studies demonstrated small to moderate effect sizes and 25 % of studies demonstrated

moderate to large effect sizes (Lundahl, Kunz, Brownell, Tollefson, & Burke, 2010). Importantly, this meta-analysis found that MI can produce at least equal positive health behavior changes in comparison to strong psychosocial interventions and can do so in a shorter length of delivery time. Thus, research has demonstrated that MI is successful in motivating clients to change across a variety of problem domains. In addition, the meta-analysis found that MI effects are durable across time, MI can be delivered in various formats but may work best as a prelude to further treatment, and a higher dosage of MI treatment time may produce better outcomes. The use of a manual to deliver MI does not necessarily contribute to effectiveness, and there may be certain populations such as socially excluded individuals that fare better with MI approaches.

Adolescents are a vulnerable population with high rates of substance use, yet treatment options remain limited and adolescents are difficult to engage and retain in treatment. As such, a MI-consistent autonomous and collaborative approach is more appealing for adolescents. In a review of 39 studies exploring mechanisms of change for MI in adolescents with SUD, 67 % of the studies showed a positive effect on substance use outcomes (Barnett, Sussman, Smith, Rohrbach, & Spruijt-Metz, 2012). Differences in MI intervention designs made establishing specific mechanisms for change in adolescent populations difficult. Specific MI adaptations for adolescents include school-based interventions and the involvement of family members. Studies in the review examined various forms of MI, including MI implemented alone, added to other interventions such as CBT, delivered with feedback, given following MD advice, and used as a booster to skill-based classroom or social network interventions. Attitudinal (readiness/intention to change, engagement in treatment, perceived risk) and behavioral (reduced dependence criteria, drug refusal skills, self-monitoring) mechanisms for change were reported in some studies. In two studies whereby process of change was measured, client change talk and commitment were related to substance use outcomes (Baer et al., 2008; Engle, Macgowan, Wagner, & Amrhein, 2010). Miller and Rose (2009) proposed a hypothesized path relationship between MI and behavior change. Clinicians using MI-consistent counseling style elicit preparatory (DARN: desire, ability, reason, need) change talk followed by an increasingly stronger pattern of commitment talk over the course of the session which is predictive of positive change outcomes. Alternately, clinicians using a MI-inconsistent confronting style elicit more sustain talk or resistance to change, which is predictive of poor outcomes.

22.2.2 MI as a Stand-Alone Approach

MI can be implemented as a stand-alone approach, a preamble to another intervention, an adjunct to another intervention, and delivered with feedback as is done in motivational enhancement therapy (MET). MI is particularly useful in approaching clients who are reluctant to change or complacent with the status quo. Studies have explored the use of brief MI interventions in engaging clients in treatment and

improving retention. In one large National Institute of Drug Abuse (NIDA) Clinical Trials Network (CTN) multisite study, 423 individuals presenting for treatment at community substance abuse treatment programs were randomized to receive either a standard intake assessment only or a standard intake assessment with 20 min of MI techniques integrated into the intake session. Participants were followed for retention in treatment and substance use outcomes. Those receiving the MI intervention attended more treatment sessions and were more likely to be enrolled in substance abuse treatment services at 1 month than those receiving the standard intake assessment only (Carroll et al., 2006).

22.2.3 MI as an Adjunct or Prelude to Psychosocial Interventions

Given that MI is a style of communication that engages individuals in behavior change, it is not surprising that MI has been integrated into other psychosocial therapies to improve treatment engagement and outcomes. MI coupled with client assessment feedback (motivational enhancement therapy—MET) and delivered in three to four sessions provides clients with objective information that can help guide decisions about change. In a large multisite community study, 461 substance abuse treatment-seeking clients were randomized to receive either three individual sessions of MET or three individual sessions of counseling as usual (CAU) in addition to the regular group counseling offered at the perspective community programs (Ball et al., 2007). In addition to employing MI spirit and style, MET clients were given personalized feedback reports collected and summarized from assessments they completed at study entry. Clinicians used MI skills to explore ambivalence, develop discrepancies between current behavior and stated values, elicit change talk, and collaboratively work a change plan. Clients participated in the three sessions within the first 28 days of community treatment entry. Retention and substance use outcomes were followed out to 16 weeks. Although both groups reduced the number of days of primary substance use by the end of the study intervention (4 weeks), those in the MET condition maintained this reduction into the follow-up period, while those in the CAU returned to baseline levels of use during this time (Ball et al., 2007).

A five-session motivational enhancement therapy plus cognitive behavioral therapy (MET/CBT) intervention used in the Cannabis Youth Study included two individual MET sessions focused on resolving ambivalence and increasing motivation, followed by three group sessions of cognitive behavioral coping skills (Ramchand, Griffin, Suttorp, Harris, & Morral, 2011). Adolescents receiving the MET/CBT intervention were followed out to 12 months and compared to outpatient community treatment as usual (TAU) that was deemed "exemplary" by the Substance Abuse Mental Health Service Administration (SAMHSA) standards. At 12 months, greater reductions in substance use and illegal behaviors were observed in the adolescents receiving MET/CBT than those in the TAU condition. These results demonstrate that five sessions of MET/CBT delivered weekly can

outperform community TAU that typically is longer in duration and involves more treatment sessions.

A study targeting individuals with cocaine use disorders compared the addition of one MET session followed by two CBT sessions (MET + CBT) to three sessions of CBT delivered alone (McKee et al., 2007). Clinicians in the MET + CBT condition were trained to also use an MI style when delivering the CBT component. Participants were referred to community substance abuse treatment following the interventions. Primary outcomes included changes in treatment motivation, treatment satisfaction, and retention in standard community treatment. By the end of the intervention, those receiving MET + CBT reported greater desire for abstinence and greater expectation of success than those receiving CBT alone. While the proportion of participants attending ongoing community treatment was similar across groups, those in the MET + CBT attended significantly more treatment sessions than the CBT alone group (5.66 vs. 1.57, respectively).

Motivational interviewing has also been successfully integrated with compliance enhancement therapy (MI-CET) to increase retention and medication adherence in pharmacotherapy studies. Sessions use MI techniques and personalized feedback (i.e., alcohol use severity, lab results) coupled with medication instructions and problem-solving discussions related to medication adherence. A nine-session manual-guided MI-CET intervention delivered with the antidepressant, citalopram, was evaluated for its impact on treatment adherence and retention in 121 treatment-seeking adults with alcohol dependence (Heffner et al., 2010). Compared with another CET without MI delivered as an adjunct to medication, MI-CET participants had 20–30 % higher treatment completion rates. Thus, studies clearly show the enhanced benefits of integrating MI into other therapy approaches.

22.2.3.1 Group MI

Substance abuse programs typically deliver treatment in a group format. Although MI was originally designed for implementation in individual sessions, several studies have explored its efficacy in group therapy. Female college drinkers ($N = 110$) cited for campus alcohol violations attended one gender-specific motivational interviewing group session followed by 12 weeks of completing online drinking dairies (LaBrie, Thompson, Huchting, Lac, & Buckley, 2007). The sessions were 120 min and included personalized information regarding their alcohol consumption patterns, normative feedback, gender differences in alcohol effects, and discussions about participants' reasons for drinking. Reductions in alcohol consumption (29.9 %) and drinking consequences (35.9 %) from pre-intervention to 3 months post-intervention were seen, with the most pronounced effects in the heaviest drinking groups.

Another MI group intervention (GMI) was compared to an attention activity control group (TAAC) in 101 inpatients with comorbid SUD and psychiatric disorders (Santa Ana, Wulfert, & Nietert, 2007). Patients received two sessions lasting 120 min and outcome measures collected at 1 and 3 months included aftercare attendance and substance use. Patients in the GMI attended more aftercare sessions than those in the TAAC group at 1 month (25.4 vs. 16.8, respectively) and

at 3 months (21.1 vs. 10.7, respectively). At 1 month, patients in the GMI group reported significantly fewer days of illicit substance use and binge drinking, and they consumed less alcohol than those in the TAAC group. These effects were sustained for alcohol consumption and binge drinking at the 3-month follow-up. More randomized controlled studies exploring the efficacy of group MI are needed to advance the field and increase the potential for adoption in community programs where the primary mode of treatment delivery is group therapy.

22.2.3.2 MI Delivered in Medical Settings

Only a small proportion of individuals who have problems with alcohol and illicit drug use actually receive formal substance abuse treatment. Many of these individuals are seen in emergency rooms or in primary care clinics when medical problems associated with problematic use are encountered. One study using a state trauma registry found that 21 % of injuries requiring hospitalizations were related to substance use and the most severe injuries were substance related (Socie, Duffy, & Erskine, 2012). Offering brief MI interventions targeting alcohol and drug use can address substance use problems and also reduce healthcare utilization. Studies show that using brief screening assessments in emergency department and primary care clinics followed by a 15–20 min brief MI intervention can decrease alcohol and drug use in patients presenting with modest problematic use and can identify and refer those patients in most need of referral to substance abuse treatment programs (Pilowsky & Wu, 2012; Saitz, 2005).

In one large study implemented across multiple medical treatment settings, 22.7 % of patients screened positive for problematic use to abuse/dependence of alcohol or illicit substances (Madras et al., 2009). Sixteen percent received a brief intervention, and another 7 % received referral to treatment. Brief interventions included discussing the patient's perspective of his or her substance use, providing feedback from screening assessments compared with normative values, providing recommended use guidelines (i.e., National Institute of Drug Abuse [NIDA], 2010), and collaboratively developing a change plan. Several sites in this study used a brief intervention referred to as FRAMES that employs an MI approach: feedback, responsibility, advice, menu of options, empathy, and self-efficacy (Bien, Miller, & Tonigan, 1993). In a random selection of the population that screened positive at baseline, there were significant reductions in illicit drug use (67.7 %) and heavy alcohol use (38.6 %) at the 6-month follow-up. In the more problematic group referred to specialty substance abuse treatment, there were significant improvements in other functional domains such as mental health, criminality, unemployment, and housing instability. Clearly, implementing SBIRT (screening, brief intervention, referral to treatment) interventions in emergency departments, primary and specialty medical care settings can help to prevent risky alcohol/substance use from becoming a significant medical concern and to refer patients who are already experiencing medical complications to formal treatment (NIDA, 2010).

22.2.4 MI and Behavioral Addictions

Motivational interviewing has also been implemented for pathologic gambling, a DSM-5 substance-related and addictive disorder diagnostic category. A meta-analysis of psychotherapy outcomes for gambling behavior included three motivational interviewing studies that demonstrated modest reductions in gambling frequency and less financial loss as a result of gambling (Cowlishaw et al., 2012). Petry, Weinstock, Morasco, and Ledgerwood (2009) conducted a randomized control study comparing three interventions (brief advice, MET, or MET + CBT) for problem gambling in college students. Although all interventions improved problematic gambling behavior, at the 9-month follow-up, students receiving the 50-min session of MET wagered substantially less of their income than those students receiving the other interventions. With the addition of pathologic gambling to the addiction diagnostic category, more research exploring substance use treatment approaches such as MI for pathologic gambling and perhaps other behavioral addictions can be expected in the future.

22.2.5 Dissemination of MI

MI has been one of the most widely disseminated approaches in substance abuse treatment programs over the last 10–20 years. In a recent survey administered to over 1,400 clinicians in 345 private community substance abuse treatment programs across the USA, over 80 % reported using MI in their practice (Bride, Kintzle, Abraham, & Roman, 2012). Although MI has been adopted in many substance abuse programs, the lack of supervision in MI is a challenge to the field because it is necessary to maintain MI fidelity. In order to adequately implement MI, training involves intensive skill building workshops, post-workshop experiential practice and role plays, and ongoing supervision and coaching guided by fidelity ratings of taped sessions (Miller, Yahne, Moyers, Martinez, & Pirritano, 2004). Studies have shown that the best supervision model is to directly observe or review taped sessions for clinician MI-consistent and MI-inconsistent style and provide clinicians with feedback and coaching. Implementing MI with proficiency has been shown to increase patient motivation and decrease resistance (Gibbons et al., 2010; Martino, Ball, Nich, Frankforter, & Carroll, 2008; Miller et al., 2004). In one large multisite community program study, a reduction in drug use during the first 4 weeks of treatment was related to clinicians' MI skill ratings (Martino et al., 2008). The National Institute of Substance Abuse (NIDA) in conjunction with the Substance Abuse and Mental Health Services Administration (SAMHSA) created a MI supervision toolkit called Motivational Interviewing Assessment: Supervisory Tools for Enhancing Proficiency (MIA:STEP) (Martino et al., 2006). This product contains a tool for rating MI fidelity that can be used by supervisors to evaluate clinicians' use of MI, provide feedback, and coach clinicians to implement MI with fidelity (Martino et al., 2006). Without supervision and coaching, clinicians are more likely to experience therapy drift and fall below recommended proficiency

levels. Discrepancies between clinician self-report and supervisor/expert fidelity ratings on MI style, MI-consistent, and MI-inconsistent proficiency ratings validate the need for continued post-workshop supervision (Martino, Ball, Nich, Frankforter, & Carroll, 2009). Clinician characteristics and organizational climate have been shown to influence acquisition and retention of MI skills and adoption of practice (Baer et al., 2009). The MIA-STEP toolkit ensures best practices for delivering MI.

22.3 MI Applied to Eating Disorders

Individuals with eating disorders (ED), particularly those with binge-eating symptoms, often describe their disorder as akin to an addiction (Cassin & von Ranson, 2007). Many similarities have been noted between binge eating and SUD, including preoccupation with thoughts about the substance (i.e., psychoactive substance or food), cravings and repeated urges to consume the substance, mounting tension until the substance is consumed, loss of control over the behavior, consuming larger amounts than intended, feeling ashamed following use of the substance, feeling unable to reduce or stop the behavior, and continuing to engage in the behavior despite knowledge of adverse effects (Cassin & von Ranson, 2007; Gold, Frost-Pineda, & Jacobs, 2003; Wilson, 1991).

Similar to SUD, ED symptoms are also reinforcing and fulfill important and valued functions for the individual. For example, individuals with anorexia nervosa (AN) report that their ED provides a sense of safety and protection, allows them to feel in control, and makes them thinner and more attractive as a result of weight loss (Serpell, Treasure, Teasdale, & Sullivan, 1999). In addition to these benefits, individuals with bulimia nervosa (BN) also report that their ED allows them to avoid or manage their emotions and to eat forbidden food without gaining weight (Serpell & Treasure, 2002). Not surprisingly, given their ego-syntonic nature, individuals often feel reluctant to engage in treatment and make changes to their ED symptoms. However, individuals also acknowledge some costs associated with their ED, including feeling that the ED has taken over their life (ironically, making them feel more out of control), hindering relationships and career opportunities, and damaging physical health (Serpell et al., 1999; Serpell & Treasure, 2002). MI is designed to capitalize on this ambivalence and to enhance intrinsic motivation and readiness for change.

Readiness for change is an important target for treatment because it has significant implications for ED recovery. For example, longitudinal research demonstrates that readiness for change predicts enrollment in intensive ED treatment, completion of recovery-related activities, weight gain, treatment dropout, and relapse following treatment (Bewell & Carter, 2008; Geller, Cockell, & Drab, 2001; Geller, Drab-Hudson, Whisenhunt, & Srikameswaran, 2004; Rieger et al., 2000). It is important to note that, in addition to fluctuating over time, readiness for change also varies across specific ED symptoms. For example, an individual might feel

ready to stop bingeing and purging, yet still feel pre-contemplative about reducing dietary restriction (Geller et al., 2001).

22.3.1 Outcome Research

Empirical research examining the impact of MI on readiness for change and psychosocial functioning in the ED has been accumulating over the past decade. In the treatment of ED, MI has been used as both a stand-alone intervention (in conjunction with a self-help manual) (Cassin, von Ranson, Heng, Brar, & Wojtowicz, 2008; Dunn, Neighbors, & Larimer, 2006), and as a prelude or adjunct to another treatment, such as outpatient cognitive behavioral therapy (Gowers & Smyth, 2004; Katzman et al., 2010; Treasure et al., 1999) or intensive inpatient or day patient ED treatment (Dean, Touyz, Rieger, & Thornton, 2008; Feld, Woodside, Kaplan, Olmsted, & Carter, 2001; Geller, Brown, & Srikameswaran, 2011; George, Thornton, Touyz, Waller, & Beumont, 2004; Wade, Frayne, Edwards, Robertson, & Gilchrist, 2009; Willinge, Touyz, & Thornton, 2010). A recent systematic review of 10 studies concluded that MI holds promise in the treatment of ED, particularly with respect to its impact on readiness for change (Macdonald, Hibbs, Corfield, & Treasure, 2012). Although heterogeneity in study design and methodology was noted as limiting comparison across studies, the bulk of the evidence suggests that MI can be effective in increasing readiness for change and improving eating pathology and psychosocial functioning (e.g., depression, anxiety, self-esteem, quality of life).

22.3.2 MI as a Stand-Alone Treatment

Studies examining the impact of single-session, stand-alone MI interventions in reducing binge eating have generated promising results. College students ($N = 90$) with full or subthreshold BN or binge-eating disorder (BED) who were randomly assigned to receive MET + self-help handbook reported increased readiness for change compared to those who received a self-help handbook only (Dunn et al., 2006). Although both groups decreased the frequency of their binge eating to a similar extent, the MET group had higher binge abstinence rates at 4 months (24 % vs. 9 %). In another study, women with BED ($N = 108$) recruited from the community who were randomly assigned to receive MI + self-help handbook reported greater improvements in binge eating, depression, self-esteem, and quality of life over a 4-month follow-up period compared to those who received a self-help handbook only (Cassin et al., 2008). In addition, the MI + self-help handbook group reported higher binge abstinence rates (28 % vs. 11 %). Although research on stand-alone MI interventions for EDs is still in its infancy, the studies conducted to date suggest that MI can be effective in improving binge eating and psychosocial functioning in non-treatment-seeking samples.

22.3.3 MI as a Prelude or Adjunct Treatment

Studies examining the application of MI in clinical ED samples have used MI as a prelude to inpatient, day patient, or outpatient ED treatment with the aim of increasing readiness for change and engagement in treatment or as an adjunct to treatment with the aim of improving remission rates and preventing dropout.

22.3.3.1 Intensive Treatment

Individuals with AN and BN ($N = 19$) participating in a pilot study examining a four-session group MI intervention as a prelude to a specialized ED treatment reported improvements in readiness for change, depression, and self-esteem over the course of treatment (Feld et al., 2001). Improvements were not reported in eating pathology over the 6-week period; however, it is notable that 90 % of participants enrolled in specialized ED treatment afterwards.

A more recent study examining the impact of a four-session group MI intervention as an adjunct to treatment as usual in an inpatient sample ($N = 42$) reported that MI did not improve eating pathology to a greater extent than treatment as usual; however, the MI intervention fostered greater engagement in therapy and promoted treatment continuation (Dean et al., 2008).

Another study examining the impact of a four-session MI intervention as an adjunct to treatment as usual in an inpatient AN population ($N = 47$) reported that a greater proportion of individuals in the MI group moved from "low" to "high" readiness for change over the study period relative to the treatment-as-usual group, whereas a greater proportion of the treatment-as-usual group dropped out from the study (Wade et al., 2009). However, there were no significant group differences in eating pathology among individuals who completed treatment.

A randomized controlled trial examined the efficacy of MI on readiness for change, eating pathology, and psychosocial functioning in a mixed diagnosis (AN, BN, EDNOS) tertiary care clinical population ($N = 113$) (Geller et al., 2011). In this study, a smaller proportion of individuals in the MI group were rated as "highly ambivalent" at 6-week and 3-month follow-up compared with those in the control condition. However, both MI and control groups reported similar improvements in eating pathology and depression. This latter finding might be attributed to two sources of "MI contamination" in the control condition. First, all study participants completed the readiness and motivation interview to assess readiness and motivation for change, which has several ingredients (e.g., curious, nonjudgmental interviewer stance when discussing readiness) that are similar to a single-session MI interventions. Second, the majority of participants in both groups were exposed to additional treatment from care providers within an ED program that used an MI stance during the study period (Geller et al., 2011).

22.3.3.2 Outpatient Treatment

Adolescents with AN ($N = 42$) who participated in a motivational assessment as part of a pilot study reported improved motivation following the assessment, and 80 % enrolled in an outpatient CBT program (Gowers & Smyth, 2004). Moreover,

individuals who reported greater motivation following the assessment gained significantly more weight over a 6-week period.

A large randomized controlled trial examined the role of readiness for change in treatment engagement and outcome in individuals with BN (Katzman et al., 2010; Treasure et al., 1999). The first phase of treatment compared 4 sessions of either MI or CBT in engaging patients in treatment and improving symptoms in women with BN ($N = 125$) (Treasure et al., 1999). Despite a focus on motivation rather than symptom reduction, MI was as effective as CBT in reducing the frequency of binge eating, vomiting, and laxative abuse over the first 4 weeks of treatment. In terms of clinically significant change, 53 % of individuals in the MI group showed improvement in binge eating, 58 % in vomiting, and 27 % in laxative abuse. In a subsequent two-stage randomized control trial, individuals with BN and EDNOS ($N = 225$) were randomly assigned to receive 4 sessions of MI or CBT in phase 1 as a prelude to 8 sessions of either individual or group CBT in phase 2 (Katzman et al., 2010). All groups reported significant improvements in binge eating, vomiting, and laxative abuse, and they did not differ from one another. In addition, the groups did not differ with respect to treatment completion/dropout.

Taken together, the body of literature suggests that MI holds promise in the treatment of ED, particularly among individuals engaging in eating binges and/or compensatory behaviors that are not sufficiently severe to require treatment at a tertiary care facility. In individuals presenting for treatment at specialized inpatient and day treatment programs, MI alone does not appear to have an immediate impact on eating pathology. This finding is not surprising given that existing gold standard evidence-based treatments for ED, such as CBT, typically require 20–40 sessions depending on the client's weight status (Fairburn, 2008). However, the empirical research published to date does suggest that MI increases readiness for change and promotes enrollment in, and engagement with, action-oriented treatment.

MI is increasingly being incorporated into evidence-based treatments for ED. For example, treatments such as CBT and dialectical behavioral therapy (DBT) have a client-centered focus and emphasize the importance of using a collaborative stance throughout treatment. In addition, they both frequently make use of specific MI techniques, such as using a decisional balance to explore ambivalence regarding making and maintaining changes (e.g., reducing dietary restriction or binge eating). The decisional balance can be used at the beginning of treatment to increase clients' readiness for change. In addition, it can be used during the course of treatment if progress comes to a standstill (e.g., to explore ambivalence about completing between-session homework assignments such as food records) or if the client is contemplating dropping out of treatment (e.g., to examine the benefits and costs of staying in treatment vs. dropping out from treatment). Other ED treatments explicitly incorporate MI directly into the treatment protocol. For example, a novel cognitive interpersonal treatment for AN, the Maudsley Anorexia Nervosa Treatment for Adults (MANTRA) uses MI techniques to explore pro-anorexia beliefs and examine their potential role in the maintenance of the ED.

22.4 Using MI in Comorbid ED and SUD

Individuals with an ED appear to be over four times more likely to develop a comorbid SUD compared to the general population (Gadalla & Piran, 2007). The specific rates of comorbidity vary depending on the type of ED symptoms and type of substance. For example, rates of alcohol use disorders tend to be elevated in individuals with bingeing symptoms and purging symptoms (Calero-Elvira et al., 2009; Franko et al., 2005; Gadalla & Piran, 2007), whereas they do not tend to be elevated in individuals with AN-restricting subtype (Franko et al., 2005). A potential reason for this pattern of comorbidity is that individuals with restricting AN avoid alcohol due to its high caloric content, whereas alcohol reduces inhibitions thereby increasing susceptibility to binge eating in individuals with AN-binge/purge subtype, BN, and BED. Stimulant use disorders also tend to be elevated in ED populations, and it has been postulated that individuals with ED might begin taking stimulants to suppress appetite and promote weight loss (Nøkleby, 2012). As mentioned previously, in addition to having elevated rates of comorbidity, individuals with SUD and ED share many "addictive" features, such as feeling a loss of control over the behavior and inability to reduce or stop the behavior despite adverse physical and psychological effects (Cassin & von Ranson, 2007; Gold et al., 2003; Wilson, 1991). Particularly relevant to the topic of MI, ambivalence about recovery tends to be the norm among individuals with SUD and ED and can pose a significant barrier in treatment if not addressed.

22.4.1 Limited Research

In light of the shared features and high rates of comorbidity, an integrated treatment approach that concurrently addresses SUD and ED symptoms is recommended (Harrop & Marlatt, 2010). Although MI has a large evidence base in the treatment of SUD, and a growing evidence base in the treatment of ED, no empirical studies have examined MI in the treatment of comorbid SUD and ED. Individuals with both types of disorders are often characterized as ambivalent about change, difficult to engage in treatment, and prone to relapse. Therefore, MI could be particularly helpful given that it is intended to increase readiness for change and promote engagement in treatment. Unfortunately, individuals who present for treatment with comorbid SUD and ED are often excluded from research studies and clinical services at specialty ED programs and addiction programs on the basis of their coexisting disorders.

22.5 Future Directions

The clinical practice and empirical investigation of MI has grown exponentially over the past decade. With respect to ED, many studies either lack a control condition or have insufficient follow-up periods to determine the longer-term

impact of the MI intervention. With these caveats in mind, the research conducted to date suggests that MI has the potential to increase readiness for change and improve eating pathology, particularly in individuals who binge eat and/or engage in compensatory behaviors. Although MI has been shown to increase readiness for change in individuals with AN, the studies conducted to date have reported relatively little impact on eating pathology. However, with a few exceptions (Gowers & Smyth, 2004), MI has been conducted primarily in a group format with individuals with AN. It would be informative to examine the impact of individual MI on readiness for change, dietary restriction, and weight restoration in individuals with AN.

MI was originally intended for individuals not yet ready to take action, whereas CBT is an action-oriented treatment intended for individuals ready to make changes; however, this patient-treatment matching assumption has not yet been examined empirically. Thus, this patient-treatment matching assumption (i.e., what treatment works best for whom) would be a valuable avenue for future research. When compared to other evidence-based treatments, such as CBT, MI has been shown to exert similar effects on readiness for change and treatment outcome, suggesting that MI may be used as an alternative to CBT in the preliminary stages of treatment (Katzman et al., 2010; Treasure et al., 1999). When MI has been delivered as a prelude to CBT, MI has typically consisted of a set number of sessions (one to four sessions). Future research would benefit from empirically examining when it is best to transition from the engagement to action-oriented (i.e., symptom reduction) phases of treatment. Finally, given that MI is increasingly being incorporated into action-oriented evidence-based treatments, it would also be informative to conduct dismantling studies to determine the effective components of these integrated treatments.

Finally, given the elevated rates of comorbidity and shared features between ED and SUD, it would certainly be a worthwhile endeavor to examine the effectiveness of MI in individuals with comorbid disorders.

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Nutrition Therapy for Eating Disorders, Substance Use Disorders, and Addictions

23

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Abstract

Medical nutrition therapy for individuals with co-occurring eating and substance use disorders includes assessment, planning, nutrition intervention, and counseling. This involves individual education, meal planning, and monitoring of compliance. Recognized eating disorders include anorexia nervosa, bulimia nervosa, and binge eating disorder. Substances commonly abused include alcohol, stimulants, opiates, and various over-the-counter substances such as diet pills, laxatives, and diuretics. Integrated treatment can be challenging when the synergistic effects of combined entities are complex and are poorly understood. Clinicians working with patients who have dual diagnoses should be educated about each disorder separately, as well as their interactions. Patients with substance use disorders often develop disordered and dysfunctional eating patterns during abstinence, and eating disorder patients can similarly progress into substance abuse. Traditionally, addiction has been addressed first; however, delaying eating disorder treatment can hinder recovery; therefore, it is important to alert treatment providers who treat patients with dual diagnoses how to assess and address both disorders simultaneously. Specific macro- and micronutrient supplementation treatment is described in detail and protocols for refeeding in selected cases are provided. Nutrition therapy should address the most serious medical and nutrition conditions first and then target the psychological aspects related to eating behavior in conjunction with a multidisciplinary treatment

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team. Nutrition education is important for addiction recovery, particularly those who require specialized wellness care, whereas education for disordered eaters must be sensitive to their specific needs. While these guidelines can help steer nutrition interventions for co-occurring eating and substance use disorders, nutritional needs are always best assessed on an individual basis.

Keywords
Alcohol use disorder • Anorexia nervosa • Binge eating disorder • Bulimia nervosa • Cocaine use disorder • Dual diagnosis • Opioid use disorder • Methamphetamine use disorder • Nutrition therapy • Substance use disorder

23.1 Introduction

The increasing prevalence of co-occurring eating disorder (ED) and substance use disorder (SUD) has created challenges in understanding the nutritional needs in patients presenting with both conditions. Nutrition therapy involves a careful assessment of nutritional status, developing a treatment plan, and educating clients about the need for and function of foods. Many patients also need help overcoming mental and physical barriers that may impede nutrition recommendations. Recognized (DSM-5) EDs include anorexia nervosa (AN), bulimia nervosa (BN), binge eating disorder (BED), as well as eating disorders "other specified" and "unspecified." Many researchers have linked "food addiction" to BED and obesity, noting similarities between addiction to substances and foods (see Chap. 13). Common substances include alcohol, opiates, and stimulants such as cocaine and methamphetamine as well as diet pills (i.e., fat blockers and appetite suppressants), laxatives, and diuretics. It is difficult to explore every possible nutritional deficit present in each stage of eating disorder severity, as well as untangle the effects of multiple nutrient deficiencies associated with polysubstance abuse. However, it is possible to arrive at sound recommendations based on the evidence that is currently available.

23.2 Nutrition Assessment

A comprehensive nutrition assessment includes a detailed food and beverage intake including supplements, medications, food allergies, health history, gastrointestinal and bowel function, nutrient deficiencies, substance use history (including nicotine and caffeine), and activity level. The dietary intake assessment should include current food intake as well as a consumption history over the past 6–12 months. A review of the environment related to food acquisition, food handling, and knowledge of nutrition and food preparation is also recommended. A computerized dietary analysis can confirm poor intake of both macro- and micronutrients and can be useful to establish baseline levels of nutrient consumption. This information can

be used for nutrition education—to track changes over time in treatment-resistant patients. Dietary analysis should be repeated at regular intervals in order to monitor and evaluate progress, as well as provide documentation for healthcare providers.

23.3 Anorexia Nervosa

AN is characterized by restrictive food intake, with some patients being concerned specifically with fat or carbohydrate consumption, while others more with overall caloric intake. Compromised health related to AN results from starvation and psychiatric impairment (see Chap. 15). AN often includes overevaluation and delusional outlook concerning body image and foods, plus psychiatric symptoms of anxiety, depression, and obsessive–compulsive disorders. Nutrition assessment for determining an appropriate kilocalorie range for weight gain should consider age, activity level, and medical status. Achieving adequate energy, micronutrient, and protein intake should reverse the medical complications of protein-calorie malnutrition. Kilocalorie and protein intake is preferred orally but often requires registered dietitian nutritionists and therapists who specialize in helping patients overcome their resistance to eating. Tube feeding can be implemented for food-resistant patients in hospital or licensed residential settings. Age, degree of severity of illness, length of time of illness, and family resources can be important considerations when assessing intervention strategies. In order to maximize resources, hospital refeeding protocols have recently progressed from conservative to more aggressive in order to achieve weight gain and restore health in the least amount of time (Golden, Keane-Miller, Sainani, & Kapphahn, 2013).

Medical nutrition therapy for AN described by Waterhous and Jacob (2011) involves an assessment of macro- and micronutrient intakes, potential for refeeding syndrome, and recommendations for supplements when necessary. Refeeding for patients with AN involves increasing caloric intake until weight gain is achieved while continuing to monitor calorie and protein intake until appropriate target weight range is met and medical abnormalities have been corrected. It is not possible to anticipate which patients will develop refeeding syndrome, as it is not practical to determine whole body phosphate levels when various degrees of starvation have developed. Serum phosphate can be in the normal range and then can drop as feeding progresses, hitting a low point several days after initiation of refeeding. Medical staff should monitor electrolytes including phosphate, potassium, sodium, and magnesium and replenish as needed. Magnesium levels should be checked on admission and weekly for the first 3 weeks of refeeding at a minimum (Birmingham, Puddicombe, & Hlynsky, 2004). There are reports of an elevated anion gap (indicating metabolic acidosis) serving as an initial indicator of refeeding syndrome, which normalizes once feeding is initiated (Singla, Perry, & Lavery, 2012).

Inadequate intake of vitamins and minerals accompanies restrictive eating patterns. Thiamine deficiency can occur rapidly due to its short half-life (9–18 days) and can cause oculomotor and visual sensory disturbances in AN patients

(Mroczkowski, Redgrave, Miller, McCoy, & Guarda, 2011). Riboflavin and vitamin B6 deficiency has been observed in one-third of patients with either AN or BN, associated with low weight and cognitive dysfunction, which has been corrected without the use of supplements (Rock & Vasantharajan, 1995). Although rare, both pellagra (niacin deficiency) (Jagielski, Tomaszewicz-Libudzic, & Brzozowska, 2007) and scurvy (vitamin C deficiency) (Christopher, Tammaro, & Wing, 2002) have been reported in AN. In one study, 50 % of patients treated for AN had vitamin D deficiency, and 40 % of those were also vitamin K deficient, as documented by undercarboxylation of osteocalcin (Suzuki, 2013). Zinc deficiency has been reported which may exacerbate reduced food intake via interaction with neuropeptide Y (Levenson, 2003). Differences in serum zinc status among AN patients are likely attributable to food preferences (Zepf et al., 2012). In some cases, zinc deficiency has been linked to the emergence of acrodermatitis enteropathica (Kim et al., 2010). Other dermatological signs and symptoms are important indicators of vitamin and mineral deficiencies; therefore, clinicians should familiarize themselves with the following cutaneous manifestations: xerosis, lanugo-like body hair, carotenoderma, acne, hyperpigmentation, seborrheic dermatitis, acrocyanosis, perniosis, petechiae, livedo reticularis, interdigital intertrigo, paronychia, generalized pruritus, acquired striae distensae, slower wound healing, prurigo pigmentosa, edema, linear erythema craquele, and acral coldness (Strumia, 2005).

Supplementation with a multivitamin/mineral is usually advised, and individual vitamins and minerals may be used if laboratory data or physical exam warrants specific interventions. For example, additional calcium, magnesium, and vitamin D are given if vitamin D deficiency is detected and/or if DEXA (dual-energy X-ray absoptiometry) indicates osteopenia. While vitamin supplementation is warranted, sufficient food intake and adequate weight gain remain the most important determinants of bone health, as it will replenish estradiol (antagonizes bone resorption efforts) and insulin growth factor (which is osteogenic).

23.4 Bulimia Nervosa

Cessation of binge and purge behaviors is the primary goal of treatment for BN. A comprehensive evaluation of dieting/restricting behaviors should allow the clinician to assess behavior patterns including purging methods. Purging can include self-induced vomiting (rumination/regurgitation), food restriction, substance use/abuse, and compulsive exercise. Self-induced vomiting can compromise metabolism and nutritional status including hypomagnesemia, hypokalemia, hyponatremia, and metabolic acidosis (Mehler & Anderson, 2010). Because BN is often characterized by a normal weight presentation, observation of the cutaneous signs and symptoms listed in the AN section can help clinicians detect the presence of BN. Common markers of self-induced vomiting include Russell's sign (knuckle calluses) and elevated liver enzymes (i.e., amylase) (see Chap. 15). Symptoms arising from laxative or diuretic abuse include adverse reactions to drugs (Strumia, 2005). Dieting/restricting in BN leads to excess appetite, neural dysregulation, and

finally bingeing, which can perpetuate feelings of guilt, shame, and subsequent purging behavior (Hagan et al., 2002). Excess intake of simple carbohydrates can lead to multiple daily insulin surges and drops, which have a direct impact on appetite, metabolism, and mood. Nutrition therapy includes advising patients about the importance of regular food intake throughout the day in order to achieve a normal and consistent blood glucose level and to eat adequate amounts of protein-containing foods to achieve and maintain satiety. Correction of nutrient deficiencies and normalization of eating patterns and macronutrient intake should occur during treatment.

23.5 Binge Eating Disorder

Similar to BN, many patients with BED will restrict food consumption and thereby stimulate a natural physiological response to food scarcity that leads to rebound overeating. However, patients with BED will not exhibit compensatory purging behavior. The mixing of strange food mixtures, known as "concocting," is seen in famine conditions and in historic experiments involving semi-starvation (Kalm & Semba, 2005). Recent research suggests that dietary restraint is a major predictor of concocting in people with varying traits of binge eating and dieting (Boggiano, Turan, Maldonado, Oswald, & Shuman, 2013). Nutrition interventions should focus on changing patterns of eating so that a regular sustainable amount of energy (individually estimated from predictive equations) could be eaten throughout the day. Furthermore, teaching patients to avoid any restricting and "dieting" can be beneficial (Stice, Davis, Miller, & Marti, 2008). However, some people who describe themselves as "food addicts" benefit from abstaining from certain foods based on clinical observation of patients who report success in 12-step programs.

The more practical and realistic approach for patients with BED includes "making peace with food" without implementing any specific dietary restrictions. Using techniques learned through cognitive behavioral therapy (CBT), mindful eating, and intuitive eating, some individuals with BED regain the ability to regulate their intake of "binge foods" (Kristeller & Wolever, 2011). These individuals may not meet Yale Food Addiction Scale criteria for "food addiction," which in many cases is characterized by an abnormal physical reaction or "craving" after introducing the substance followed by an unpredictable and erratic ingestion pattern (Gearhardt, Corbin, & Brownell, 2009). Roughly half of obese patients with BED meet food addiction criteria (Gearhardt et al., 2012). Whether or not a clinician recommends the inclusion of "highly palatable" foods (typically refined grains with added sugar, salt, and fat) depends on the individual client and how they respond to certain foods. It is not realistic to exclude desserts and snack foods permanently for all individuals, but in many cases these foods should not be included regularly if they are a risk factor for bingeing. There are clinical programs that do not allow highly palatable foods, because of the potential to create anxiety or trigger bingeing (O'Toole, 2010). Monitoring diet over time allows the treatment team to determine how certain foods impact appetite and behavior.

Cognitive behavioral therapy leads to the greatest remission rates for BED, although it is not always associated with weight loss (VanBuskirk & Potenza, 2010). A weight-neutral approach to BED that incorporates body acceptance and Health at Every Size (HAES) concepts may reduce shame-based thinking and has been endorsed by the Academy of Nutrition and Dietetics (Ozier & Henry, 2011). These approaches focus less on weight as the sole indicator of health and more on the overall wellness of the individual, focusing on health behaviors and other measures such as blood pressure. Programs targeting eating behaviors, stress and lifestyle management, increased physical activity, and counseling offer the greatest efficacy for people who desire weight loss.

CBT-Enhanced (Fairburn, Cooper, & Shafran, 2003) uses a regular eating plan of three meals and two to three snacks per day in treatment of binge eating. Herrin and Larkin (2013) have outlined a food plan for disordered eaters that include three meals and three snacks, with a complex carbohydrate, a calcium-containing food, and a serving of fruit or vegetable at each meal. Protein and fat servings are required at lunch and dinner but are considered optional for breakfast. A "fun food" is offered at lunch and dinner but should be selected in conjunction with the dietitian and treatment team. This plan may be modified for those who identify with food addiction because of the "fun food" inclusion, perhaps selecting a savory food for those who have trouble consuming sweetened foods moderately. Additionally, it may be effective for those struggling with moderation to include more protein and fat at the breakfast meal. Research suggests that balanced and regular intakes at all meals, including protein, low glycemic carbohydrate, and fat, act to provide satiety for the longest amount of time, thus reducing food-seeking behaviors (Herrin & Larkin, 2013). A diet of 30 % calories from protein has been shown to sustain satisfaction from eating and delay hunger (Jonsson, Granfeldt, Erlanson-Albertsson, Ahren, & Lindeberg, 2010). Research on carbohydrate craving demonstrates that combining protein and carbohydrate at snacks reduces cravings for carbohydrate (Corsica & Spring, 2008).

23.6 Alcohol Use Disorders

According to the Centers for Disease Control (CDC), a standard alcoholic beverage is equal to 14.0 g (0.6 ounces) of pure alcohol. Generally, this amount is found in:

- 12 ounces of beer
- 8 ounces of malt liquor
- 5 ounces of wine
- 1.5 ounces or a "shot" of 80-proof distilled spirits/liquor (gin, rum, vodka, whiskey, etc.)

Moderate drinking defined by the CDC is one drink per day for women and two drinks per day for men. Heavy drinking is defined as exceeding these amounts per day. Refer to Chap. 14 for details about assessment of alcohol use disorders (AUD).

Ethanol (alcohol) has an energy value of 7.1 kcal/g, does not require digestion, and is absorbed throughout the gastrointestinal (GI) tract, primarily the duodenum. Similar to restrictive ED, excessive drinking can lead to malnutrition through a reduced intake of foods and/or imbalanced dietary intake, commonly referred to as primary malnutrition. Secondary malnutrition occurs when there are alterations in absorption, metabolism, utilization, and excretion of nutrients, which can be due to compromised oral, gastrointestinal, circulatory, metabolic, and neurological health. Dietary analysis can reveal missing foods that contribute micronutrients, but will not pinpoint secondary causes of micronutrient deficiencies. Increased swelling of the gut can cause decreased absorption of nutrients and increase exposure of toxins to the liver. GI inflammation may be responsible for reduced appetite in chronic alcoholics. The small intestine, liver, and pancreas, which are involved in the digestion and absorption of nutrients, are major organs compromised in long-term alcohol use (Griffith & Schenker, 2006).

Alcohol can interfere with the uptake of essential amino acids thereby compromising protein status, leading to a negative nitrogen balance. Protein-calorie malnutrition and numerous micronutrient deficiencies are common (Halsted, 2004), and special attention should be paid to thiamine levels, which usually require immediate supplementation to overcome dangerous deficits that can lead to Wernicke-Korsakoff syndrome, a state of psychosis also referred to as alcoholic encephalopathy. Doses of 50 mg/day are recommended, parenterally if necessary, until an oral dose of that magnitude can be taken (Lieber, 2000). One group of researchers suggested daily intramuscular supplementation with thiamine 250 mg for 3–5 days to avoid encephalopathy (Thomson & Marshall, 2006).

Vitamin and mineral deficiencies may be further assessed through conventional lab testing and examination of clinical signs and symptoms. Interpretation of lab results must consider that some nutrients such as vitamin B12 may be normal or elevated in the serum of AUD patients, yet depressed in the liver due to decreased uptake by hepatocytes (Halsted & Medici, 2011). Deficiencies of B12 and folate can cause optic neuropathy (Mroczkowski et al, 2011). Folate deficiencies may be partly due to acetaldehyde (a by-product of ethanol), which has been shown to catabolize folate (Homann, 2001), or due to alcohol's inhibitory effects on absorption and/or interference with renal excretion and enterohepatic reabsorption (Porter & Kaplan, 2011).

23.6.1 Alcohol and Metabolism

Metabolism of all three macronutrients (carbohydrates, protein, fat) is adversely affected by heavy drinking. Overall macronutrient intake is depressed as alcohol approaches 50 % of total calories consumed and as a decreased desire for food takes place (Griffith & Schenker, 2006). As the liver's functional mass is reduced and

fatty tissue accumulates, there is less storage space for glycogen (the major storage form of carbohydrate); therefore, this critical energy reserve becomes less available. Reduced glycogen reserves cause the breakdown of muscle proteins for energy, which may underlie the muscle wasting and negative nitrogen balance seen in alcoholic liver disease (Griffith & Schenker, 2006).

Recent research indicates that metabolism of methionine by the liver is adversely affected by chronic ethanol exposure and this underlies much of the hepatic damage (Halsted & Medici, 2011). Methionine metabolism is dependent on vitamins B12, B6, and folate, all of which are negatively affected by ethanol. Damage from moderate to heavy alcohol consumption affects metabolism in the liver, which stores, activates, and transports many nutrients, including fat-soluble vitamins A, D, E, and K, as well as zinc, iron, copper, vitamin B12, and magnesium. In the liver vitamin D is activated, β-carotene converts to vitamin A, and folate converts to 5-methyl tetrahydrofolic acid. Damage to the liver compromises metabolism of these micronutrients, thus deficiencies may occur (Lieber, 2000). Generally, a complete multivitamin/mineral supplement is sufficient to augment the diet upon cessation of alcohol.

Alcohol causes a depressed glucose response mediated in part by damage to glucoreceptors in the hypothalamus or persistent alterations in cellular energy regulation, all of which have implications for feeding and appetite (Hasselblatt et al., 2006). Higher baseline glucose levels predicted increased days of heavy drinking in human subjects (Leggio, Ray, Kenna, & Swift, 2009). AUD can lead to higher blood glucose levels, which can lead to glucose intolerance, insulin resistance, metabolic syndrome, and cardiovascular disease. Thus, glucose manipulation to modulate insulin resistance could play a role in treating patients with AUD (Leggio et al., 2009). Carbohydrate consumption may vary in those with AUD versus the general population. Research indicates a higher preference for sweets (Krahn et al., 2006) and higher glycemic carbohydrates (Kampov-Polevoy, Garbutt, & Janowsky, 1997) in abstinent alcoholics, which may be linked to an increased reward from dopaminergic pathways (Blum et al., 1996). According to Alcoholics Anonymous (2001), originally published in 1939, chocolate/sweets help alcoholics in recovery confront cravings for alcohol. Findings from Umhau et al. (2002) suggest that while this may be true during initial withdrawal, it is not always the case after 6 or more months of abstinence.

Alcohol also has profound effects on lipid metabolism including increased fatty acid mobilization from adipose tissue, increased liver synthesis of fatty acids and triglyceride production, a decrease in fatty acid oxidation, and an overall trapping of triglyceride in the liver (Yeomans, 2010). Fatty liver is related to an increase in NADH (reducing coenzyme) responsible for several metabolic shifts. Liver disease decreases production of bile salts, necessary for absorption and digestion of lipids. Alcoholic liver disease (ALD) (see Chap. 15) progresses through distinct stages as alcohol abuse continues, progressing from fatty liver (hepatic steatosis) to hepatitis, to fibrotic alterations, and finally cirrhosis. Protein deficiency exacerbates the effects of ethanol by impairing lipoprotein secretion from the liver (Lieber,

2000). While most individuals who drink heavily will develop fatty liver, not all individuals develop advanced stages of liver disease (Griffith & Schenker, 2006).

23.6.2 Interventions for AUD

There is evidence showing that saturated fatty acids and fructose can contribute to the development of nonalcoholic fatty liver disease (NAFLD), and therefore reduction of these foods is advised when counseling a person with history of AUD (Carvelhana, Machado, & Cortez-Pinto, 2012). Rat studies have shown consistent associations between fructose intake and liver damage, including fatty liver, which is exacerbated by marginal copper deficiency (Song, Schuschke, Zhou, Chen, Pierce, et al., 2012; Song, Schuschke, Zhou, Chen, Shi, et al., 2013). Copper transporter synthesis is deregulated by feeding fructose to rats, inducing copper deficiency. Since liver damage is exacerbated by marginal copper deficiency, it is prudent to recommend low fructose consumption by individuals who are at risk for liver damage. Fructose intake can be reduced by avoiding sweets and processed foods/beverages such as soda and common snack foods, which often contain high-fructose corn syrup (HFCS). Teaching patients to monitor for added sugars and staying below a certain range (30–35 g/day for men and 20–25 g/day for women) can be useful for treatment. Discussion of hidden sugar in foods is helpful.

An individualized diet of nutrient-dense foods from all of the five food groups, with supplementation when indicated, is the most effective treatment for macro- and micronutrient deficiencies. The use of supplements should be based on objective evidence and individual nutritional assessment, as opposed to random, overzealous administration. Guidelines have been suggested for those with ALD, and these can also apply to people with AUD who do not have ALD, as these guidelines have been designed to correct protein-calorie malnutrition:

- Protein: 1.0–1.5 kg/body weight.
- Total kilocalories: 1.2–1.4 times resting energy expenditure (determined by height, weight, and gender). Minimum of 30 kcal/kg with 50–55 % (primarily complex carbohydrate), 25–30 % fat (mainly unsaturated fat), and provide adequate essential fatty acids (protein with high biological value).
- Salt and water should be adjusted for patient's fluid and electrolyte status.
- Provide multivitamin/mineral supplements.
- Provide the conditionally essential amino acids choline, cysteine, taurine, and tyrosine to those with cirrhosis (the body cannot synthesize these amino acids given the status of their liver and hence the term conditionally essential).

(Adapted from Griffith & Schenker, 2006)

There is also interest in the use of antioxidant supplementation since much of the damage from chronic ethanol ingestion is due to increased production of reactive oxygen species. In general, the use of the "anti-inflammatory dietary approach" can be useful (Dean & Hansen, 2012). This dietary regimen promotes liberal amounts of produce, whole grains, use of antioxidant herbs and plants (onion, garlic, turmeric, cayenne), and reduced amounts of added sugars and saturated fats. This approach has been advocated to reduce low-level chronic inflammation and oxidative stress.

23.7 Alcohol Use Disorders and Eating Disorders

Abstinence from alcohol may partially restore appetite and awareness of hunger-satiety cues, which are critical components of treatment for people with AUD and ED, either separately or in combination. Alcohol stimulates appetite at low doses, but as intake increases, appetite is suppressed and alcohol is substituted for food. This is especially dangerous for a person who has AN. Both AUD and AN can put individuals at risk for refeeding syndrome. Persons with either or both of these conditions should be evaluated for potential refeeding syndrome in the early stages of treatment. Similar to AUD, thiamine deficiency is prevalent in patients with AN and has been implicated in some of neuropsychiatric symptoms observed in AN (Winston, Jamieson, Madira, Gatward, & Palmer, 2000). Vitamin B6 deficiency, hypomagnesemia, and low serum zinc have also been observed in both AUD and AN. While the association between decreased vitamin B12 and AUD has been documented, AN patients who restrict animal products are also likely to present with deficiencies in vitamin B12. It will be difficult to determine which disorder is the primary cause of malnutrition when there is a dual diagnosis.

Individuals with BN who have a dietary pattern of cyclic restricting and bingeing have impaired appetites, and alcohol abuse will further compromise hunger and fullness cues. Additionally, a person diagnosed with BN may find that binge drinking can facilitate self-induced vomiting; thus, monitoring for binge drinking and binge eating with subsequent purging is an important part of ongoing care. Similar to AUD patients with liver damage, alcoholic patients who binge eat can be helped by reducing fructose, which can cause increased insulin secretion followed by decreased blood glucose, which may then increase appetite and/or have a negative effect on mood. Additionally, the ingestion of highly palatable foods such as sweetened dessert items can stimulate reward pathways in the brain in a manner similar to reward sensations caused by substances such a nicotine and cocaine (Volkow & Wise, 2005) (see Chap. 4), suggesting that overuse of such foods can perpetuate cycles of binge/purge seen in BN and/or binge/guilt seen in BED.

There is a growing body of research that shows people with AUD in rehabilitation respond favorably to nutrition education (Barbadoro et al., 2011; Cowan & Devine, 2012; Grant, Haughton, & Sachan, 2004), but this does not extend to patients with co-occurring AUD and ED. Malnutrition symptoms of AUD and

ED can be similar (making diagnoses difficult), but identifying the primary disorder can be important for developing effective treatment. Occasional drinking in ED patients should be closely monitored because of the potential for alcohol (even at low doses) to adversely affect appetite, food preferences, and inhibition. Underreporting of alcohol consumption is common and should be respectfully considered. Based on clinical observation, many individuals with EDs use addiction treatment to mask the ED, while some AUD and SUD patients end up in ED treatment.

23.8 Substance Use Disorders

Malnutrition impacts all body systems including the immune system, leading to an inadequate response to disease. While the negative effect of alcohol on nutritional status has been well described, mechanisms behind illicit drug-induced malnourishment remain largely unknown. It is difficult to differentiate between primary and secondary malnourishment within drug-addicted populations. In addition, nearly 40 % of substance abuse treatment admissions nationwide report polysubstance abuse (Substance Abuse and Mental Health Services Administration, 2011), which creates challenges in reaching sound conclusions about the effects of individual drugs. Most of the data that links nutritional deficiencies to substance abuse is speculative, underpowered, and retrospective. The current evidence linking nutrition to substance abuse in humans is summarized below with recommendations for treatment.

23.8.1 Polysubstance Abuse

Much like AUD patients, SUD patients suffer from calorie and protein malnutrition. Santolaria et al. (1995) found malnutrition to be related to female sex, intensity of drug addiction, and anorexia, as determined by poor food and drink consumption. These authors speculate that increased infection rates in this population may contribute to malnutrition by the subsequent hypermetabolic (catabolic) states. Islam, Hoassain, and Ahsan (2001) analyzed 253 treatment-seeking male polysubstance abusers and found that their vitamin A, C, and E levels were significantly lower than nonaddict controls. These authors recommend initiating antioxidant vitamin therapy to drug addicts who are at higher risk of infection. A follow-up analysis of the same population concluded that illicit drug use impairs serum mineral value, causing an increase in copper and zinc and a decrease in iron (Hossain, Kamal, Ahsan, & Islam, 2007). The authors recommend a careful micronutrient intervention in the clinical management of drug-dependent patients, recognizing that nutritional deficiencies can induce immunodeficiency and may influence susceptibility to other infections including HIV. Using 24-h dietary recalls and interviews with 20 female intravenous (IV) drug users, more than half of the foods consumed could not be classified into the "food group" system

(Baptiste & Hamelin, 2009). Within the food groups, the women reported consumption of easily ingested and digested foods such as cereals, having difficulty with eating raw vegetables and meat. Digestive issues and preference for hedonistic foods rich in sugar, salt, and fat appear to be common themes among drug addicts, regardless of their "drug of choice" or geographic location.

Added sugar accounted for 30 % of the energy intake of drug addicts (not in treatment) living in Norway ($n = 220$), with results mirrored in biomarkers (Saeland et al., 2011). Sugar and sugar-sweetened foods were preferred by over 60 % of the respondents. Nearly every subject failed to meet recommended intake of vitamin D, and 70 % did not reach the lower reference value for 25-hydroxyvitamin D3 after blood testing. Vitamin C levels were low, and suboptimal polyunsaturated fatty acid intake was also identified. These researchers also discovered elevated serum copper suggesting inflammation among respondents, a finding that is in agreement with elevated C-reactive proteins as documented by Hossain et al. (2007). In another study, Ross, Wilson, Banks, Rezannah, and Daglish (2012) examined 67 detox patients to show that half of all subjects were deficient in iron or in vitamins, specifically antioxidant vitamins A and C. Of those found to have low potassium, over 90 % were alcohol dependent. These researchers noted that plasma albumin and total protein levels are not good indicators of nutritional status in drug-addicted populations. The prevalence of malnutrition in their patient population is likely to be underestimated because administration of oral multivitamin supplementation and parenteral thiamine is routine upon admission.

23.8.2 Opiates

Individuals under the influence of heroin eat infrequently and have little interest in food. Food consumption is most commonly quick, convenient, cheap, and sweet foods (Neale, Nettleton, Pickering, & Fischer, 2012). Heroin is an appetite suppressant, reduces gastric motility (White, 2010), and delays gastric emptying via impaired gastrin release. These conditions are reversible after cessation of the drug. Many users report constipation while using, diarrhea while detoxifying, and GI abnormalities for up to several months. Because of GI discomfort, foods commonly consumed are low in fiber, easily digestible, and calorically dense, such as ice cream. Fibrous fruit and vegetable consumption is generally low. Compromised gut health leading to impaired absorption of amino acids, vitamins, and minerals is a contributing factor to secondary malnutrition. Varela, Marcos, Santacruz, Ripoll, and Requejo (1997) documented malnutrition (determined by immune function in response to seven antibodies) in 36 heroin-addicted females prior to stopping the drug. In a classic study of heroin addicts ($n = 149$), 45 % were deficient in vitamin B6, 37 % in folate, and 19 % in thiamine (Nakah, Frank, Louria, Quinones, & Baker, 1979). Findings of significantly lower vitamin B6 levels have been replicated (Heathcote & Taylor, 1981). Other investigators have reported low levels of retinol (vitamin A), α-tocopherol (vitamin E), folic acid, potassium, and selenium and elevated levels of copper in opiate addicts (Estevez et al., 2004;

Hossain et al., 2007). Meanwhile, previous research has linked heroin addiction to hyperkalemia (Mohs, Watson, & Leonard-Green, 1990), and it is likely that the findings of low potassium were due to concurrent use of alcohol (Ross et al., 2012).

Preference and "craving" for sweets such as chocolate and candy are also common in patients on methadone maintenance (Nolan & Scagnelli, 2007). Furthermore, prolonged opioid dependence appears to be associated with lower than normal bone mass, as evidenced by the presence of osteopenia or osteoporosis in a sample of relatively young patients (mean age: 37 ± 7 years) on methadone maintenance (Dursteler-Macfarland et al., 2010). These authors speculate that low levels of circulating luteinizing hormone, estrogen, and testosterone or impaired adrenal function may be the underlying mechanism. Interestingly, low calcium intake was not identified, and other research on heroin addicts has failed to indicate decreased serum calcium levels. Confounding variables such as overall nutrition habits (particularly intake of foods containing vitamin D), smoking, physical activity, or alcohol use were not adequately controlled. Estevez et al. (2004) found that addicts receiving methadone had elevated levels of both magnesium and phosphorus. Since magnesium and phosphorus are both involved in bone health, it is possible that the utilization of these minerals is impacted by methadone use.

23.8.3 Stimulants

Stimulant drugs such as cocaine and methamphetamine (meth) invariably decrease appetite, prevent sleep, and can lead to significant weight loss. Methylphenidate, dextroamphetamine, and mixed amphetamine salts are prescription amphetamines used for attention deficit hyperactivity disorder (ADHD) that have a high potential for abuse, but with fewer physiological ramifications. For this reason, many ED patients gravitate towards their use. Caffeine and nicotine can also dampen sensitivity to appetite, and it has been reported that many patients with AN/BN use these substances to suppress appetite (Burgalassi et al., 2009), which is also common among AUD/SUD patients. Individuals with daily stimulant intake are more likely to "snack" than eat meals. On the other hand, individuals who restrict food intake while using stimulants may exhibit binge eating behavior after they have stopped using. Subsequent stimulant use often becomes a preferred compensatory behavior. Although not yet clinically accepted as "purging," this may represent a growing manifestation of co-occurring ED and SUD. It is important to remember that much of the research on drug addiction occurs in an inpatient setting, which represents the most severe cases. An ED patient who occasionally uses substances with subthreshold SUD is unlikely to present with the same characteristics. Similarly, drug users who use stimulants that suppress the appetite do not necessarily harbor classic psychological ED psychopathologies such as "drive for thinness" or "body dissatisfaction." For example, Curran and Robjant (2006) found that women who use ecstasy (a popular club drug classified as an empathogen or entactogen often mixed with other stimulants) are not necessarily taking it as a deliberate means of weight control.

23.8.3.1 Cocaine

Cocaine reduces appetite and can lead to nausea. Not surprisingly, affinity for high-sugar food and drink has been documented in individuals with cocaine use disorders (Janowsky, Pucilowski, & Buyinza, 2003). These investigators found that cocaine abusers within their first few days of hospitalized detoxification preferred the highest concentration of sucrose offered. The findings extend beyond "sweet liking" to suggest that cocaine users receive additional reward from increased sugar content, compared to sweet likers without history of SUD who chose the lowest concentration of sucrose solution. Inadequate food intake and preference for nutrient-poor foods contributes to primary malnutrition. Mechanisms causing secondary malnutrition resulting from cocaine use remain speculative and lack human studies. Akkina et al. (2012) found that cocaine users were more likely to have elevated blood pressure. This association was not found for users of heroin or methamphetamine.

Other research has linked low levels of omega-3 and omega-6 polyunsaturated fatty acids (PUFA) to relapse in cocaine addicts (Buydens-Branchey, Branchey, McMakin, & Hibbeln, 2003), which may stem from increased anxiety associated with low PUFA (Buydens-Branchey & Branchey, 2006). Efficacy of omega-3 PUFAs in the treatment of depression has been established in connection with the dopaminergic brain system, particularly docosahexaenoic acid (DHA) and eicosapentanoic acid (EPA), yet a recent meta-analysis suggests that the positive effects of omega-3 on depression have been overstated and can be attributed to publication bias (Bloch & Hannestad, 2012). Some authors have stated that addictive substances strip the brain of essential fats, as well as impair absorption and utilization of amino acids (AAs) necessary for neurotransmitter synthesis. Low AA levels leading to low dopamine (DA) in the brain contribute to aberrant substance-seeking behavior. In addition to DA, serotonin appears to be involved in depression and stimulant regulation. Because tryptophan converts into serotonin in the brain, and tyrosine becomes DA, several popular press authors have proposed specific AA therapies to combat addiction, but evidence suggesting long-term efficacy of such programs is lacking.

N-acetylcysteine (NAC) reduces cocaine-seeking behavior in animal models and has been proposed as possible pharmacologic treatment for relapse prevention in cocaine addiction in humans (LaRowe et al., 2007). The authors found that NAC reduced the desire to use cocaine in the presence of cocaine-related visual cues. Rather than focusing on individual AAs, increasing overall protein in the diet can promote neurotransmitter synthesis in a less urgent manner, assuming the addict is in an environment where high protein foods are readily available and eating behavior can be monitored. Consumption of protein-rich foods such as meat, fish, dairy products, and nuts is a more reasonable approach that will also promote long-term sustainable behavior change. Meanwhile, controlled trials should continue to investigate the impact of amino acid therapy in drug addiction treatment.

23.8.3.2 Methamphetamine

Recent research has indicated that meth disrupts energy metabolism by causing changes in gene expression and proteins associated with muscular homeostasis/contraction, maintenance of oxidative status, oxidative phosphorylation, and iron and calcium homeostasis (Sun et al., 2011). Pyruvate pathways are diverted away from oxidative respiration and towards fermentation to lactic acid. Numerous mechanisms have been implicated in the promotion of oxidative stress, including degradation of mitochondria by multiple pathways. According to these authors, an imbalance in calcium homeostasis due to oxidative stress is an important factor in heart disease. Investigators observed a downregulation of ferritin (iron chelator), leading to an increase in free iron, causing harmful free radicals through the Fenton reaction. Based on these findings, iron supplementation would be a contraindication for meth addicts. Animal models have shown that the antioxidant mineral selenium plays a protective role in meth-induced neurotoxicity (Imam & Ali, 2000). Additionally, coenzyme Q10 has been shown to attenuate meth and cocaine neurotoxicity in mice (Klongpanichapak, Govitrapong, Sharma, & Edabi, 2006).

A relationship between meth and dental disease has been documented in the literature, containing noteworthy implications for nutrition therapy. Over 40 % of meth users had dental or oral disease and almost 60 % had one or more missing teeth (Shetty et al., 2010). Intravenous meth users have higher rates of dental disease compared to those who smoke or snort and compared to other IV drugs (Laslett, Dietze, & Dwyer, 2008). In addition to poor oral hygiene, altered calcium utilization (Sun et al., 2011) may be one of the factors linked to high rates of dental disease, also referred to as "meth mouth." According to Hamamoto and Rhodus (2009), high intake of refined carbohydrates, high calorie carbonated beverages, increased acidity in the oral cavity from oral meth ingestion, and gastrointestinal regurgitation or vomiting also contribute to dental caries and overall poor oral health in meth abusers. Cessation of meth and subsequent improvements in nutrition and oral hygiene is the first line of treatment. Because oral health affects one's capacity to consume food, meth has the potential to impact all areas of nutrition. Dietitians must take into account the compromised ability of the patient to utilize their mouth, designing realistic interventions that monitor and evaluate potential xerostomia, chewing ability, and taste. Decreased consumption of refined carbohydrates and substitution with fruits and vegetables should be a primary goal.

23.8.4 Diet Pills, Laxatives, and Diuretics

Some of the most common substances abused by those with EDs are legal substances including diet pills, laxatives, and diuretics. Diet pills include lipase inhibitors (i.e., orlistat) and appetite suppressants. Lipase inhibitors have known side effects including fecal incontinence, flatulence, soft stools, and possible malabsorption of fat-soluble vitamins. Appetite suppressant abuse is more common and can lead to insomnia, high blood pressure, tachycardia, restlessness, and constipation (US Department of Health and Human Services, 2013). Cessation of diet pill

use is necessary for successful treatment, in order to restore the appetite and ameliorate the metabolic effects. Nutrition education about the effects of diet pill use and appetite suppression is warranted along with education about normalization of food intake. A multivitamin/mineral supplement along with a diet addressing the potential protein-calorie malnutrition would be the primary nutrition intervention.

Laxative abuse is common in those diagnosed with ED, ranging from 10 to 60 % of individuals in this group (Roerig, Steffan, Mitchell, & Zunker, 2010). Chemically, laxatives fall into several categories including stimulants, osmotic/saline, fiber/bulking, and surfactants, with the stimulant laxatives having the highest potential for abuse and medical consequences. Cessation of stimulant laxatives is associated with rebound edema via the renin-aldosterone system. Medical stabilization includes close monitoring of serum electrolytes and acid/base balance. Nutrition therapy involves a program to restore normal bowel function, which should include education about hydration and inclusion of high fiber foods. Hydration needs are assessed as laxative use is gradually discontinued. Education about the transient nature of rebound edema is necessary for those with ED due to their great concern about the added "weight," which is actually regained fluid as opposed to fat mass.

Diuretics are another class of substances frequently misused by those with EDs. These substances cause renal sodium retention, and edema occurs in a rebound fashion when the diuretic is discontinued. Medically, diuretics need to be tapered gradually. Nutrition intervention should include education regarding the fact that diuretic use or misuse is not associated with permanent weight loss, as this is a common reason they are abused. Nutrition education about hydration needs and overall balanced diet is appropriate. Diuretic use is associated with increased risk of kidney stone formation, a potential risk that can be partly managed by diet. If calcium oxalate, cystine, or uric stones have been identified, the food plan may be adjusted to be more alkaline with greater inclusion of fruits and vegetables, supplemental citrate, or drinking mineral water that is alkaline. Similarly, to reduce incidence of calcium phosphate or struvite stones, use of cranberry juice or betaine has been shown effective (Frassetto & Kohlstadt, 2011).

23.8.5 Interventions for SUD

In general, the goal of nutrition therapy in SUD patients should emphasize correcting associated nutrient deficiencies, utilizing laboratory studies to warrant more precise interventions. Most importantly, the primary goal for recovery should be complete abstinence from all illicit and/or nonprescribed mind-altering substances. If an individual begins to use again, efforts at correcting nutrient deficiencies are futile, as they are likely to redevelop; therefore, the primary aim of a nutrition intervention should be to support recovery by any means necessary. Once sobriety has been achieved, altered biochemistry and dysfunctional behavior resulting from substance abuse often persists. Appetite and taste return in the post-drug state, which may be due to a rebound effect following prolonged suppression

of the hypothalamus from drug abuse. The practice of making healthful food choices after achieving sobriety may be very challenging in the early stages of recovery, particularly if a tobacco use disorder is present. In addition to creating oxidative stress, tobacco use negatively affects oral health and will therefore impact taste/smell and food intake; thus, cessation should become an important goal. Caffeine intake should be monitored but complete abstinence is not always indicated. Nutrition education should be used as a helpful rather than punitive component of recovery. Nutrition counseling can be used as an effective adjunctive approach towards tobacco and caffeine cessation or reduction by highlighting discrepancies between current behaviors and overall treatment goals.

Immediate intervention with pills and other supplements may not necessarily support recovery, even though there are some physiological advantages. The first goal is to help the patient get past the immediate crisis, which may require a liberalized diet. The use of supplements should be used with caution, as drug addicts are likely to prefer a "quick-fix" approach to recovery and avoid a painful introspective recovery process. Additionally, nutritional supplements may give patients the idea that as long as they are taking their pills, they have no need to improve their eating habits. Finally, street drugs can exert tremendous strain on the liver, and supraphysiological doses of nutrients may actually conflict with the healing process, with potential for hypervitaminosis. Eating behavior should be the primary intervention, and supplements should be used secondarily, unless there is a severe micronutrient deficiency that warrants aggressive intervention, such as thiamine deficiency in alcoholics.

Nutritional deficiency lowers the antioxidant potential of cells and may increase potential for cell damage. In addition to needing increased amounts of antioxidant vitamins, individuals recovering from clinically significant SUD are likely to have higher protein needs than the general population to promote neurotransmitter synthesis, unless contraindicated by a medical condition such as renal disease. Excessive carbohydrate consumption can lead to an unpredictable physiological response such as cravings or subsequent low blood sugar, especially from high glycemic refined grains and sugars. An ideal macronutrient breakdown would be 45–50 % carbohydrate, 25–30 % protein, and 20–30 % fat. Of the carbohydrates consumed, a minimum of 75 % should come from whole grain, fruits, vegetables, and dairy, with some leeway for added sugars or refined grains in the early stages of recovery. As individuals approach 6 weeks of abstinence, sugar use can be reevaluated, with the goal of preventing intake from increasing. Maintaining tight control of blood glucose can prevent the development of impaired glucose tolerance and decreased insulin sensitivity and prevent hypoglycemic mood swings that may increase relapse risk.

For individuals with a history of SUD (including AUD), the gradual reintroduction of fiber-rich foods into the diet will be of paramount importance. Long periods of low fiber intakes create significant barriers for nutrition therapy that involves fruits, vegetables, whole grains, and beans. Fiber intake should increase on average by 2–4 g/week in order to meet recommendations of 38 g/day for men and 25 g/day for women ages 14–50. Focusing on improved gut health will promote optimal

absorption of amino acids, vitamins, and minerals, and probiotic supplements can prove to be beneficial. Fiber supplements should be used to maintain gradual and progressive weekly increases if oral intake is poor. Increased water intake should accompany increased fiber with a goal rate of 2–3 L/day.

It is very likely that compromised gastrointestinal function creates barriers for absorption of supplemental vitamins. In this case, liquid forms of vitamin supplements can be very useful, and meal replacement drinks will provide protein in addition to the vitamins and minerals. Increased serum copper observed in drug addicts is likely due to a rise in ceruloplasmin, and some investigators have noted that serum copper concentrations are not the best index of total body copper (Bo et al., 2008). It is unknown if copper chelating agents would be a useful intervention. Meanwhile, multivitamins with low metal content should be used. Antioxidant compounds such as coenzyme q10, alpha lipoic acid, resveratrol, and flavonoid polyphenols can be considered as well.

23.9 Substance Use Disorders and Eating Disorders

Restriction or reduction of added sugars and highly refined grains may be indicated for some SUD patients actively seeking recovery from binge eating, if they have identified these foods as problematic, as commonly seen in food addiction. This is a well-known approach for individuals who choose to seek support from one of the many food-based 12-step programs. In other cases, extreme restriction of processed carbohydrates can lead to the development of disordered thinking patterns including "orthorexia" (i.e., eating only foods that one considers healthy) or can lead to rebound bingeing. Limiting processed foods in SUD patients is at odds with nutrition therapy for AN/BN, where patients often need to recover from their extreme apprehension against eating nonnatural and nonorganic foods. Promoting a meal plan to a person recovering from ED needs to be done carefully so the patient is not hearing a message to restrict. Patients with AN/BN need coaching to see food as beneficial instead of threatening, which involves letting go of strict rules around eating and adopting more flexible, moderate approaches.

It is important to be realistic about nutrition interventions for those in recovery from substance abuse. An individual who has been using alcohol or illicit drugs for several years may enter treatment with behavior that resembles BED. It is easy to assume that an ED is present. If the binge eating persists after the first month of recovery, the potential for ED should be assessed by a qualified professional and addressed in treatment. There is a difference between an individual with SUD who binge eats and a patient with BED who uses drugs; however, many individuals display characteristics of both. A true SUD patient may only binge eat during the early stages of abstinence from drugs and may not continue those behaviors once sobriety has been achieved. False positives can also occur in the reverse situation, where a true BED patient presents with a history of substance abuse but does not meet full criteria for SUD. In other words, the patient will use substances when food is not available but the impairment, distress, and social consequences actually stem from the dysfunctional relationship to food rather than the alcohol/drugs.

The healthcare team should carefully consider the potential conflict in nutrition therapy for ED versus SUD when individualizing treatment. Many patients with a history of SUD will have an aversion to processed foods because it acts on their brain similarly to drugs, leading to overconsumption. Additionally, certain elements of processed food such as dyes and other additives may interfere with biochemical processes. Drug addicts should not be forced to eat highly palatable foods under the guise of protection from potential ED. For these reasons, the recommendation of 75 % unrefined carbohydrates (focus on complex carbohydrates) will keep blood sugar stable and support recovery from SUD while offering protection against development of ED, in most cases. An anti-inflammatory type diet will be conducive to regaining health. Food should precede supplementation, which should eventually be discontinued after approximately 6 months of sobriety and balanced dietary intake from all food groups. Long-term use of supplemental vitamins remains a controversial issue, but can be indicated for individuals who are unable to obtain all of the essential nutrients from food.

Specific Recommendations for SUD Categories

- *Polysubstance Abuse Involving Alcohol*: Multivitamin (low metal), additional B vitamins (primarily thiamine for AUD), and diet rich in vitamins A, C, E, selenium, and iron. Probiotics if gastrointestinal distress is present
- *Opiates*: Liquid multivitamin (low metal), additional vitamin B6, additional calcium/vitamin D, digestive enzymes, probiotics, fiber supplement if constipated, and diet rich in vitamins A, C, E, selenium, and iron
- *Cocaine*: Multivitamin (low metal), omega-3 supplement rich in DHA, protein-rich diet, and diet rich in vitamins A, C, E, selenium, and iron
- *Meth*: Multivitamin (low metal, no iron), omega-3 supplement rich in DHA, protein-rich diet, lower refined carbohydrate intake, and diet rich in vitamins A, C, E, and selenium

Sources of Vitamin A: Carrots, pumpkin, sweet potatoes, spinach, and kale
Sources of Vitamin C: Bell peppers, kiwi, broccoli, and strawberries
Sources of Vitamin E: Almonds, sunflower seeds, turnip greens, and peanut butter
Sources of Selenium: Brazil nuts, yellowfin tuna, turkey, and halibut
Sources of Iron: Red meat, lentils, pumpkin seeds, and kidney beans

Conclusion

Clinicians working with people diagnosed with SUD and ED need to be educated about each disorder separately and then understand how the various disorders interact. Nutrition therapy recommendations should address the most serious medical and nutrition conditions first and then target psychological

aspects related to eating behavior in conjunction with the treatment team. Nutrition education is critical for SUD patients, who clearly require specialized care, whereas education for ED patients must be sensitive to their conditions and needs. Recommendations for nutrition therapy should be used as guidelines, remembering that nutritional needs are best assessed on an individual basis.

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Cognitive Behavior Therapy for Co-occurring of Eating and Substance Use Disorders

24

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Abstract

Cognitive behavior therapy has received empirical support for the treatment of eating disorders, alcohol use disorders, and substance use disorders; however, there is no empirical research on the use of cognitive behavior therapy specifically for comorbid eating disorders and substance use disorders. Common cognitive behavioral treatments of alcohol use disorder and substance use disorders include (1) cue exposure interventions, (2) contingency management interventions, and (3) coping skill interventions. Treatment for eating disorders initially focuses on establishing regular eating, changing disturbed beliefs about dieting, and the importance of shape/weight and transitions into modular-based treatment that targets a number of hypothesized maintaining factors. We present a model for combining these existing approaches into a single treatment that builds on core overlapping interventions targeting behavioral change (e.g., self-monitoring, functional analysis, etc.) and allows for use of modular-based

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integration of comorbidity-specific interventions targeting aspects of comorbidity (e.g., PTSD) and personality (e.g., impulsivity). Special considerations for treating the comorbid population are discussed.

Keywords
Alcohol use disorders • Anorexia nervosa • Bulimia nervosa • Binge eating disorder • Case formulation • Cognitive behavior therapy • Comorbidity • Substance use disorders

24.1 Introduction

As highlighted in previous sections of this volume (see Chap. 11), a substantial proportion of patients with eating disorders (ED) who seek treatment experience symptoms of another psychiatric disorders, including a notable percentage with a co-occurring alcohol use disorders (AUD) and/or substance use disorder (SUD). This chapter presents an overview of cognitive behavior therapy (CBT) for patients with comorbid ED and SUD. Information is provided about treatment efficacy in ED and SUD, treatment considerations for comorbid patients, and future directions for research on this topic.

24.2 Cognitive Behavior Therapy and Eating Disorders

Cognitive behavior therapy (CBT) is a well-validated intervention for two of the three primary ED diagnoses described in the Diagnostic and Statistical Manual of Mental Disorders (DSM-5; American Psychiatric Association, 2013): bulimia nervosa (BN) and binge eating disorder (BED). For patients with BN, typical remission rates for binge eating and purging of 30–50 % are observed, and CBT is superior to antidepressant medications for achieving abstinence from bulimic symptoms (Wilson, Grilo, & Vitousek, 2007). The effects of CBT for BN are seen not only for improvements in binge eating and purging but also for other psychiatric comorbidities (e.g., depression), self-esteem, and social functioning (Wilson, 2010). Although CBT is a treatment with long-lasting effects, a significant portion of patients with BN remain symptomatic even after receiving a complete course of treatment. An enhanced version of CBT (CBT-E; Fairburn, 2008) has been developed to address limitations noted with the original form of this treatment (Fairburn, Marcus, & Wilson, 1993). The first controlled evaluation for BN indicated that it may be a more effective treatment for complex cases of BN (Fairburn et al., 2009).

For individuals with BED, CBT results in posttreatment binge eating remission rates between 55 % and 70 %, which are generally maintained at a 1-year follow-up, and improvements are noted in specific ED symptoms and general psychopathology (Wilson et al., 2007). As with BN, CBT is more effective than pharmacological treatments for the treatment of BED (Devlin et al., 2005; Grilo, Masheb, & Salant,

2005) and is superior to behavioral weight loss treatment (Grilo, Masheb, Brownell, & White, 2007; Munsch et al., 2007).

More limited information is available to evaluate the utility of CBT for the acute treatment of outpatients with AN. Some studies have observed no differences in outcome for patients receiving CBT and other comparison therapies (e.g., Ball & Mitchell, 2004; Channon, De Silva, Helmsley, & Perkins, 1989). Further, for patients with broadly defined AN, on the primary outcome measure of global AN rating, McIntosh and colleagues (2005) found that patients receiving nonspecific supportive clinical management had significantly better outcomes than an interpersonal psychotherapy group; however, other comparisons between CBT, interpersonal psychotherapy, and nonspecific supportive clinical management were not significantly different. A long-term follow-up assessment of study participants (mean = 6.7 years ± 1.2) found approximately half of participants had a "good" outcome while the other half had a "poor" outcome. No significant differences among the three treatment conditions were found on the primary, secondary, or tertiary outcome measures (Carter et al., 2011). A more recent study found substantial increases in weight and reduction in ED psychopathology among adults with AN who completed 40 sessions of CBT-E (Fairburn et al., 2013). Specific effects of CBT have been more consistently documented in the prevention of post-hospital relapse among patients with AN (Carter et al., 2009; Pike, Walsh, Vitousek, Wilson, & Bauer, 2003).

24.3 Content of Cognitive Behavior Therapy for Eating Disorders

The first version of CBT for AN (Garner & Bemis, 1982, 1985; Garner, Vitousek, & Pike, 1997) is similar to CBT for BN (described below), but with an additional focus on issues related to low weight, weight gain (Garner et al., 1997), and increasing motivation for change and collaboration (Vitousek, Watson, & Wilson, 1998). The amount of time for the CBT also differs substantially for AN, with a recommendation of 1–2 years of treatment for patients with acute AN, and about 1 year for relapse prevention among individuals who enter treatment after weight restoration (Wilson et al., 2007). CBT for BN is a briefer treatment (16–20 sessions) delivered over 4–5 months. In the model underlying CBT for BN, the critical role of both cognitive and behavioral factors in maintaining bulimic symptoms is emphasized, with the overvaluation of body weight and shape as a cognitive schema that leads to the behavior of rigid dietary restriction (e.g., going for long periods of time without eating; Fairburn et al., 1993). Dietary restriction subsequently produces a vulnerability to additional problem behaviors, such as periodic binge eating episodes, which are characterized by a loss of control over eating. Attempts to counteract the effects of binge eating, such as self-induced vomiting and other extreme weight control behaviors, also maintain binge eating by reducing anxiety about weight gain and disrupting the development of satiety. Binge eating and purging cause distress and low self-esteem, which reinforce circumstances that

will lead to continued dietary restraint and binge eating (Fairburn, 1997; Fairburn, Cooper, & Shafran, 2003). CBT assists in the development of a regular pattern of eating and problem-solving skills for coping with high-risk situations, modifying problematic shape and weight concerns, and preventing relapse (Fairburn et al., 1993). Few adaptations are needed between CBT for BN and BED, although in the latter case, compensatory behaviors are not included as part of the model.

24.4 Cognitive Behavior Therapy and Alcohol/Substance Use Disorders

Cognitive behavior therapy for AUD/SUD has a long history of application originating with initial behavioral strategies designed to alter associations and reinforcement contingencies by capitalizing on classical and operant conditioning to alter drink/drug use behavior. A number of different CBT/BT models have garnered empirical support over time and can be generally categorized as (1) cue exposure approaches, (2) contingency management approaches, and (3) cognitive-behavioral coping skill approaches. All of these CBT approaches are considered to have empirical support (Kleber et al., 2007; McHugh, Hearon, & Otto, 2010).

The initial assessment frequently includes the use of interviews, questionnaires, and biological measures (e.g., breathalyzers, urine toxicology reports, and liver function tests) to determine the degree of impairment and overall functioning. Particular patterns (e.g., duration, intensity, and frequency) and contexts (e.g., alone, with others, physical environment, affective states, specific cognitions, or physical experiences) associated with substance use are investigated. The information gathered in the assessment can be used to develop a case formulation to understand the antecedents and consequences of substance use in addition to highlighting the maintaining factors. This case formulation illustrates the specific targets for intervention in the context of developing treatment goals.

Cue exposure is based on the principles of classical conditioning. This approach uses repeated exposure to the cues in a controlled environment with the intent of eliciting craving and a desire to engage in substance use. The goal is to reduce patients' sensitivity to the cues that are often associated with their substance of abuse (e.g., the site and/or smell of alcohol, drug paraphernalia, in addition to specific environments, moods, or situations) eventually extinguishing the response. During the exposure, patients are able to practice utilizing coping skills (discussed below) to resist the urge to engage in substance use (Monti, Kadden, Rohsenow, Conney, & Abrams, 2002; Rohsenow et al., 2001). This strategy has been shown to reduce cravings and relapse in high-risk situations for problem drinkers (Loeber, Croissant, Heinz, Mann, & Flor, 2006). A limitation of this approach is that the extinguished conditioned response may not generalize to other cues. An important consideration when doing in-session cue exposure is the risk of the patient using the substance once they leave their session.

Contingency management uses behavioral principles of operant conditioning to reinforce the occurrence of a targeted behavior. Substance use provides some

degree of positive reinforcement (e.g., the pleasurable effects of the substance) and negative reinforcement (e.g., the avoidance of withdrawal symptoms). In order for contingency management to be successful, the positive reinforcement for abstinence (as measured by negative urine toxicology or breathalyzer results) must be stronger than the reinforcement of use. In order to increase effectiveness, patients typically visit the treatment facility several times per week in order to quickly and accurately detect substance use (Higgins et al., 1993).

One commonly used contingency management strategy is voucher-based reinforcement therapy (Higgins et al., 1994; 1993). In this system, the patients earn "vouchers" of increasing monetary value for each negative drug test. Many programs use a reinforcement schedule that promotes abstinence by incrementally increasing the incentive rate with each subsequent indication of abstinence (Prendergast, Podus, Finney, Greenwell, & Roll, 2006). When the patient fails a drug test or fails to show up for a scheduled appointment, the value of the incentive returns to the starting point to incentivize continued abstinence.

One common limitation of contingency management programs is the availability of funds to provide increasing reinforcers for sustained abstinence. To circumvent this, a different contingency management technique was developed using a variable magnitude of reinforcement (Petry & Martin, 2002: Petry, Martin, Cooney, & Kranzler, 2000). With this approach, patients are given the opportunity to draw a prize out of a bowl that is of random value (e.g., ranging from $1 to $100). Both voucher- and prize-based contingency management interventions have been demonstrated to increase the rate of abstinence compared to standard treatment (Petry, Alessi, Marx, Austin, & Tardif, 2005).

In addition to voucher- or prize-based programs, methadone is often used as a component of treatment for opioid and cocaine abuse based on principles of contingency management. In these programs, patients are given methadone as a positive reinforcement for abstinence. Over time, they are able to earn increased doses, less frequent clinic visits, and even "take home" privileges (Griffith, Rowan-Szal, Roark, & Simpson, 2000; Rawson et al., 2002).

Bandura's work on modeling (1977) is a primary tenant for CBT which takes advantage of observational learning to introduce more effective coping strategies (Longabaugh & Morgenstern, 1999). These strategies can be modeled and role-played with the therapist in individual therapy or other patients in group-based CBT. This process creates exposure to, practice of, and mastery of skills through the role plays in addition to homework to be completed outside of session. A typical CBT session for AUD/SUD is divided roughly into thirds. The initial portion of the session is focused on reviewing any substance use and the overall functioning during the past week. The middle section typically emphasizes didactics and skills training, and the final portion is used to plan for the week ahead including the integration of new skills (Carroll, 1999). The range of interventions is quite large and includes motivational techniques, emotion regulation, goal setting, stimulus control, and cognitive interventions targeting beliefs about substance use and consequences. The aid of an effective functional analysis guides behavioral experiments or a trial and error processes to match coping skills to substance use

triggers or craving (Carroll & Onken, 2005). Situations identified as high risk are given emphasis as part of relapse prevention (Marlatt & Donovan, 2005).

24.5 Combined Cognitive Behavior Therapy for Eating and Alcohol/Substance Use Disorders

Limited information is available about the concurrent treatment of ED and SUD, particularly as related to the use of CBT for addressing symptoms of both conditions. Whereas patients with BN and another comorbid diagnosis (e.g., anxiety, depressive, or personality disorder) are routinely eligible to participate in controlled psychotherapy treatment trials of CBT, with few exceptions (Fairburn et al., 1991; Freeman, Barry, Dunkeld-Turnbull, & Henderson, 1988), individuals with BN and current SUD are excluded (Sysko & Hildebrandt, 2009). O'Malley and colleagues (2007) treated alcohol-dependent women with ED features (a score of 70 or more on the Bulimia Test-Revised; BULIT-R, Thelen, Farmer, Wonderlich, & Smith, 1991) with cognitive behavioral coping skills therapy and naltrexone or placebo. Although the treatments focused on alcohol dependence, significant decreases in ED psychopathology and objective bulimic episodes were observed over the 12 weeks of treatment, demonstrating that successfully treating one disorder (alcohol dependence) improved, but did not completely eliminate, the ED.

In the absence of empirical data, it is not clear whether CBT would be the treatment of choice for the treatment of individuals with BN and a current SUD. CBT as developed for ED, without modifications for substance use, could be difficult to complete due to problems with engagement (Wilson, Fairburn, & Agras, 1997), and the outcomes for BN could be negatively impacted by the presence of a co-occurring SUD (Bulik, Sullivan, Joyce, Carter, & McIntosh, 1998). Adaptations to existing cognitive-behavioral treatments for ED therefore appear necessary to address issues with substance abuse (National Institute of Clinical Excellence, 2004), and an integrated cognitive-behavioral treatment for ED and SUD should be considered (Sysko & Hildebrandt, 2009). As aforementioned, CBT is efficacious for the treatment of BN (e.g., Agras, Walsh, Fairburn, Wilson, & Kraemer, 2000), BED (Brownley, Berkman, Sedway, Lohr, & Bulik, 2007), some patients with AN (Fairburn et al., 2013), and SUD (Carroll & Onken, 2005; Finney, Noyes, Coutts, & Moos, 1998). However, cognitive-behavioral treatments for ED and SUD developed independently, without any attempts to address comorbidity (Sinha & O'Malley, 2000). It is unclear whether the effectiveness of CBT would be maintained when integrating treatment across disorders. However, an integrated treatment might be preferable to sequenced treatment, or addressing the SUD followed by the ED, because patients with both disorders report being refused treatment for either the ED or SUD because of the presence of the other problem (Dunn et al., 2007), and sequenced treatment requires substantial time and resources (Sysko & Hildebrandt, 2009).

Some support for integrated treatment models for SUD and comorbid conditions does exist. For instance, Mills and colleagues (2012) developed an integrated

treatment for SUD and posttraumatic stress disorder (PTSD) that increased reductions in PTSD symptoms relative to a treatment as usual condition suggesting that patients did not need to be completely abstinent from substance use to benefit from the integrated PTSD treatment. Both SUD treatment as usual and the integrated treatment resulted in similar reductions in substance use. Foa and colleagues (2013) also investigated combined treatment of PTSD and AUD with a four-arm study comparing prolonged exposure plus naltrexone to prolonged exposure plus pill placebo, naltrexone plus supportive counseling, and pill placebo plus supportive counseling. Results did not support a main effect for prolonged exposure on PTSD symptoms, but did indicate that combined treatment including prolonged exposure did not worsen AUD. Naltrexone was significantly more effective at reducing days drinking compared to placebo.

It is also worth noting that many patients who are comorbid for both are likely to be comorbid for other conditions. Comorbidity with other forms of mental illness is common for both SUD (Cuffel, 1996; Petrakis, Gonzalez, Rosenheck, & Krystal, 2002) and ED (Wilson, Becker, & Heffernan, 2003). For instance, comorbidity with PTSD and other anxiety disorders is common both for ED (Becker, Zayfert, & Pratt, 2009; Brewerton, 2007; Hudson, Hiripi, Pope, & Kessler, 2007) and SUD (Dore, Mills, Murray, Teeson, & Farrugia, 2012; Grant et al., 2004) (see Chaps. 11, 12, and 17). As such, clinicians who embark on integrated treatment for SUD and ED need to be prepared to at least manage and possibly treat other comorbid conditions if they do not sufficiently remit or reduce as a result of integrated ED/SUD treatment.

24.6 An Integrated Treatment for Bulimia Nervosa, Binge Eating, and Substance Use Disorders

The integration of two empirically supported CBT interventions (e.g., McCrady & Epstein, 2008; Fairburn et al., 2008) represents a natural progression to address the full range of comorbidity experienced by patients with ED. Blending two existing treatments can be complicated (see Sysko & Hildebrandt, 2009), but the available theoretical data supports the use of a combined treatment.

One strategy for conceptualizing blended treatments, addressing comorbid conditions, and resolving obstacles as they develop in treatment is to utilize the evidence-based case formulation approach described by Persons and her colleagues (Persons, 2005; Persons & Tompkins, 2007). The case formulation approach facilitates organization of an array of clinical information in a theoretically coherent manner, which then guides systematic selection and ordering of evidence-based interventions and techniques. When using this approach, a clinician would start by identifying predisposing, precipitating, and maintaining factors. Next, the clinician generates hypotheses about the interrelationships between the multiple problems presented by the patient. To the greatest degree possible, the clinician relies on evidence-based models to generate hypotheses so as to reduce clinical judgment errors that can occur when relying solely on more idiosyncratic models (Wilson, 1996). Reliance on evidence-based models is critical because such models offer the

clinician research-based guidance on common patterns associated with disorders established across the relevant populations. Next, the clinician uses the hypotheses to guide selection of treatment strategies, drawing as much as possible from treatments with established empirical support. Lastly, the clinician engages in ongoing assessment of response to treatment. This allows the clinician to determine if the patient is responding as predicted by the hypotheses and to correct the course of treatment if results suggest that the hypotheses need to be revisited. In summary, the evidence-based case formulation approach facilitates the use of treatment research and evidence-based models of psychopathology while also allowing for flexible individualized intervention that has a self-correcting process when patients do not respond as expected.

In the case of integrated treatment for ED and SUD, the clinician would want to include shared strategies in CBT for ED and SUD and common maintaining mechanisms (Harvey, Watkins, Mansell, & Shafran, 2004). These shared strategies include self-monitoring and recording relevant behaviors, thoughts, feelings, and events in the moment. This self-monitoring facilitates the identification of high-risk situations such as negative affect, stressful situations, boredom, and associations to binge eating or substance use. Once a high-risk situation has been identified, skills can be implemented to reduce the likelihood that there will be a subsequent loss of control (Sinha & O'Malley, 2000). Prior to engaging in an integrated treatment, therapists primarily familiar with CBT for ED should become familiar with unique aspects of SUD treatment, including in-session monitoring of use behaviors (e.g., breathalyzer, urine drug screen) and procedures for ensuring safety if patients arrive intoxicated/high. Further, the provider should evaluate whether the patient is sufficiently stable, on the basis of eating disordered and substance use symptoms, to engage in an outpatient treatment, and that detoxification or acute medical stabilization is not immediately necessary. Use of the case formulation approach described above would assist with this process.

As outlined by Sysko and Hildebrandt (2009), an integrated cognitive behavioral treatment should initially focus on gaining control over alcohol or drug use behaviors, such that the patient reduces their risk of overdose and can better engage in therapy without interference from alcohol/drug use or recovery from use. Early sessions should emphasize motivation (e.g., motivational interviewing, CBT techniques) and personalized feedback regarding substance use to increase readiness for change and engagement with CBT. After alcohol and drug use has decreased, subsequent sessions would evaluate changes in drinking/drug use and help the patient maintain changes in alcohol or substance use and making additional improvements and address ambivalence about change as needed (Wilson & Schlam, 2004).

Following this focus on substance behaviors, the therapist could begin instituting components of cognitive-behavior therapy for ED, beginning with self-monitoring of eating behavior and establishing a pattern of regular eating. Self-monitoring records should include a method for tracking drinking and/or drug use and bulimic behaviors, such that the therapist and patient can evaluate whether there are overlapping antecedents for substance use and bulimic behaviors. Once both

substance use and eating pathology occur less frequently, other factors maintaining the substance or eating problems can be addressed. A clinical assessment of common maintaining mechanisms for substance use and eating should occur, similar to that described by Fairburn (2008) to be used in the development of an extended treatment formulation that includes no more than two additional domains to target in treatment. In this assessment, consider whether the features of the AUD/SUD are (1) directly attributable to the ED or its consequences, (2) if they will interfere with successful treatment of the ED, and (3) their likelihood of dissipating if the ED is successfully treated. For individuals with co-occurring ED and SUD, we have suggested modules addressing motivation, interpersonal relationships, reward sensitivity, and impulsive behaviors as potential targets of this extended treatment formulation (Sysko & Hildebrandt, 2009); however, other characteristics (e.g., low self-esteem, depressed mood) commonly noted for ED may also serve to maintain substance use and binge eating and purging behaviors (Fairburn, 2008). Practically, sessions should be divided, with half of the session addressing ongoing substance use or bulimic behaviors (e.g., review of self-monitoring, problem solving) and half for a modular treatment (e.g., Fairburn et al., 2003) with a focus on common mechanisms (e.g., mindfulness activities, assertiveness training). The final stage helps the patient to maintain and build upon change and constructs a maintenance and relapse prevention plan for both the eating disordered symptoms and substance use.

24.7 Special Considerations for Integrated Cognitive Behavior Therapy with Eating and Substance Use Disorders

Many commonalities are present in the clinical features and treatment of individuals with ED and SUD; however, one important characteristic is distinct. The behavioral disturbance observed among AUD and SUD is consuming alcohol or drugs, while individuals with ED typically attempt to avoid specific foods and rigidly control caloric intake. Patients with comorbid ED and SUD therefore must simultaneously increase control over substance use and decrease rigid control over eating (Fairburn, 1995). This distinction should be explicitly discussed with patients at the beginning of treatment to highlight the contrast between the reasons for abstinence from alcohol and drugs (e.g., controlled drinking is difficult to maintain for individuals with problem use) and reducing dietary restraint (e.g., significant dietary restraint is likely to maintain binge eating behaviors).

Additional safeguards are required for mental health professionals providing treatment to patients with co-occurring ED and SUD diagnoses. As noted above comorbidity in this population is common. As such, clinicians need to be prepared to manage additional acute dangerous behaviors (e.g., non-suicidal self-injury and suicidality). Use of the case formulation approach described above can facilitate the development of a coherent treatment plan that includes necessary safeguards and safety procedures when indicated. Medical complications (e.g., hypokalemia from self-induced vomiting, Birmingham & Beumont, 2004) can also occur in comorbid

patients, and ongoing monitoring through collaboration with a primary care physician should be a component of any ongoing treatment intervention. Combined treatment for AUD/SUD and ED should be deliverable in any context in which outpatient treatment for either diagnosis is indicated. Established guidelines for when to refer to a higher level of care can be used to guide clinical decision points (Fairburn, 2008; Minkoff, 2001). In our experience, most referrals from a substance abuse program have ongoing support such as a 12-step program in place, and they may be prescribed medication to help manage their addiction (Ries, Galanter, Tonigan, & Ziegler, 2011). Referrals from an ED source may also be prescribed psychotropic medications, and they may participate in ED-specific groups.

24.8 Summary

Limited data are available to evaluate the treatment of patients with ED and SUD; however, an integrated form of CBT appears to be a viable option if adaptations are made to standard therapies used with the disorders separately. Use of the evidence-based case formulation approach can assist clinicians in tailoring treatment to individual patients with ED and SUD comorbidity, and an expanded version of CBT for ED (Fairburn, 2008) offers an additional example of a means by which to address shared characteristics through modules that allow for flexibility and individualization of treatment. Future studies should evaluate common features to target in treatment and the efficacy of a treatment adapted to address salient features observed among individuals with this particular dual diagnosis.

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Mindfulness Approaches in the Treatment of Eating Disorders, Substance Use Disorders, and Addictions

25

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Abstract

Clinicians and researchers alike struggle to find ways to ameliorate or support current effective treatments for patients suffering from eating disorders (ED), substance use disorders (SUD), or the comorbid occurrence of both. Although mindfulness has been around for hundreds of years, its application to the field of ED and SUD is relatively new. The current chapter describes several ED and SUD interventions that use mindfulness as a core therapeutic strategy, treatments that add mindfulness to established Cognitive Behavioral Therapy treatments, as well as interventions that include a mindfulness component (e.g., Acceptance and Commitment Therapy, Dialectical Behavior Therapy) within an existing paradigm. Finally, the authors will consider the mechanisms by which mindfulness may aid in the treatment of these disorders.

Keywords

Eating disorders • Mindfulness • Substance use disorders • Treatment

Although there are several empirically supported treatments for eating disorders (ED) and substance use disorders (SUD), researchers and clinicians constantly seek to enhance the treatments they provide. Both professionals and patients are

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interested in learning about mindfulness, "the awareness that emerges by way of paying attention on purpose, in the present moment, and non-judgmentally to the unfolding of experience moment by moment" (Kabat-Zinn, 2003), and its potential to improve outcomes.

Mindfulness in the form of eastern philosophies and practices has existed for hundreds of years. Jon Kabat-Zinn popularized mindfulness in the West while proposing its usefulness in helping people to better manage stress. Mindfulness is believed to promote general well-being; a focused practice can result in awareness, insight, wisdom, compassion, and equanimity (Kabat-Zinn, 2003). While more traditionally western treatments such as Cognitive Behavioral Therapy (CBT) focus on identifying and changing problematic or "dysfunctional" thoughts, mindfulness-based approaches emphasize the totality and acceptance of one's private experiences (thoughts, feelings, and sensations). In this way, mindfulness-based approaches have the potential to more comprehensively address the problems of patients suffering from ED and/or SUD.

This chapter describes several ED and SUD interventions that use mindfulness as a core therapeutic strategy such as Mindfulness-Based Relapse Prevention (MBRP), as well as two interventions that add a mindfulness component: Acceptance and Commitment Therapy (ACT) and Dialectical Behavior Therapy (DBT). After a review of how each intervention has been applied to patients with ED or SUD independently, we will consider the mechanisms by which mindfulness may aid in the treatment of these disorders.

25.1 ED and SUD Treatments in Which Mindfulness Is a Central Component

Mindfulness-Based Stress Reduction (MBSR) teaches mindfulness through formal meditation practice. The goal is to increase awareness and the ability to respond skillfully to experiences that contribute to emotional distress or problematic behavior. MBSR training has been used successfully in several populations (Grossman, Niemann, Schmidt, & Walach, 2004). However, research on the use of MBSR to address eating behavior is in its infancy, and its impact on eating behavior is not yet clear. For example, it has been demonstrated that participation in a MBSR program without a specific focus on eating was *not* associated with reductions in emotional eating or uncontrolled eating in a group of veterans with chronic health conditions (Kearney, McDermott, Malte, Martinez, & Simpson, 2012). Another study that combined MBSR with brief eating exercises found a significant reduction in binge eating (BE), anxiety, and depression (Smith, Shelley, Leahigh, & Vanleit, 2006) in a non-ED population.

MBSR has been applied to SUD as well. Specifically, Mindfulness-Based Therapeutic Community (MBTC) was adapted from MBSR to specifically address addiction-related changes in behaviors, attitudes, and emotions. MBTC was evaluated in the context of a substance abuse therapeutic community, which is a long-term, structured residential program designed to better prepare individuals to

reenter the community-at-large. The MBTC was manualized and implemented during the early phase of an 18-month treatment. Participants practiced meditation and discussed the usefulness of meditation in recovery life. Participants in the MBTC, but not the usual treatment group, demonstrated significant reductions in physiologic measures of stress, as well as lower scores on emotional irritability and muscle tension subscales of the stress assessment measurement. Also of interest is the fact that an increase in the MBTC intervention participation was associated with retention in treatment (Marcus et al., 2009).

Mindfulness-Based Cognitive Therapy (MBCT) (Segal, Williams, & Teasdale, 2013), which is based on MBSR and believed to combine cognitive therapy and meditative practices and attitudes, was developed to treat individuals suffering from chronic depression. In a small pilot study, MBCT was shown to decrease binge eating (Baer, Fischer, & Huss, 2005). In a study of women with disordered eating, an MBCT intervention that included a focus on eating demonstrated improvements in body image as well as emotional and external eating (Alberts, Thewissen, & Raes, 2012).

MBCT has also been adapted to address SUD. Mindfulness-Oriented Recovery Enhancement (MORE) was designed to address stress-precipitated alcohol relapses via ten sessions of mindfulness instruction and practice. In one study, MORE was compared to an evidence-based alcohol-dependent support group (ASG) for patients residing in a therapeutic community. The results suggest that patients in the MORE group had less thought suppression, decreases in self-reported perceived stress, and increased heart rate variability recovery following alcohol cue exposure than those receiving the ASG. In addition, decreases in thought suppression were associated with improved ability to manage drinking urges (Garland, Gaylord, Boettiger, & Howard, 2010).

The most widely used adaptation of MBCT designed specifically to address SUD is MBRP. In an eight-session manualized intervention, MBRP integrates coping skills from CBT with mindfulness practices, raising awareness of substance use triggers and reactive behavioral patterns and teaching skillful coping responses (Bowen, Chawla, & Marlatt, 2011). MBRP trains individuals to experience and monitor cravings and urges in a nonevaluative, nonjudgmental manner. For example, as opposed to managing cravings through distraction and/or suppression, individuals practicing mindfulness increase conscious awareness of cravings and associated body sensations. For example, the skill of "urge surfing" is a method of experiencing a craving in a more curious, accepting manner. Patients imagine they are riding an ocean wave experiencing the rise, peak, and decline of the wave/craving. Patients learn that such experiences are time limited and they do indeed have control over their responses.

In a pilot study ($N = 168$), research suggests that MBRP is promising when delivered as aftercare following inpatient or intensive outpatient treatment of SUD when compared to treatment as usual (TAU) (Bowen et al., 2009). Participants attended more hours in the MBRP than in the TAU intervention, and 86 % of MBRP participants reported practicing meditation during follow-up. Acceptance and acting with awareness were significantly higher, and craving was significantly

lower in the MBRP participants. At 2-month follow-up, participants in the MBRP had significantly fewer days of alcohol and drug use than participants in the TAU group. One of the important findings of this study is the impact of MBRP in altering reactivity to negative emotional states. Craving mediated the relationship between depressive symptoms and subsequent alcohol/drug use at 4 months post-intervention in the TAU, but not in the MBRP group. This was not an unexpected finding, since the goal of MBRP is to allow individuals to experience negative emotions and challenging situations without reacting automatically or impulsively (Witkiewitz & Bowen, 2010).

Mindfulness and Modification Therapy (MMT) is an adaptation of MBRP designed to address aggressive behavior in SUD. In a small, uncontrolled pilot study, 14 court-mandated women with alcohol use disorders and aggression participated in 12 weekly sessions of MMT to evaluate feasibility, acceptability, and impact on behavioral dysregulation. Researchers delivered the MMT in individual sessions along with coping skills focusing on behavioral regulation. Results demonstrated a significant reduction in drinking during treatment, as well as a decrease in drug use and physical aggression by the end of treatment (Wupperman et al., 2012).

Some mindfulness-centered treatments have been developed specifically for ED patients. Mindfulness-Based Eating Awareness Training (MB-EAT; Kristeller, Baer, & Quillian-Wolever, 2006; Kristeller & Hallett, 1999; Kristeller, Wolever, & Sheets, 2013; Wolever & Best, 2009) treats binge eating disorder (BED) and obesity. The MB-EAT model is based on the assumption that the hunger and satiety systems of individuals with BED are significantly influenced by nonnutritive stimuli, resulting in a disconnection with internal signals. The program incorporates "traditional meditation techniques as well as guided meditation practices, to address eating-related self-regulatory processes including emotional vs. physical hunger triggers, gastric and sensory-specific satiety (SSS), food choice, and emotional regulation pertinent to self-concept and stress management" (Kristeller et al., 2013, p. 2). Preliminary research on the MB-EAT is promising in that it significantly decreases BE, food preoccupation, and binge concerns. In addition, increased control of eating and changes in BE are positively correlated to the amount of time spent in meditative practice (Kristeller et al., 2013).

Although there are few interventions designed for the treatment of comorbid SUD and ED, we are aware of one study that examined the impact of mindfulness-based substance abuse interventions on eating behavior. Mindfulness Awareness in Body-Oriented Therapy (MABT) uses hands-on massage and mindfulness meditation to bring awareness to body sensations associated with inner experiences. Individuals notice and attend to the effects of emotions and stress on certain body areas. There are 8 weekly individual sessions with take-home practices. In a small pilot study, 46 women attending an intensive outpatient substance abuse treatment program were randomized to receive either MABT plus TAU or TAU alone. Thirty percent of the women had an eating disorder. Women in the MABT group had greater abstinence from alcohol and substance use following treatment and at follow-up assessments. There were large effect sizes showing improvements in

eating disorder symptoms, depression, and dissociation. Interventions using mindfulness meditation approaches that target emotional regulation may benefit both SUD and ED where impulsivity and dysregulation are underlying etiologic factors (Price, Wells, Donovan, & Rue, 2012).

Finally, we have found one study that provides evidence of a mindfulness-based treatment designed specifically to meet the needs of the comorbid BED/SA patient (Courbasson, Nishikawa, & Shapira, 2010). Mindfulness Action Cognitive Behavioral Therapy (MACBT), a 16-week group treatment, teaches mindfulness, as well as mindful eating, and promotes the regulation of emotions without food or substances. Preliminary results showed decreases both in BE and in severity of alcohol and drug addiction.

25.2 Mindfulness Added to Existing ED or SUD Treatments

Mindfulness meditation approaches have been successfully coupled with other psychosocial approaches such as CBT and motivational interviewing (de Dios et al., 2012; Hepworth, 2011; Leahey, Crowther, & Irwin, 2008; Woolhouse, Knowles, & Crafti, 2012) to address the anxiety, preoccupation, and emotional reactivity that often drive substance abuse and eating disorder behavior. Enhanced control over such inner experiences can assist these patients with implementing adaptive coping skills and pursuing meaningful life goals.

25.3 Treatments That Include a Mindfulness Component

DBT and ACT rely on a wider variety of mindfulness exercises. These are typically shorter in duration and do not necessarily require a formal meditation practice.

25.3.1 Dialectical Behavior Therapy

DBT is a comprehensive treatment intervention originally developed by Marsha Linehan to treat patients with chronic suicidality who also suffered from Borderline Personality Disorder (BPD) (Linehan, 1993a). Since the publication of Linehan's treatment manual in 1993, the literature has witnessed a rapid growth and application of DBT to a wide variety of mental disorders. Randomized trials have shown that DBT is associated with fewer hospital admissions, lower rates of substance abuse and angry outbursts, increased patient commitment to therapy, fewer dropouts, and decreased therapist burnout (see reviews by Lynch, Trost, Salsman, & Linehan, 2007; Robins & Chapman, 2004). In its standard format, DBT involves a 1-year commitment to weekly individual therapy, group skills training, telephone skills coaching, and a therapist consultation team (Linehan, 1993a, b). DBT utilizes a combination of behavioral principles, cognitive modification techniques, eastern meditative practices, and acceptance-based strategies, with the aim of helping

patients eliminate impulsive and self-destructive behaviors while building a "life worth living" (Harned, Banawan, & Lynch, 2006).

Mindfulness, one of the core DBT skills modules, was designed to teach people how to focus their attention on the present moment without judgment. Mindfulness skills teach patients how to step away from distracting and painful thoughts that may trigger symptoms. The category of mindfulness skills is further broken down in the "what" and "how" skills. The "what" skills teach individuals to observe, describe, and participate with the goal of living a lifestyle that includes participation with awareness. The "how" skills refer to the way in which DBT recommends that one observes, describes, or participates: nonjudgmentally, doing one thing in the moment and doing what works (participating effectively).

In the last decade numerous book chapters and articles have been published on the use of DBT with ED patients (see review by Bankoff, Karpel, Forbes, & Pantalone, 2012). The majority of this literature describes the evaluation of an adaptation (specifically 20 weeks of a modified DBT skills training) for individuals with BN (Safer, Telch, & Agras, 2001) and BED (Safer, Robinson, & Jo, 2010; Telch, Agras, & Linehan, 2001) who present with low to moderate levels of ED symptoms. Thus, therapists treating patients with primary BN and BED (e.g., those with low to moderate illness severity and little or no diagnostic comorbidity) are directed to these resources, including a superbly written treatment manual (Safer, Telch, & Chen, 2009).

The treatment of patients with more significant ED symptoms and multiple treatment targets such as an ED plus SUD, however, is more complicated. The literature to date recommends more comprehensive treatment models for patients who suffer from multiple problem behaviors (Ben-Porath, Wisniewski, & Warren, 2009; Chen, Matthews, Allen, Kuo, & Linehan, 2008; Kröger et al., 2010).

Because patients who suffer from multiple treatment problems are often considered complex and/or difficult, they pose unique problems for their care providers. One treatment conundrum for patients who suffer from multiple problems such as an ED as well as a SUD is the sequencing of treatment. However, there is limited research on guidelines as to what disorder to address first. DBT provides a framework with which to approach multiple treatment targets.

In DBT there is an emphasis on developing a hierarchy for addressing target behaviors; this provides a clear path to prioritizing the goals of treatment (Linehan, 1993a). Target I behaviors are those that are imminently life threatening. In standard DBT, suicidal and self-harming behaviors are considered Target I. For eating disorder patients who are not experiencing suicidality, ED-specific behaviors fall into Target I if engaging in that behavior exacerbates existing medical instability (Wisniewski, Bhatnagar, & Warren, 2009; Wisniewski, Safer, & Chen, 2007). An example of this would be purging while diagnosed with an electrolyte imbalance.

Target II behaviors are defined as any behavior on the part of the patient or therapist that impedes the patient receiving effective treatment. The idea is that a therapy that is not received cannot succeed. In standard DBT, the patient's Target II behaviors are non-attentive, non-collaborative, or nonadherent (e.g., failure to

attend sessions, lying, or not filling out diary cards). A DBT therapist can also engage in behaviors that interfere with treatment, such as pushing for change without a focus on acceptance or engaging in behaviors that indicate a lack of respect for the patient (e.g., taking a phone call during a session). Outside of these more general Therapy Interfering Behaviors (TIBs), patients with EDs may exhibit TIBs specific to the disease (Wisniewski et al., 2007, 2009). These include, but are not limited to, vomiting that reduces the effectiveness of medication, failure to eat prior to a therapy session resulting in unclear thinking, water loading prior to being weighed, hiding food during therapeutic meals, and not fully disclosing the extent of problematic behaviors.

Target III behaviors are those that are so problematic that a failure to change them would likely result in the patients inability to have a reasonable quality of life. Behaviors typical to EDs that do not fall into Target I or II are addressed as Target III. These can include restricting, binging, purging, pill use, and compulsive exercise when these behaviors are not occurring in the context of medical instability or are interfering with treatment.

The target hierarchy is a useful frame for therapists addressing complex ED patients. Take, for example, the BN patient who canceled her last two appointments (Target II) and arrives late for the session (Target II) without her diary cards (Target II), has lost 4 lbs secondary to restriction (Target III), reports that she cut her arm (Target I) the previous evening after drinking alcohol (Target III) following an argument with her mother, and has been vomiting three times daily (Target III) over the past 2 weeks. In this situation, even a seasoned therapist would have difficulty knowing where to start! The DBT clinician's job would be to assess suicidality, address the self-harm directly via a behavior chain analysis, acknowledge and problem-solve the TIBs of canceled and late appointments as well as missing diary cards, and *then* make a plan for managing ED and SUD behaviors.

Only a few studies have explored DBT in patients suffering from BPD as well as SUD. One study randomized opiate-dependent women to either 12 months of DBT or a control intervention called Comprehensive Validation Therapy (CVT). There was a reduction in opiate use in both groups; between-group differences favored women in the DBT group during the last 4 weeks of treatment (Linehan et al., 2002). At 4 months posttreatment, both groups had a low percentage of opiate-positive urine drug screens with no between-group differences. The small sample size and use of a specific population of comorbid BPD and SUD patients limited generalizability of findings to the broader substance abuse field. Also, the length of treatment may make this intervention less feasible to implement in substance abuse programs.

There is at least one study demonstrating that DBT may be a practical alternative for the treatment of co-occurring ED and SUD (Courbasson, Nishikawa, & Dixon, 2012). DBT's unique synthesis of behavioral principles, dialectical philosophy, and Zen influences makes it particularly useful to those patients who struggle with issues of motivation and commitment. Patients suffering from an ED and SUD differ from many other mental illnesses in the considerable degree of ambivalence they feel concerning their symptoms and recovery. Treatment of ED and SUD

symptoms, therefore, requires a sophisticated use of commitment strategies that focus not only on helping patients change problematic behaviors but also on coaching patients to accept their present state and condition. This emphasis on both change-based and acceptance-based therapeutic strategies is a key aspect of DBT. In addition, DBT's practical and theoretical framework for managing multiple treatment targets (i.e., target hierarchy) may aid the clinician in effectively addressing all problematic behaviors.

Other crucial elements of DBT, such as mindfulness and acceptance, are similarly suited for those professionals treating patients suffering from a comorbid ED and SUD, whose behavior may evoke intense emotions in them. For example, ED as well as SUD behaviors can be perceived as conniving, dishonest, and superficial by therapists as well as family or friends. Such negative attributions can interfere with treatment. The consultation team, one of DBT's primary components, implements DBT approaches to provide guidance on therapist self-awareness and ensure fidelity to the treatment model. The therapist support delivered via the consultation team is fundamental for the successful treatment of EDs, especially those that are chronic and/or suffering from SUD.

25.3.2 ACT: The Essentials of ACT

ACT is a treatment that belongs to a discipline called Contextual Behavioral Science (CBS; see Hayes, Strosahl, & Wilson, 2012) and incorporates mindfulness as a core strategy. While, like DBT, ACT is a form of CBT, it differs fundamentally in its assumptions. Rather than examining a thought in a general sense as "functional" or "dysfunctional" as in classical CBT, the unit of analysis in CBS is the ongoing "act-in-context." ACT does not limit its analysis to the form of a behavior, but seeks to understand how context determines function. For example, if an individual was rolling peas on her plate, the function or meaning of that behavior would be determined by her particular history or current situation. She could be playing a game that she had learned with peas, she could be manipulating the solar system according to some delusional belief, or she could be forestalling the completion of her meal due to her anorexia nervosa (among other possible functions). In each case, it is the historical or situational context that determines the function of the behavior.

ACT asserts that much of human behavior is under verbal contextual control rather than direct experiential control, a double-edged sword that is a major source of human suffering as well as a potential advantage over other nonverbal creatures on the planet. According to Hayes (2004), one of the chief developers of the ACT approach, ACT assumes that dramatic and rapid change is possible in mental health sufferers because the general context and purpose of their actions is the true problem, not the mental content of their learning histories. In ACT, the problem to be solved is not the inability to control or eliminate anxiety about eating/weight/shape or urges to binge and purge, or the use of substances, but rather the tendency to take these mental events literally and to struggle against them as if one can

readily eradicate the contents of the mind. The therapeutic focus, therefore, is always on the function of mental events, not their form or frequency outside of a historical or situational context. Problems arise when mental events such as thoughts, feelings, urges, body sensations, and memories become obstacles to valued living. ACT does not debate whether or not these events are "true" in terms of social convention but how they function in terms of a person's chosen values. For example, what value does the thought "I'm fat" serve? If the thought does not serve a value, it has little utility. "What is true is what works" (Hayes et al., 2012).

ACT posits that excessive verbal control of behavior can result in the rule-boundness characteristic of ED sufferers. Rule-bound behavior can be quite rigid or inflexible. Rules may specify behavior and expected outcome to the extent that natural contingencies of behavior are obscured. For example, the rule that says "If I don't eat, I won't get fat" or "If I don't take a drink, I can't talk to her" specifies a behavior and an expected outcome but may hide the real physiological consequences of the behavior from the individual. ACT employs nonlinear uses of language since language processes themselves are a source of rigidity. There is less use of logical analysis and more reliance on metaphor, stories, pictures or graphics, exercises, and experiential and behavioral tasks.

ACT does not replace dysfunctional thoughts with functional ones; it changes the relation of the thinker to the thought. For example, if the thought "I'm fat" functions to motivate restrictive eating which results in negative physical effects, the individual must examine whether this thought serves a values context or some other context such as experiential avoidance. It is not necessary to change the thought itself, just its harmful functions.

According to ACT, there are six fundamental processes that result in psychological inflexibility (1) experiential avoidance or intolerance of private mental events, (2) cognitive fusion or taking mental events literally, (3) inflexible attention or rule-boundness, (4) attachment to a conceptualized self that constricts personal options, (5) dominance of values that have lost their positive reinforcing qualities, and, lastly, (6) inaction or impulsivity driven often by immediate reward or relief (Hayes et al., 2012). The ED literature is replete with evidence supporting the importance of the ACT processes of inflexibility in maintaining ED behavior (see Juarascio et al., 2013; Merwin & Wilson, 2009 for reviews). With SUD the role of experiential avoidance of both private mental experiences as well as drug responses such as cravings or withdrawal symptoms has been discussed and evaluated (Gratz, Bornovalova, Delany-Brumsey, Nick, & Lejuez, 2007).

ACT theorists describe acceptance and mindfulness processes such as flexible attention to the present moment, acceptance, defusion, or deliteralization of thinking, and self-as-context or an observing self. In the rule given above "If I don't eat, I won't get fat," the ACT approach could encourage the person to step back from the thought with mindful reflection, seeing it only as a thought, and opening up to their direct experience rather than listening to what their mind labels the thought to be. A thought such as "This feeling is unbearable without a drink" could be seen as an

evaluation and a prediction rather than a necessary representation of reality. This is also mindful awareness.

25.3.3 Preliminary Evidence for the Use of ACT in ED and SUD

A number of publications ranging from case reports to RCTs describe the use of ACT strategies in the treatment of ED and SUD. In a small case series of AN patients treated with manualized ACT, patients demonstrated clinically significant improvement on some measures of mood and eating at posttreatment and maintained weight at 1-year follow-up (Berman, Boutelle, & Crow, 2009). In an experiment comparing ACT with individuals with subthreshold eating pathology (Juarascio, Forman, & Herbert, 2010), ACT produced significantly larger decreases in ED pathology than did CBT at posttreatment.

ACT-based groups have been evaluated in a residential treatment center for ED (Juarascio et al., 2013). In this study, patients received the usual intensive residential treatment or the usual treatment plus ACT groups. The participants in the ACT group showed larger decreases in ED pathology and lower rehospitalization rates at 6-month follow-up. ACT also achieved larger, though not statistically significant, increases in willingness to experience distressing thoughts and feelings while engaging in valued behaviors. This willingness appeared to be the chief mediator of change in Eating Disorder Examination (EDE; Fairburn, 2008) global scores.

Wildes and Marcus (2011) described a case series of five patients with AN in which they employed a treatment called Emotion Acceptance Behavior Therapy (EABT). EABT used elements of ACT, DBT, and MBCT to increase emotion awareness, decrease emotion avoidance, and redirect behavior toward valued activities. Four of the five patients completed at least 90 % of the 24 program sessions and demonstrated improvements in depression, anxiety, emotion avoidance, and quality of life.

Likewise there have been some initial forays into the use of ACT in the treatment of SUD. In one study, adding ACT to methadone maintenance improved outcome (Hayes, 2004), and in another study participants receiving a smoking-focused version of ACT fared better than nicotine treatment at 1-year follow-up (Gifford et al., 2004). An abbreviated version of ACT was studied in three adults with marijuana dependence (Twohig, Shoenberger, & Hayes, 2007). All participants were abstinent from marijuana at posttreatment. At the 3-month follow-up, one was still abstinent, while the other two had reduced their use below baseline levels. Smout and colleagues (2010) completed an RCT that compared ACT to CBT in the treatment of methamphetamine users. Unfortunately, due to high attrition rates, the study results are difficult to interpret. However, results indicate there were no significant differences on outcome measures for the two groups as both treatment conditions demonstrated positive effects upon the severity of dependence over time. Finally, ACT has been evaluated against drug counseling (DC) in methadone detoxification (Stotts et al., 2012). Of the 56 opioid dependent patients, 37 % of the ACT participants versus 19 % of the DC condition were successfully detoxified at

posttreatment. ACT also reduced fear of detoxification across time in comparison to the DC condition. Although not as impressive as the evidence of ACT with ED, more study is certainly warranted.

25.3.4 ACT for Patients Suffering from Both ED and SUD

ACT offers direct strategies well suited for ED and SUD, including individuals who exhibit high harm avoidant temperaments, low self-directedness, difficulty in set shifting, poor central coherence, and rule-boundness or excessive supervisory cognitive control as frequently seen in individuals with ED (Fassino, Amianto, Gramaglia, Facchini, & Daga, 2004; Kaye, Fudge, & Paulus, 2009; Schmidt & Treasure, 2006). For SUD, there is also some evidence that low self-directiveness predicts negative outcome as well (Arnau, Mondon, & Santacreu, 2008). Self-directedness is particularly important because it is strongly associated with well-being in population samples (Cloninger & Zohar, 2011). For individuals with the traits enumerated above, ACT promotes mindful awareness of how these traits serve or hinder valued living.

Poor set shifting, rule-boundness, and excessive supervisory cognitive control all contribute to the characteristic cognitive rigidity particularly seen in ED sufferers; ACT, as a psychological flexibility strategy, addresses this with acceptance and mindfulness processes. These processes employ well-accepted principles of mindfulness including present moment awareness of experience coupled with a nonjudgmental receptivity and nonreactivity (Wilson & DuFrene, 2008). ED as well as SUD individuals often show strong fusion with their thoughts and have difficulty taking other perspectives. A mindful stance opens the person to their direct experiences rather than fusion with verbal statements about those experiences. The self-as-context and present moment processes of ACT, along with acceptance and defusion can help with increasing flexibility in service of self-directedness.

ACT often employs metaphor to derail the literal use of language. One of the central metaphors of ACT and of self-directedness is the "Passengers on the Bus" metaphor (Hayes et al., 2012), which states:

> "You are a bus driver with a bunch of unsavory characters on your bus. They are dressed in black leather jackets with gruesome logos carrying switchblades and chains. Naturally, you don't want to look at them so you strike a deal with them. If they stay out of sight, you will do what they tell you. They tell you to turn right, you turn right. They tell you to turn left, you turn left. The only problem is that, although they stay out of sight, you cannot get to where you want to go. Who are these passengers on the bus? They are thoughts, feelings, body sensations, and memories (your private mental events)." (pp. 157–158)

When one is directed by experiential avoidance, one cannot be self-directed (or, in other words, values directed). This is essentially the goal of ACT—self-directedness and the well-being that accompanies it.

25.4 Putting It All Together: Mindfulness and Treatment for ED/SUD

In this chapter, we have reviewed the research on mindfulness-based approaches for the treatment of ED and SUD and have presented data demonstrating that increased levels of mindfulness are related to decreased levels of dysfunctional behavior. We conclude that mindfulness-based interventions may be useful in the treatment of ED and SUD in that they hold the potential to promote awareness and acceptance of private mental experiences and thus may reduce the need to avoid or escape internal phenomena.

Mindfulness interventions also have the potential to help patients learn to tolerate rather than avoid unwanted experiences. This may be particularly helpful when behaviors are driven by cravings for food or substances. The ability to focus attention on unwanted or distressing experiences or urges without impulsive reactivity could permit the individual to utilize skillful goal-directed behavior.

New treatments are needed for patients suffering from both ED and SUD since traditional interventions are plagued by relapse. In more cognitive behavioral interventions, patients are encouraged to use distraction as well as cognitive and behavioral avoidance as a means of managing cravings, urges, and thoughts about engaging in problematic behaviors. Alternatively, mindfulness-based techniques, with their focus on awareness and acceptance, may provide a more complete set of skills and potentially a more sustained recovery. In support of this theory, it has been demonstrated that individuals who have a regular mindfulness practice display less rumination and improved emotional and behavioral regulation (Lykins & Baer, 2009).

Finally, mindfulness interventions may be helpful in the treatment of ED and SUD by directly influencing the brain. Regions involved in selective attention, learning and memory, emotion regulation, and self-referential processing are affected by mindfulness practice, which can even reverse some of the neuroadaptive changes brought on by addiction (Hölzel et al., 2011). Mindfulness has also been shown to be related to thickness of the insula (Lazar et al., 2005), the area of the brain believed to be associated with body attention and body awareness and implicated in the development of AN (Nunn, Frampton, Fuglset, Törzsök-Sonnevend, & Lask, 2011). Thus, mindfulness may not only be able to promote changes in the behavioral and experiential realms but also at the level of observable brain structure itself.

This chapter has raised important questions about the role of mindfulness and mindfulness-based interventions in the treatment of ED and SUD. Future research evaluating mindfulness-based interventions with individuals suffering from both ED and SUD is warranted.

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Family and Couples Therapy for Eating Disorders, Substance Use Disorders, and Addictions

26

Stuart B. Murray, Zandre Labuschagne, and Daniel Le Grange

"Only the family, society's smallest unit, can change and yet maintain enough continuity to rear children who will not be "strangers in a strange land", who will be rooted firmly enough to grow and adapt."

Salvador Minuchin, 1978.

Abstract

Family therapy, since its emergence in the 1960s, has continued to offer advances in our understanding and treatment of many psychiatric illnesses. Of particular interest to family therapists has been the family-based treatment of eating disorders and substance use disorders. We discuss the historical development of family therapy for eating disorders and also outline the practical application and empirical evidence base for the current gold standard treatment for adolescent anorexia nervosa; family-based treatment. Furthermore, we outline alternate approaches to the family-based treatment of anorexia nervosa, including multifamily therapy that involves the simultaneous treatment of several families together, and family-based approaches to the treatment of bulimia nervosa in adolescents. In addition, we outline the practical application and empirical evidence base for a multitude of approaches to the family-based treatment of substance use disorders in adolescents. It is evident that the involvement of family members in the treatment of adolescents with eating disorders and substance use disorders offers a highly efficacious alternative to the individual treatment approaches outlined elsewhere in this book, although it

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is also evident that further research is needed to locate the agent of change and further enhance the efficacy of family-based treatments.

Keywords

Addictions • Anorexia nervosa • Bulimia nervosa • Eating disorders • Family-based treatment • Family therapy • Family and couples therapy • Substance use disorders

26.1 The Development of Family Therapy for Anorexia Nervosa

Since the very first documented cases of eating disorders (ED), the role of the family has been of significant clinical interest. For instance, the earliest recorded case descriptions of anorexia nervosa (AN) were accompanied by clinical observations of the patient's families, with Lasegue noting that any clinical description of AN "would be incomplete without reference to the patient's home life" (Lasegue, 1883).

Indeed, family therapists have demonstrated a sustained interest in the treatment of ED, which dates back for more than 40 years and encompasses a variety of theoretical lenses. For instance, as early as the late 1970s, both structural (Minuchin, Rosman, & Baker, 1978) and systemic (Selvini Palazzoli, 1974) family therapy approaches reported promising findings in the treatment of AN, which fell in stark contrast to the efficacy of the treatment of adults with AN at the time. These early family therapy approaches centrally implicated the role of parents in attempting to interrupt what were thought to be dysfunctional family dynamics, and despite later research demonstrating that the theoretical foundations may have been flawed (Eisler, 2005), these works profoundly impacted the later development of family therapy for ED.

Subsequent approaches maintained the firm emphasis on parental involvement, although crucially conceptualized the family of those afflicted as a major resource in their child's recovery rather than being implicated in the etiology of their illness (Dare, 1985; White, 1987). This conceptual shift resulted in the development of a family-based treatment (FBT) for AN which respected the role of parents in the treatment of their child while remaining agnostic as to the origin of the illness and thus absolving family members from any notion of having caused AN (Dare, Eisler, Russell, & Szmukler, 1990).

However, despite promising theoretical advances in the family-based treatment of AN, a long-standing criticism centered around the lack of controlled research trials, which hindered the extent to which family therapy practices were endorsed beyond family therapy contexts. To this end, the first randomized control trial (RCT) of FBT for AN was published in 1987, illustrating that adolescents who had AN for less than 3 years demonstrated greater weight restoration when having undergone 1 year of family therapy, as opposed to 1 year of individual supportive

therapy (Russell, Szmukler, Dare, & Eisler, 1987). Although these trends also prevailed at 5-year follow-up (Eisler et al., 1997), it is important to note that FBT did not offer significant benefits to adolescents who had been unwell for longer than 3 years (despite similar clinical observations in terms of the structure and organization of the families). Additionally, FBT was not found as effective as individual therapy for adults with either AN or bulimia nervosa (BN).

Thus, this seminal RCT was of immense importance in identifying a subgroup of those with AN whose recovery was significantly augmented with family therapy. These results suggested that the response to family therapy may be most centrally determined by illness factors such as age of onset and illness chronicity, rather than family factors. Further, in a follow-up to this seminal paper, the same research group described the clinical implications of their findings, offering detailed descriptions of the family therapy interventions utilized in their earlier trial, which allowed replication of their methods (Dare et al., 1990). As a result, a second RCT compared a form of family-based treatment with ego-oriented individual therapy, noting that family therapy was superior to individual therapy in terms of weight restoration (as measured by average BMI) and physiological functioning (i.e., return of menses in post-menarcheal adolescents) both at end of treatment and at 1-year follow-up, and further resulted in positive increments in communication between family members (Robin et al., 1999).

Further research evaluating FBT supported its efficacy, demonstrating that both conjoint family therapy (in which the whole family was seen together) and separated family therapy (in which parents and children were seen separately by the same therapist) were efficacious in bringing about weight restoration, although illustrated that conjoint FBT better allowed wider familial issues to be addressed (Eisler et al., 2000; Le Grange, Eisler, Dare, & Russell, 1992).

26.2 A Theoretical Model of Family-Based Treatment for Adolescent Anorexia Nervosa

FBT is a sophisticated amalgamation of the principles and concepts of many previous schools of family therapy and was designed as an outpatient treatment which is aimed at empowering parents to directly intervene in their child's symptom profile and rehabilitate them back to nutritional and psychological well-being (Lock & Le Grange, 2013; Lock, Le Grange, Agras, & Dare, 2001). In accordance with the increasing evidence failing to support any causal role of families in the development of AN (Eisler, 2005), FBT adopts an agnostic stance to the etiology of AN and is inherently non-blaming of family members. This agnostic stance has helped focus on families as a major resource in their child's recovery from AN (Le Grange & Eisler, 2009) while acknowledging the significant impact that the presence of AN can have on the vicissitudes of family life, noting that the crisis presented by the presence of AN may mean that families come to focus more and more on this crisis, inadvertently organizing family life around AN (Eisler, 2005). Thus, given that everybody in the family is typically affected by the increasingly

central presence of AN in the family, FBT affords clearly defined roles for everybody in the family in helping the adolescent's recovery from AN.

The core theoretical tenets of FBT advocate firm parental control and implore direct parental intervention into all individual and ecological maintaining factors of AN until symptoms are abated (Lock & Le Grange, 2013). However, FBT posits that the spirit of this parental control ought to be undertaken from a compassionate standpoint, free of any expressed criticism for the adolescent, given that expressed criticism is consistently associated with treatment dropout and poor outcome (Le Grange, Eisler, Dare, & Hodes, 1992). To this end, externalization of "the anorexia" helps separate the adolescent from their illness, placing the illness external to family functioning and alleviating any parental guilt, which is often expressed as criticism for the adolescent and is thus associated with treatment dropout (Le Grange, Eisler, Dare, & Hodes, 1992).

Further theoretical tenets of FBT underscore the need for parental unity in coordinating and undertaking the collective parent-led nutritional rehabilitation and also promote sibling support of the adolescent with AN, thus aligning and strengthening familial subsystems in the face of the challenge presented by AN (Lock & Le Grange, 2013). In evaluating the centrality of each of the theoretical tenets of FBT, a recent study found that parental control, externalization of AN, parental unity, and lack of parental criticism were all directly related to treatment outcome, and while sibling support was not related to outcome directly, it was directly related to parental firmness (Ellison et al., 2012), suggesting that the core theoretical components of FBT are crucial in bringing about symptom reduction.

26.3 The Practical Application of Family-Based Treatment in Adolescent Anorexia Nervosa

FBT is separated into three distinct phases of treatment and is most commonly conducted over approximately 20 sessions across the course of a 1-year period. Each phase of treatment is characterized by distinct therapeutic aims and levels of intensity. Furthermore, the delivery of FBT is most commonly undertaken by a team comprising the FBT clinician, a psychiatrist, and in some cases a pediatrician. Dietitians are not typically involved throughout FBT, as treatment compels and empowers parents to make their own decisions in selecting foods that will help their child gain weight.

26.3.1 Phase 1 of FBT

The first phase of FBT is exclusively focused on ensuring consistent weight gain in the adolescent by concentrating on parent-led refeeding and weight restoration until all medical crises and behavioral features of AN are abated. The magnitude of the medical crises potentially facing those with AN necessitates swift and consistent weight gain, and this takes precedence over all other areas of adolescent and family

functioning. This phase is therefore characterized by an exclusive and single-minded focus on the behavioral symptoms of AN, and parents are continually encouraged to work together to collectively devise strategies to ensure that their child consumes an adequate volume and variety of calories, in addition to problem solving all AN-maintaining behaviors which may be inhibiting consistent weight gain in their child, such as food restriction and overexercise. The adolescent is weighed at the start of each session, and this information is shared with the family, which in many instances may be helpful in mobilizing parental anxiety and providing feedback for their parental efforts at refeeding, in addition to providing exposure to feared weight ranges for the adolescent.

This phase of treatment is also inclusive of highly specialized family meal sessions, in which the family members are invited to bring a picnic to eat in the therapist's office. This session is particularly important, as it allows the therapist an in vivo observation of the family's interactions around food, in addition to allowing for direct coaching through reenactment of interactions when food becomes challenging for the adolescent.

26.3.2 Phase 2 of FBT

Phase 2 of FBT is marked by the gradual transition of age-appropriate guardianship of the adolescent's recovery from the parents to the adolescent herself or himself, with the aim of fostering independent management of healthy eating practices in the adolescent (Lock & Le Grange, 2013). The initiation of this phase is normally indicated upon the adolescent reaching a healthy weight (90 % of ideal body weight, as calculated according to height and weight percentiles for the adolescent's age group), in addition to demonstrating more varied and spontaneous eating and less resistance around foods that were previously challenging. In addition, parents may begin to indicate an increasing degree of confidence in their child's eating. Upon commencing this transition, parents may be encouraged to develop ideas about how to facilitate adolescent independence in the context of their food choices/consumption under increasingly less supervision, before moving on to broader challenges such as the recommencement of any suspended social activities and exercise.

This phase may last for several months, although sessions are typically conducted on a fortnightly basis, as opposed to weekly sessions in phase 1. However, it should be noted that if this transition is marked by consistent weight loss, the therapist may encourage the parents to reinitiate phase 1 and revisit any maintaining factors which were not successfully overcome during this phase.

26.3.3 Phase 3 of FBT

This phase typically commences when the adolescent is able to maintain their ideal body weight and demonstrate spontaneous and varied eating in a variety of social

contexts (Lock & Le Grange, 2013). The focus of treatment is completely shifted from food and weight management and is reoriented toward adolescent issues such as individuation, sexuality, and social relationships. Thus, the restoration of normal family and adolescent functioning becomes the central focus of treatment, and a thorough discussion of how AN may have impacted family life-cycle development assists the family in maintaining any structural changes which may have taken place throughout treatment. Parents and siblings may be encouraged to recommence any activities which were suspended or impacted throughout treatment, while the adolescent is supported in their exploration of their adolescent life without the presence of AN.

During this phase, the frequency of sessions further decreases, with sessions typically being held on a monthly basis. If any comorbid pathology remains, the family treatment may shift its focus to addressing the most prominent pathology remaining following the resolution of AN. Alternatively, if clinically indicated, the adolescent and other family members may be referred for individual treatment in further attempting to reduce any remaining psychopathology.

26.4 Empirical Evidence Evaluating FBT for Adolescent AN

The manualization of family-based treatment developed at the Maudsley hospital made FBT more accessible to a much wider audience, allowing widespread dissemination and more rigorous empirical evaluation, which has subsequently set FBT apart from other forms of family therapy. To date, a wealth of randomized control trials, meta-analyses, open trials, dissemination studies, and case series studies have been conducted (Couturier, Kimber, & Szatmari, 2013; Eisler et al., 2000; Ellison et al., 2012; Le Grange, Binford, & Loeb, 2005), and FBT has been established as the gold standard treatment for both restricting-type and binge-purge-type AN in adolescents (Le Grange et al., 2010).

Current research demonstrates that FBT results in 50–75 % of adolescents being weight restored within 1 year of commencing treatment (Le Grange & Eisler, 2009), typically resulting in greater improvements in body weight and reduction in eating disordered cognitions as compared to adolescent-focused individual treatment both at the end of treatment and at 6- and 12-month follow-up (Lock et al., 2010). Further still, the long-term benefits of FBT are sustained at 5-year follow-up in up to 90 % of cases (Lock, Couturier, Bryson, & Agras, 2006), which is thought to be particularly important given the high rates of relapse common in AN. In addition to long-term symptom remission, FBT also demonstrates swifter rates of symptom remission (Robin et al., 1999; Russell et al., 1987), thus mitigating the harmful medical effects of AN and reducing the costs of treatment by up to 70 % through a reduction in costly hospitalizations (Lock, Couturier, & Agras, 2008).

A recent Cochrane report concluded that FBT is a particularly effective form of treatment as compared to standard treatment for adolescents under 18 years of age who have had AN for less than 3 years, although did note the lack of comparison

between FBT and other forms of family therapy (Fisher, Hetrick, & Rushford, 2010). To date, Eisler and colleagues have compared separated versus conjoint forms of FBT (Eisler et al., 2000; Eisler, Simic, Russell, & Dare, 2007), although controlled comparisons between FBT and other theoretical approaches to family therapy are still warranted in further explicating the efficacy of family-based approaches. In addition, preliminary evidence demonstrates support for the use of FBT in young adults with AN (Chen et al., 2010), although further research is warranted before concluding on the efficacy of FBT in adults.

26.4.1 Dissemination of FBT

With the majority of the original research trials originating from the Maudsley group in London, and subsequent major research trials being undertaken by the authors of the treatment manual, an important question that remains is the extent to which FBT can be disseminated and implemented by other treatment providers. Current empirical evidence suggests that FBT can indeed be effectively disseminated, with outcome studies in several treatment centers demonstrating comparable rates of treatment efficacy and dropout to those reported by the specialized teams who initially developed and manualized FBT (Couturier, Isserlin, & Lock, 2010; Goldstein et al., 2012). However, despite evidence demonstrating that FBT may be effectively disseminated, current evidence suggests that clinicians working with adolescent presentations of AN may be unlikely to practice FBT with fidelity to the treatment model (Couturier et al., 2012). The barriers typically reported to clinician uptake of FBT include lack of professional support within treatment centers, the high demand which the treatment places on families, and lack of clinician comfort in working with families (Couturier et al., 2013). Thus, a directive for future research may be oriented toward enhancing clinician uptake of FBT, given that its efficacy is replicable in nonspecialized treatment centers (Goldstein et al., 2012).

26.4.2 Optimal Dosage of FBT

In evaluating the optimal dose of FBT, Lock and colleagues evaluated the efficacy of short-term versus long-term FBT (Lock, Agras, Bryson, & Kraemer, 2005). The short-term course of FBT consisted of 10 sessions over 6 months, whereas the long-term course of FBT comprised 20 sessions over 12 months. The results suggested that many adolescents with AN can be as effectively treated with short-dosage FBT as with long-term FBT, with both forms of FBT generally leading to comparable symptom resolution after 1 year. However, this study also indicated that adolescents with more severe AN and more obsessive AN-type thinking and those from non-intact families fared better in long-term FBT (Lock et al., 2005).

26.4.3 Mediators and Moderators of FBT

Despite FBT demonstrating a strong evidence base for the majority of adolescents, a significant minority of those with AN fail to benefit from treatment. As such, the identification of mediators and moderators of treatment outcome is essential to determine which patients are likely to benefit from FBT and identify ways in which treatment may be enhanced (Le Grange et al., 2012). For instance, early studies identified that families in which high levels of expressed emotion and criticism were present (from parents to children) did not typically fare very well in FBT (Le Grange, Eisler, Dare, & Hodes, 1992), while more recent studies have identified that parental warmth is predictive of favorable treatment outcome (Le Grange, Hoste, Lock, & Bryson, 2011). As such, in the context of high levels of expressed emotion, such families typically fare better in a separated form of family-based treatment, both at end of treatment and at 5-year follow-up (Eisler et al., 2000; Eisler et al., 2007).

Also mediating the efficacy of treatment, the presence of adolescent comorbidity has been found to be associated with both greater rates of treatment dropout and poorer rates of symptom remission, as compared to adolescents without comorbid concerns (Lock, Couturier, Bryson, & Agras, 2006). However, it is important to note that the mediating role of psychiatric comorbidity in FBT parallels the mediating role of psychiatric comorbidity in alternate adolescent-focused treatment modalities, although, when compared to other treatment modalities, adolescents with greater eating disorder severity, and greater eating-related obsessionality, tend to fare somewhat better in FBT (Le Grange et al., 2012).

Furthermore, in keeping with many other treatment modalities, the process of early change appears particularly important in terms of long-term prognosis throughout FBT. For instance, weight gain as early as session 2 has been found to be predictive of overall treatment outcome (Lock et al., 2006), while others have noted that a weight gain of approximately 3 % of one's ideal body weight in the first 4 weeks is accurately predictive of overall treatment outcome (Doyle, Le Grange, Loeb, Doyle, & Crosby, 2010).

26.4.4 Modifications and Augmentations to FBT

In the context of the minority of cases in which FBT fails to impact significant symptom remission, several attempts have been made to augment standard FBT treatment. For instance, parent-to-parent consultations were recently piloted as an augmentation to standard FBT and involved "graduating families" (whose child had recovered from AN) sharing their story of how they brought about their child's recovery in a therapist-led consultation with beginning families starting their course of FBT (Rhodes, Baillee, Brown, & Madden, 2008). Similarly, the introduction of online parent chat rooms in which parents consulted with up to 5 other parents actively engaged in FBT has demonstrated some promise in supporting parents through the challenges of FBT (Binford-Hopf, Le Grange, Moessner, & Bauer,

2013). Further modifications have included the development of a 1-week intensive form of outpatient FBT which, in addition to the core theoretical tenets of FBT, incorporated systemic family therapy practices, continuous parental coaching, and distress tolerance training and also implemented a behavioral contract to increase positive behaviors and reduce negative behaviors (Rockwell, Boutelle, Trunko, Jacobs, & Kaye, 2011).

26.5 Multifamily Therapy for Anorexia Nervosa

26.5.1 Theoretical Foundations of Multifamily Therapy for Anorexia Nervosa

Uniting several families in the treatment of ED was first undertaken in Dresden (Scholz & Asen, 2001) and London (Dare & Eisler, 2000), respectively, and was deemed especially suited to presentations of AN since both programs aimed to unite multiple families grappling with shared concerns, thus directly disrupting parents' frequently reported sense of struggling in isolation with their child's eating (Asen, 2002). Indeed, the presence of other families with eating disordered children in treatment was found to have strong destigmatizing effects for both parents and children alike, who, upon realizing the similarities between their own and other families, noted a sense of solidarity which allowed common feelings of failure, guilt, fear, and embarrassment to be shared and diluted between one another (Scholz & Asen, 2001).

A natural outgrowth of this destigmatized forum in which families become increasingly aware of the similarities facing them as they grapple with their child's eating disorder is an increasing awareness of the differences between families. Families sharing ideas and feedback among one another typically creates an atmosphere of mutual learning and reflection, allowing families to have experiences of both being helped by other families and acting as consultants to other families, further solidifying one's own sense of efficacy and agency (Dare & Eisler, 2000).

Thus, one distinct difference in the process of multifamily therapy for AN, as compared to FBT for individual families, lies in the fact that the role of the therapist may be further decentralized and may be more oriented toward ensuring that families connect and encourage mutual curiosity and feedback. Thus, a key characteristic of the therapist in multifamily therapy is the constant encouragement for families to respond directly to one another and share their observations, relying on the families' capacity to help drive the process of change rather than the therapist directly charging the families with the process of change.

26.5.2 The Practical Application of Multifamily Therapy for Anorexia Nervosa

While not yet widely disseminated, the foci of treatment of multifamily therapy for AN are oriented toward similar aims to those adopted throughout FBT, focusing on (1) the process of parents and children developing temporary strategies to manage eating disordered symptoms and (2) the process of adolescent development and how this may have been impacted by the presence of an eating disorder (Dare & Eisler, 2000). Further in keeping with FBT, multifamily therapy for AN follows 3 distinct phases which are addressed sequentially (Scholz, Rix, Scholz, Gantchev, & Thömke, 2005). Phase 1 is largely symptom-oriented and focuses entirely on parents adopting full authority in managing the eating difficulties of their child. Phase 2 typically commences after 6–8 weeks (after weight restoration) and focuses on relationship issues within the family, typically addressing issues such as family hierarchies, alliances, coalitions, communication patterns, and, in particular, the role of AN in the family context. The third and final phase of treatment is largely future-oriented and focuses on relapse prevention, in terms of both AN symptomatology and unhelpful patterns of family interaction, ensuring that issues of adolescent autonomy are explored.

Multifamily therapy for AN typically involves a combination of multiple family (with up to 6–8 other families) and individual family therapy meetings, which incorporate the reflexive use of structural, systemic, and narrative family therapy practices including video-recorded family meals, group feedback, "foster family meals" (in which adolescents rotate into different families for family meals), psychodrama and family sculpting practices, and psychoeducational information regarding the nature of ED and the nature of the "tricks" that those with ED are often coerced into employing in attempting to avoid confronting their food-based fears and anxieties (Dare & Eisler, 2000).

26.6 Empirical Evidence Evaluating Multifamily Therapy for Adolescent AN

To date, multifamily therapy has not undergone rigorous evaluation through controlled empirical trials, although preliminary findings demonstrate promising findings (Dare & Eisler, 2000; Scholz & Asen, 2001). Preliminary evidence suggests that patients and parents alike find this form of treatment extremely desirable and beneficial, which is evidenced by strikingly low rates of treatment dropout and adolescents themselves reporting enthusiasm about the multifamily group processes in deconstructing their perceived sense of having caused the eating disorder (Dare & Eisler, 2000; Scholz & Asen, 2001). In terms of symptom reduction, early research with relatively small sample sizes of seriously unwell adolescents supports the efficacy of this approach, noting that the majority of families who participated were able to develop strategies to bring about weight improvement and stabilization of eating in their children, although it should be

noted that sample sizes in these studies were too small to draw firm conclusions (Dare & Eisler, 2000; Scholz & Asen, 2001). However, despite ongoing clinical trials evaluating the efficacy of multifamily therapy for AN, the extant evidence base has identified several contraindications including the presence of a psychotic family member, severe depression or learning disability in family members, and the presence of intrafamilial violence or abuse (Scholz et al., 2005). What is clear from the extant evidence base is that multifamily therapy for AN remains a highly promising novel approach to the treatment of adolescent AN which conceptualizes change in a slightly different manner to the relatively well-validated FBT for individual families, although more controlled empirical research is required before firm conclusions can be drawn.

26.7 Couples Treatment for Anorexia Nervosa

Alongside the advances made through family-based treatment in the context of adolescent presentations of AN, poor rates of full symptom remission in adult presentations of AN have resulted in the call for age-appropriate adaptations of treatments which centrally leverage the involvement of family members in adult populations. However, the developmental complications of installing full parental control over adult children, in addition to many adults with AN reporting the most significant relationships to be with their partner as opposed to their parents, have resulted in a focus on centralizing the partners of those with AN in treatment.

One recent approach to centralizing the involvement of adult partners in treatment has been termed Uniting Couples in the treatment of Anorexia Nervosa (UCAN) (Bulik, Baucom, Kirby, & Pisetsky, 2011), which represents an innovative attempt at augmenting existing adult treatments with an adapted form of cognitive behavioral couples therapy. UCAN is closely tied to cognitive behavioral couples therapy, acknowledging that while one member of the couple may have AN, the disorder exists in an interpersonal and dyadic context (Bulik et al., 2011). For instance, a patient's partner may make up a large component of the patient's social landscape, which research demonstrates can both alleviate and exacerbate AN symptomatology. More specifically, UCAN employs cognitive behavioral methods and aims to address the core symptoms of AN within the couples context, helping each partner understand how AN has impacted the couple, focusing on body image, affection, sexuality, and relapse/recovery. The presence of both partners is thought to augment standard CBT and provide a source of in vivo support for the person with AN, in addition to allowing one's partners to reinforce appropriate eating patterns while ensuring that partners refrain from inappropriately providing reassurance. Furthermore, it is thought that addressing these concerns in the context of the couple also helps develop couple communication and problem-solving skills which benefit the couple in sustaining treatment gains (Bulik et al., 2011).

However, while preliminary anecdotal evidence suggests that UCAN may be an effective alternative treatment for adults with AN, no controlled empirical trials

have been completed, and as such, further research is warranted in establishing the efficacy of this promising treatment approach.

26.8 Family-Based Treatment for Bulimia Nervosa

FBT-AN is currently regarded as the first-line treatment for adolescent AN; however, the role of familial involvement in adolescent BN is relatively less well explicated. Studies of FBT-AN have provided indirect evidence supporting the use of FBT-BN (in curtailing bulimic features such as purging in the context of AN binge-purge subtype) (Eisler et al., 2000), though the first direct evidence describing significant symptom reduction in adolescents with BN came in a small case series in 1995 (Dodge, Hodes, Eisler, & Dare, 1995).

As a result, concerted efforts have been made to advance the family-based treatment of BN, and in a similar fashion to AN, a family-based treatment approach has been manualized, evaluated, and disseminated (Le Grange & Lock, 2007). Based largely on the blueprint of its predecessor for AN, FBT-BN acknowledges some of the core clinical similarities between AN and BN, including an overvaluation of weight and shape, elevated eating concerns, and a tendency to underreport symptom severity, and as such centrally leverages parental involvement in treatment. However, the treatment also takes into account some of the key differences between AN and BN. For instance, in general, BN symptoms are ego-dystonic; therefore, those with BN are less emotionally invested in their symptoms and are inherently more motivated to reduce symptoms such as bingeing and purging, which are often subjectively deemed shameful and distressing (Le Grange & Lock, 2007).

As a result, FBT-BN is similar to FBT-AN, with the same theoretical tenets and sustained behavioral symptom focus driving treatment. Similarly, parents are mobilized in assisting in the dissolution of symptoms, although crucially adolescents are more actively involved in FBT-BN, and a more collaborative effort between parents and their child is established. However, since many adolescents with BN may be within a healthy weight range, the aim of parental intervention is not to bring about weight restoration but rather to help install more stable patterns of eating, although the adolescent is weighed in every session to monitor fluctuations in weight. The sequential progression of FBT-BN follows a similar three-phase structure to FBT-AN, although the role of the parents throughout phase one differs in that they are not charged with the sole responsibility of symptom dissipation. Instead, parents actively solicit the input of the adolescent, since adolescents with BN typically present when somewhat older than those with AN and do not typically feature the same developmental regression (Le Grange & Lock, 2007). This ego-dystonic symptom profile allows for more adolescent involvement.

In evaluating the efficacy of FBT-BN, a relative paucity of controlled outcome studies is apparent, although to date two large-scale controlled trials have been conducted (Le Grange, Crosby, Rathouz, & Leventhal, 2007; Schmidt et al., 2007), and while they differed slightly in the form of family therapy delivered, they point

toward promising preliminary findings. In the Le Grange et al. study (2007), FBT-BN was found to be statistically and clinically superior to supportive psychotherapy for adolescent BN, both at end of treatment and at 6-month follow-up. Somewhat contrastingly, Schmidt and colleagues (2007) compared FBT-BN to a form of cognitive behavior therapy-guided self-care (CBT-GSC) in adolescents and young adults, noting no statistical differences in outcome between the two treatments at end of treatment and 6-month follow-up. This is a particularly important finding in light of the established evidence base for cognitive behavior therapy (CBT) in adult presentations of BN, and to extend these findings, a controlled comparison of FBT-BN and CBT may be important in establishing best practice for adolescent presentations of BN (Le Grange & Lock, 2007).

26.9 Substance Use Disorders and Addictions: Family-Based Approaches

26.9.1 Link Between Family Functioning and Substance Use Disorders

Substance use disorders (SUD) and family relationships are interconnected and therefore impact both the afflicted individual and the larger family structure. Given this notion, family members can play an important role in the treatment process (Copello, Templeton, & Velleman, 2006). A large body of literature has emerged that supports the theoretical and clinical rationale for including families in the treatment process (CSAT, 2004). According to a recent meta-analysis comparing outpatient treatment for adolescents with SUD, results provide strong support for family-based interventions as they yielded the greatest effect sizes when compared to other treatment approaches (Tanner-Smith, Wilson, & Lipsey, 2013). Additionally, there is strong evidence to support the inclusion of partners and adult family members when working with adult substance abusers. Leveraging the power of these relationships in adults can greatly improve engagement in treatment and treatment outcomes (Rowe, 2012). Based on these potential benefits, practice guidelines have started emphasizing the importance of incorporating families of substance abusers in treatment (AACAP, 1997), and influential policy-making groups have supported these standards (Drug Strategies, 2002).

26.10 Family-Based Approaches for Adolescent Substance Use Disorders

SUD typically onsets during adolescence, and similar to many other psychological disorders, early detection and intervention yields better long-term prognosis (Dennis & Scott, 2007). Given this information, treatment programs for adolescent SUD have received considerable attention over the last few decades. Currently, family-based treatment approaches for adolescents suffering from SUD have been

researched extensively with empirical evidence supporting the efficacy of such approaches (Hogue & Liddle, 2009). Consistent across the theoretical foundation of all family-based approaches for adolescent SUD is the aim to leverage the support of family members to achieve adolescent sobriety by making changes within the family environment and to change problematic behaviors and attitudes that will help maintain and encourage long-term recovery (Rowe, 2012). A brief review of the family-based approaches utilized with adolescents will be discussed below.

26.11 Multidimensional Family Therapy

26.11.1 Core Theoretical Tenets of MDFT

Multidimensional Family Therapy (MDFT; Liddle, 2002) is an outpatient treatment intervention that combines family therapy, individual therapy, drug counseling, and multiple systems-oriented intervention approaches. MDFT is a manualized treatment consisting of different treatment modules targeting four areas of social interaction: (1) the adolescent, (2) the parents, (3) the family environment, (4) and other extrafamilial interactions (e.g., educational system, child welfare, and justice system). Thus, sessions could involve either only the adolescent, both parents and teen, or only the parents. A core feature of treatment is to decrease risks related to dysfunction (e.g., parenting problems and interacting with substance-using peers) and enhance protective factors within the most accessible areas (Liddle, 2002).

Within the adolescent module, therapeutic goals that are both practical and meaningful to the adolescent are established. An objective of treatment is to attend to the "big picture" dimensions by promoting prosocial behaviors that can replace substance use and delinquent behaviors. Remediation efforts are made within the context of the teen's own life, values, and personal meaning. On the other hand, the parent module focuses on their role as caretakers and as individuals who may experience a decline in their motivation and capabilities to influence their child. The primary objectives are to promote and encourage parental love and commitment toward their offspring, instilling hope, and improving parenting practices. Expanding on the parents' commitment toward their child is a fundamental principle of MDFT and sets the stage for future changes. In the extrafamilial module, MDFT assumes that working with the adolescent and his/her parents is not sufficient to maintain behavior change. Therefore, clinicians often work with schools and/or juvenile justice systems to help motivate the adolescent and assist in the task of encouraging and maintaining sobriety. Thus, MDFT is a flexible treatment approach, tailored to the individual needs of the family. Treatment consists of 12 to 16 weekly or twice weekly 60- to 90-min sessions and can be applied in the home, community clinics, residential programs, and correction facilities (Liddle, 2002).

26.11.2 Empirical Evidence Evaluating MDFT for Adolescent SUD

Multidimensional interventions have shown promising results. Previous studies (Austin, MacGowen, & Wagner, 2005) demonstrate that MDFT has consistently been one of the top youth substance use treatments tested in the Cannabis Youth Treatment (CYT) multisite clinical trial and showed promising effects at 12-month follow-up when compared to control conditions (Dennis et al., 2002). An RCT was conducted to determine the efficacy of MDFT compared to an individual-based CBT intervention. In a sample consisting of 224 African American male adolescents, youth in MDFT demonstrated more rapid decreases in psychological involvement with drugs, and these findings were maintained through 12-month follow-up. These studies support the use of MDFT in reducing youth's drug use and other related problems (Austin et al., 2005).

26.12 Brief Strategic Family Therapy

26.12.1 Core Theoretical Tenets of BSFT

Brief Strategic Family Therapy (BSFT; Szapocznik, Hervis, & Schwartz, 2003) builds on earlier studies demonstrating promising results utilizing strategic-structural engagement strategies with troubled Hispanic males and their families (Szapocznik et al., 2003). BSFT is a structured, problem-oriented, and directive intervention developed specifically for adolescent SUD and other co-occurring problems such as conduct problems, oppositional behavior, delinquency, aggressive and violent behavior, and risky sexual behavior (Perrino, Gonzalez-Soldevilla, Pantin, & Szapocznik, 2000). BSFT is based on three fundamental constructs: (1) system (i.e., a family consisting of separate but interrelated parts where patterns of interaction are established), (2) patterns of interaction (i.e., maladaptive family interactions such as poor communication skills and negativity, which are not conducive to adolescent sobriety), and (3) strategy (i.e., an intervention that is directive, problem-oriented, and practical) (Szapocznik & Kurtines, 1989). At the beginning of treatment, BSFT clinicians often heighten the families' sense of urgency to motivate the family to take the appropriate action required to help their child. Thus, these three fundamental constructs (system, interactions, and strategy) of family systems make up the foundation of BSFT (Szapocznik & Williams, 2000).

BSFT is a brief treatment consisting of 12–16 sessions over 4 months. These guidelines are flexible, and the duration of treatment is always dependent on the clinical severity and the clinician's ability to facilitate positive changes within the family (e.g., reduce substance use and improve family relations). Multiple family members are encouraged to attend treatment sessions whenever possible to achieve sustainable effects in the family environment. Services extend beyond the family environment (e.g., school, peers, justice), and the locations of these services are flexible in BSFT (Szapocznik et al., 2003).

26.12.2 Empirical Evidence Evaluating BSFT for Adolescent SUD

Research indicates that the engagement strategies utilized in BSFT are effective at successfully retaining families in treatment while remaining cognizant of the cultural factors that influence retention (Coatsworth, Santisteban, McBride, & Szapocznik, 2001). Santisteban and colleagues (2000) conducted a clinical trial with 126 Hispanic adolescents suffering from SUD, and the results indicated that BSFT was more effective at reducing marijuana use when compared to group therapy. Additionally, BSFT was recently tested in the NIDA Clinical Trials Network in eight different sites including more than 400 adolescents and families (Santisteban et al., 2006), and the results are forthcoming.

26.13 Functional Family Therapy

26.13.1 Core Theoretical Tenets of FFT

Functional Family Therapy (FFT; Alexander & Parsons, 1982) is a widely disseminated evidence-based treatment developed for adolescents with SUD, conduct disorder, and delinquency. FFT is an ecological approach that incorporates tenets of systems-oriented family therapy as a means of altering maladaptive family patterns that may reinforce the adolescent's problems. Therefore, cultivating positive family interactions and functioning is key to decreasing the teen's involvement with substances. The goals of FFT are threefold: (1) reduce substance use and delinquent behaviors in the teen, (2) improve family functioning, and (3) increase productive time use in the adolescent. Underlying these primary goals is the importance of changing interaction patterns among family members and substituting substance use behaviors with more adaptive ways of functioning. Treatment goals are achieved in three distinct treatment phases: (1) engagement/motivation, (2) behavior change, (3) and generalization/termination. Each phase has detailed objectives, strategies, practices, and required therapist skills (Waldron & Brody, 2010).

A core and distinguishing feature of this approach is that the focus of treatment is relational in nature, and all family members currently living in the home are encouraged to participate in treatment. Additionally, extended family members or significant others that play a role in family functioning are also invited to partake. FFT goes beyond other treatment approaches in that it integrates and links behavioral and cognitive strategies to the ecological conception of the family to cultivate and maintain behavior change. Treatment typically occurs over 12 to 16 sessions consisting of 60- to 75-min periods. Initially, sessions are scheduled twice per week to initiate the change process and then followed with 1 weekly session and finally ending with sessions that are several weeks apart (Waldron & Brody, 2010).

26.13.2 Empirical Evidence Evaluating FFT for Adolescent SUD

Previous research indicates that FFT outperformed interventions such as juvenile court-based group therapy, group home therapy, psychodynamic therapy, or control conditions when it came to improving family relationships and decreasing recidivism among delinquent adolescents (Alexander & Parsons, 1973). Given the preliminary support for FFT (Waldron, Slesnick, Brody, Turner, & Peterson, 2001), it was compared to treatment as usual (TAU) and ecologically based family therapy (EBFT) with adolescent runaways. Results indicated that FFT was more effective at reducing substance use at 15-month follow-up when compared to TAU and EBFT. Given the efficacy of FFT, this approach has been widely disseminated in the USA as well as in Europe (Breuk et al., 2006).

26.14 Multisystemic Therapy

26.14.1 Core Theoretical Tenets of MST

Multisystemic Therapy (MST; Henggeler & Borduin, 1990) is based on a social ecological model aimed at changing the multiple risk factors that influence the development and maintenance of adolescent substance use and delinquency (Curtis, Ronan, & Borduin, 2004). The primary aims of MST are to improve parental skills and assist parents in establishing clear benefits and penalties for positive/negative behaviors. Additionally, adolescents are encouraged to interact with peers who engage in prosocial behaviors and discouraged from interacting with peers involved in delinquent activities. Thus, clinicians work both inside the home and in the community to empower parents. Depending on the case, this approach incorporates elements of family therapy, family preservation, parent training, and cognitive therapy models (Henggeler, Pickrel, & Brondino, 1999). Given that MST aims to reduce barriers that might prevent families from accessing treatment services, therapy is delivered in the home, and social support networks are established with friends, family, and community members. Generally, clinicians take on small caseloads (4–6 families) because they have to be available 24 h a day, 7 days a week, and provide treatment when convenient for the family. MST is a brief intervention (4 months) that entails multiple sessions each week (Henggeler & Borduin, 1990).

26.14.2 Empirical Evidence Evaluating MST for Adolescent SUD

MST has been empirically tested in the juvenile justice system, including juvenile substance court, and sexual offender programs. An RCT was conducted comparing MST, MST and contingency management, and standard drug court compared to family court in a sample of 161 teenagers suffering from SUD. Results indicated that the two MST conditions were more successful at decreasing drug use when

compared to standard drug court, while the three drug court approaches combined were more effective in reducing drug use and delinquent behaviors compared to family court (Henggeler et al., 2006). Therefore, these findings provide preliminary support for MST in substance-abusing youth.

26.15 Family-Based Approaches for Adult Substance Use Disorders

26.15.1 Invitational Intervention

A Relational Intervention Sequence for Engagement (ARISE; Garrett et al., 1998) is an invitational intervention that "invites" substance abusers to engage in treatment by mobilizing concerned relatives and other social networks. This method serves as a pretreatment engagement strategy that utilizes 3 phases to assist family members/friends to engage the inflicted individual into treatment. The first phase primarily focuses on coaching the concerned relative/friend to engage the substance abuser into treatment. Phase 2 begins if treatment has not been initiated during the previous stage and entails two to five sessions that could/could not involve the substance abuser. These sessions are used to mobilize the intervention network with the goal of establishing treatment engagement. The third and final level is only initiated if entry into treatment has not begun. During this meeting, relatives/friends establish consequences if the afflicted individual fails to engage in treatment or a self-help program (Garrett et al., 1998). Research suggests that ARISE is successful in engaging 83 % of treatment-resistant individuals into treatment (Landau et al., 2004).

26.15.2 Community Reinforcement and Family Training

Community Reinforcement and Family Training (CRAFT; Meyers, Miller, Hill, & Tonigan, 1999) is a family engagement approach and builds upon the community reinforcement approach for individuals suffering from alcoholism (Hunt & Azrin, 1973). A core feature of CRAFT involves restructuring environmental contingencies (i.e., social, vocational, and family life) to promote abstinence. CRAFT utilizes the leverage of concerned significant others (CSO) to encourage unmotivated substance abusers to engage in treatment and promote recovery. CRAFT teaches CSO to utilize behavioral strategies that encourage sobriety, prosocial activities, self-care, and treatment-seeking behavior in their loved ones and aims to alleviate stress (Meyers, Villanueva, & Smith, 2005). A pilot study (Kirby, Marlowe, Festinger, Garvey, & LaMonaca, 1999) with families of alcoholics shows promising results as CRAFT yielded greater retention rates and engagement of CSO and substance abusers when compared to a 12-step approach 10 weeks following the intervention. However, no significant differences were detected in terms of social or emotional functioning of CSO between the two modalities.

Similarly, another study demonstrated greater engagement in treatment among substance abusers (67 %) when compared to an Alcoholics Anonymous approach (29 %), but no differences were found in CSO functioning and showed no additional benefit of the CRAFT aftercare program (Meyers, Miller, Smith, & Tonigan, 2002). These findings demonstrate that CRAFT is effective at engaging addicts in treatment through the use of CSO.

26.15.3 Behavioral Couples Therapy

Behavioral Couples Therapy (BCT; O'Farrell & Fals-Stewart, 2006) is an approach that assumes that significant others can reward sobriety and decreasing relationship distress can reduce the risk of relapse in the afflicted individual. Therefore, BCT clinicians work with both the substance abuser and their partner to promote and encourage sobriety. BCT also incorporates a recovery contract between the partners and the clinician, integrates coping strategies to effectively deal with cravings and crises regarding relapse, teaches communication skills, and how to substitute enjoyable activities for substance use behaviors. Treatment sessions typically follow this sequence: (1) the clinician inquires about recent substance use, (2) the clinician and partners discuss compliance with regard to the contract, (3) homework is presented and discussed, (4) the partners discuss any relational difficulties, (5) the clinician introduces new material, and (6) new homework is assigned by the clinician. Treatment consists of 15–20 1-h sessions over 5–6 months (O'Farrell & Fals-Stewart, 2006). According to meta-analysis of 12 trials, BCT yielded a medium effect size when compared to individual treatments. Additionally, BCT showed greater effects at follow-up (Powers, Vedel, & Emmelkamp, 2008).

26.15.4 Behavioral Family Counseling

Behavioral Family Counseling (BFT; O'Farrell, Murphy, Alter, & Fals-Stewart, 2010) is an extension of BCT and has been developed for substance-abusing adults living with adult family members other than their significant others. The aims of treatment are similar to BCT, but treatment is targeted toward family members as opposed to significant others. Results from pilot studies show promising findings related to an increase in days abstinent, drug-related problems, and relationship functioning. In a pilot follow-up study, BCT was superior to an individual-based treatment in retaining substance-abusing individuals and was more effective in decreasing drug use and improving relationship functioning (O'Farrell et al., 2010). These encouraging pilot studies suggest the need for further investigation and larger randomized trials.

26.15.5 Comorbid Eating Disorders and Substance Use Disorders: Family-Based Approaches

Little evidence exists in documenting family-based approaches to comorbid ED and SUD, despite mounting evidence suggesting a high degree of comorbidity (Baker, Mitchell, Neale, & Kendler, 2010). However, while no specific guidelines direct best practice in such instances, family-based approaches to ED are unanimously clear in that the cessation of ED symptomatology and weight restoration ought to take priority before tackling any comorbidities (including SUD), due to the potential medical complications and cognitive impairments associated with severely low body weight and disordered eating (Lock & Le Grange, 2013). Thus, in the context of comorbid ED and SUD presentations, we recommend that disordered eating practices and weight restoration (if required) are the key foci of treatment in the first instance, with SUD being targeted once medical stability is ensured and eating disordered behaviors have become stabilized.

Concluding Comments

As the field of family therapy develops, the central involvement of family members in the treatment of many psychiatric conditions, particularly ED and SUD, continues to offer novel insights and therapeutic practices which enhance treatment outcome. FBT for ED has been established as the first-line treatment of adolescent presentations of AN, whereas an array of theoretical approaches to the family-based treatment of SUD in adolescents continue to demonstrate promising efficacy. However, further controlled research trials are needed in further explicating the process of change in family-based treatments, which in turn may further augment the clinical application of family-based approaches.

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Self-Help Approaches in the Treatment of Eating Disorders, Substance Use Disorders, and Addictions

27

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Abstract

Self-help interventions include step-by-step instructions that enable an individual to carry out an established treatment protocol either independently (pure self-help) or with minimal professional support (guided self-help). Generally, self-help interventions provide information about a given disorder as well as tools for building skills. In contrast, support groups—including 12-step programs—are relatively unstructured, do not target the reduction of symptoms, and do not involve therapists. In this chapter, we review the literature related to self-help and support groups for eating disorders, selected substance use disorders, gambling disorder, sexual addiction, and compulsive shopping; identify and describe self-help programs and resources for these problems; and provide recommendations for research and treatment. Very little research has evaluated the effectiveness of support groups in reducing symptoms of these disorders, and no research exists of self-help for individuals with comorbid eating disorders and addictions. However, reasonably strong evidence suggests that cognitive behavioral self-help treatments reduce symptoms of recurrent binge eating and bulimia nervosa. Similarly, self-help interventions for substance use disorders and gambling disorder using motivational and cognitive behavioral approaches improve outcomes. There is some evidence that assessment alone, or with personalized feedback, may be beneficial for those with gambling disorder. Further research examining the utility of self-help for these disorders, as well as indications for the use of pure versus guided self-help, is needed.

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27.1 Self-Help Approaches in the Treatment of Eating Disorders, Substance Use Disorders, and Addictions

Psychological interventions including cognitive behavioral therapy (CBT), behavioral therapy, and social network and environment-based therapy are the treatments of choice for alcohol abuse; contingency management is the treatment of choice for cannabis and stimulant abuse (National Institute for Clinical Excellence, 2011; National Institute for Health and Clinical Excellence, 2008). CBT is the treatment of choice for bulimia nervosa (BN) and binge eating disorder (BED) (National Institute for Clinical Excellence, 2004). However, dissemination of CBT to community therapists has remained quite limited (e.g., von Ranson, Wallace, & Stevenson, 2013), resulting in few individuals actually having access to it. Concerns have been raised regarding limited access to psychotherapies for other psychological disorders and the need for developing alternate models to reach more people affected by psychological symptoms (Kazdin & Blase, 2011). Self-help interventions have been proposed as a partial solution to this problem (Wilson & Zandberg, 2012), as they may be obtained regardless of one's location, improving accessibility of treatment for specific mental health problems at relatively low cost.

In this chapter, we review research related to self-help and support groups for eating disorders (ED) and selected substance and behavioral addictions, identify and describe self-help programs and resources, and provide recommendations for research and treatment. Numerous behavioral addictions have been posited, including gambling disorder (GD), binge eating, Internet addiction, video game addiction, hypersexual disorder, and compulsive buying disorder (Karim & Chaudhri, 2012). However, scant research on behavioral addictions has been conducted, and GD alone has accumulated a level of evidence that has resulted in it being grouped with substance use disorders (SUD) in DSM-5 (von Ranson, Wallace, Holub, & Hodgins, 2013). Accordingly, we limit our discussion of behavioral addictions to ED and GD in this chapter and note that the view of ED as addictions is debatable. With respect to SUD, we focus on alcohol and cocaine, which have received the most research study.

27.1.1 What Is Self-Help?

Although variously defined, we propose that self-help interventions entail the provision of step-by-step instructions to an individual, enabling them to carry out

an established treatment protocol either independently or with minimal expert support (Wilson & Zandberg, 2012). Instructions may be provided via book, CD-ROM, or Internet. Self-help interventions provide information about the disorder as well as skills training. There are two forms of self-help: one in which an individual is self-guided (*pure self-help*) and another in which an individual receives support from a professional in carrying out the intervention (*guided self-help*; also called supervised or therapist-assisted self-help). In general, the support that is provided in guided self-help is limited in both scope and time relative to traditional psychotherapy and at times is provided by someone other than a mental health professional. In guided self-help, the helper's role is to encourage use of the self-help program and answer questions about it.

Distinct from self-help are mutual-help groups and support groups, including 12-step programs such as Alcoholics Anonymous, Narcotics Anonymous, and Overeaters Anonymous. While support groups are widely used (Alcoholics Anonymous, 2013), are affordable, and provide valuable emotional support (Russell-Mayhew, von Ranson, & Masson, 2010), they differ from self-help programs in that they are relatively unstructured, do not target the reduction of symptoms, and do not involve therapists. Little evidence is available regarding the efficacy of 12-step programs in reducing symptoms, in large part due to the anonymous nature of these programs (Russell-Mayhew et al., 2010). However, one randomized controlled trial found that Twelve-Step Facilitation therapy, in which a therapist encourages clients to work on the 12 steps and to actively participate in 12-step fellowships, is associated with positive outcomes that are similar to those associated with CBT and Motivational Enhancement Therapy (Project MATCH Research Group, 1997).

27.2 12-Step Fellowships

27.2.1 Alcoholics Anonymous and Narcotics Anonymous

Alcoholics Anonymous (AA) was developed in 1935 out of a Christian evangelical movement (Humphreys, 2004). AA's first textbook, "Alcoholics Anonymous" (known in AA as The Big Book), was published in 1939 and described the philosophy and methods of the AA program as well as case histories of recovered alcoholics. The AA organization is guided by a set of spiritual principles known as the 12 traditions and is run by elected volunteers. It is the most ubiquitous mutual-help group, with approximately 114,000 groups and 2,000,000 members worldwide (Alcoholics Anonymous, 2013).

AA views alcoholism as a disease with moral, spiritual, and physical components (Humphreys, 2004). The goal in AA is abstinence from alcohol; however, recovery involves more than abstinence. In AA's program of change, the 12 steps, the ultimate goal is to experience a spiritual awakening. Spirituality is fundamental to the AA program, and belief in a Higher Power is viewed as essential for personal recovery (Lembke & Humphreys, 2012b). Other key aspects of the AA program include obtaining a sponsor, attending meetings, and helping others

(Lembke & Humphreys, 2012b). Sponsors provide one-on-one support and guidance in the completion of the 12 steps. AA meetings provide a forum for alcoholics to share their story, discuss recovery-related topics, and read and discuss the 12 steps and 12 traditions. Personal recovery is viewed as a lifelong process, and lifetime membership is encouraged.

Narcotics Anonymous (NA) was developed in 1953, and its basic textbook, "Narcotics Anonymous," was published in 1983. Currently 61,800 meetings are held in 129 countries around the world (Narcotics Anonymous, 2012). The NA program is the same as that of AA; in the 12 steps the word "alcohol" was changed to "addiction" (Narcotics Anonymous, 1988). NA makes no distinction between alcohol and other drugs, and the goal of abstinence applies to all mind-altering substances (Narcotics Anonymous, 1988).

Most of the research on the effectiveness of AA and NA has focused on their usefulness as adjuncts to formal substance abuse treatment (Kelly, 2003). Overall, research has shown that more frequent participation in AA/NA following formal treatment is associated with higher rates of abstinence, fewer substance-related problems, and lower health-care utilization (e.g., Humphreys, 2004). Furthermore, increased attendance at 12-step meetings predicts increased abstinence and improved substance-related outcomes, even after controlling for other treatment-related factors (e.g., Pagano, White, Kelly, Stout, & Tonigan, 2013). Research has also shown that increased involvement in a 12-step program by obtaining a sponsor early in recovery, becoming a sponsor, and completing the 12 steps is associated with increased abstinence rates (Pagano et al., 2013; Tonigan & Rice, 2010). Twelve-step treatment is associated with lower health-care utilization and reduced health-care costs than CBT (Humphreys & Moos, 2007).

Research has begun to investigate outcomes of individuals with comorbid psychiatric disorders in 12-step programs, although no studies have examined individuals who have comorbid ED or ED symptoms with SUD. Social phobia may be a barrier to successful 12-step treatment by making individuals reluctant to ask someone to be their sponsor (Book, Thomas, Dempsey, Randall, & Randall, 2009; Tonigan et al., 2010). A study comparing men with and without comorbid depression and SUD found that 12-step involvement was only related to improved outcomes for the non-comorbid group (Kelly, McKellar, & Moos, 2003). Taken together, these studies suggest that individuals with comorbid mood and anxiety disorders may not benefit as much from support group involvement as those who only have SUD.

Research has investigated mechanisms of change associated with 12-step membership. Two studies have shown that 12-step programs help men improve their social relationships and increase their use of active coping strategies (Humphreys, Mankowski, Moos, & Finney, 1999; Kelly & Hoeppner, 2013). These same factors do not fully account for AA's effect on substance-related outcomes for women (Kelly & Hoeppner, 2013). Overall, research suggests that participation in 12-step programs after formal treatment is associated with improved substance-related outcomes; however, individuals with certain comorbid disorders may benefit less from these programs. Although it appears that 12-step programs help men improve

their social relationships, we know very little about how they impact change in women.

27.2.2 Other Substance-Related 12-Step Programs

Although AA and NA are the most widely recognized 12-step programs for substance addictions, other 12-step programs exist to help individuals achieve abstinence from particular substances, such as marijuana (*Marijuana Anonymous*), cocaine (*Cocaine Anonymous*), heroin (*Heroin Anonymous*), crystal meth (*Crystal Meth Anonymous*), prescription pills (*Pills Anonymous*), and nicotine (*Nicotine Anonymous*). These programs are all based on the 12 steps of AA, except that the word "alcohol" is substituted with the other substance.

27.2.3 Double Trouble in Recovery

Double Trouble in Recovery (DTR) was developed in 1989 to target individuals with SUD and comorbid psychiatric disorders and currently has over 200 meetings throughout the United States (Magura, 2008). The most commonly reported comorbid psychiatric diagnoses were schizophrenia, depression, and bipolar disorder (Laudet, Magura, Vogel, & Knight, 2003). DTR is based on the 12-step model; however, the first and twelfth steps have been adapted to add mental health issues to substance abuse. Many members attend both DTR and traditional 12-step groups; however, DTR meetings allow members to receive support from their dually diagnosed peers as well as the opportunity to openly discuss issues that can be stigmatizing in traditional 12-step fellowships, such as the use of psychiatric medication (Magura, 2008).

One longitudinal study of DTR members found that rates of abstinence from substance abuse were 72 % at 1-year follow-up and 74 % at 2 years (Magura, 2008). This study also found that regular, ongoing attendance at DTR meetings was associated with better medication adherence. Thus, DTR may help dually diagnosed individuals improve substance-related problems and psychiatric difficulties.

27.2.4 Gamblers Anonymous

Gamblers Anonymous (GA) was developed in 1957. Meetings are held all over the world; however, the organization has not grown at the same rate as AA or NA (Browne, 1991). This 12-step program differs from AA in important ways (Browne, 1991). First, God and spirituality are de-emphasized in both the 12 steps and the 12 traditions, and in GA, these concepts are referred to as the "recovery program" and "unity program" (Gamblers Anonymous, n.d.). Second, GA views gambling as

the central problem for the compulsive gambler (Gamblers Anonymous, n.d.) rather than a spiritual and psychological illness.

Little research has investigated the effectiveness of GA in increasing abstinence. Two randomized controlled trials (RCTs) have shown that pathological gamblers assigned to attend GA were less likely to abstain from gambling than those who received professional treatment, but they also attended very few meetings, limiting the conclusions that can be drawn (Grant et al., 2009; Petry et al., 2006). In a study of individuals in treatment for a gambling problem, Petry (2003) found that those with a history of GA involvement were more likely to abstain from gambling after 2 months than those without a previous history of GA. One study investigated the effectiveness of GA as a stand-alone treatment. In a survey of 75 people who had been members of GA for at least 1 year, Oei and Gordon (2008) found that those with abstinence of 1 year or longer reported greater GA attendance and participation, social support, and adherence to the GA recovery program. It appears that GA attendance, coupled with professional treatment, may help individuals achieve short-term abstinence from gambling, particularly if they choose to attend GA on their own.

27.2.5 Overeaters Anonymous

Overeaters Anonymous (OA) was developed in 1960, although its first textbook was not published until 1990. Currently, there are approximately 54,000 members and 7,000 meetings in over 80 countries (Overeaters Anonymous, 2013). The only requirement for membership in OA is the desire to stop compulsive eating; therefore, members present with a wide variety of dysfunctional eating patterns and difficulties with food (Russell-Mayhew et al., 2010).

The OA program is similar to the AA program; in the 12 steps, the word "alcohol" was changed to "food." Like AA, the goal of OA is abstinence, although abstinence is defined differently by each member (Russell-Mayhew et al., 2010): it may involve abstinence from particular foods (i.e., white flour, sugar) or abstinence from behaviors (i.e., binge eating, purging). Given the impossibility of abstaining entirely from eating food, the OA program encourages the use of a food plan in addition to the 12 steps to help members achieve recovery. The OA program does not outline the food plan that should be adopted; this is left up to individual members. Members often adopt a highly structured food plan when they join OA and eventually move on to a more flexible plan as their recovery progresses (Wasson & Jackson, 2004). Another way that OA differs from other 12-step groups is that most members initially join OA to lose weight. Although members report that their focus shifts toward spiritual growth and psychological health over time, success in OA is still often measured by weight loss (Russell-Mayhew et al., 2010).

No research has examined the efficacy of OA at diminishing overeating. According to a 2010 member survey, at least half self-reported "significant improvement" in daily functioning, physical health, weight issues, mental and emotional health, and relationships (Overeaters Anonymous, 2013). However,

research suggests ED psychopathology may be common among OA members (e.g., von Ranson, Russell-Mayhew, & Masson, 2011).

27.2.6 Eating Disorders Anonymous

Eating Disorders Anonymous (EDA) was founded in 2000, with the aim of helping individuals who are suffering from an eating disorder (Eating Disorders Anonymous, n.d.). The goal of the program is to achieve balance, rather than abstinence. EDA does not utilize a food plan as part of their program, and they discourage any form of rigidity around food. Members are encouraged to seek help from outside professionals such as dieticians and therapists who are trained to work with eating disorders. The definition of recovery in EDA is to live without obsessing over food, weight, or body image (Eating Disorders Anonymous, n.d.).

27.2.7 Anorexics and Bulimics Anonymous

Anorexics and Bulimics Anonymous (ABA) was founded in 1993 (Anorexics and Bulimics Anonymous, 2013). The program is directed at helping individuals with anorexia and bulimia nervosa; however, they also welcome anyone suffering from other disordered eating patterns including compulsive overeating, binge eating, and excessive exercise. Unlike other ED-focused 12-step programs, which define the problem as centered on food, ABA defines the problem as the practices that allow members to feel *in control* of food, weight, and body shape (Anorexics and Bulimics Anonymous, 2009). Members of ABA are encouraged to obtain "meal support" in early recovery, which involves an outside professional planning, preparing, and serving all meals and snacks to the member until they are "no longer afraid of getting fat" (Anorexics and Bulimics Anonymous, 2009). In ABA, recovery is defined as surrendering to the way that your Higher Power wants you to eat and accepting the body that your Higher Power wants you to have (Anorexics and Bulimics Anonymous, 2009); in this way, the program is focused on body acceptance.

27.2.8 GreySheeters Anonymous

GreySheeters Anonymous (GSA) is a 12-step program directed at individuals who suffer from compulsive overeating (GreySheeters Anonymous, 2010). GSA has a strict food plan associated with their program, and the goal for recovery is to achieve "GreySheet abstinence." GreySheet abstinence is defined as eating three weighed and measured meals per day from the GreySheet, with absolutely nothing in between meals except for black coffee, tea, and diet soda (GreySheeters Anonymous, 2010). Members can only obtain a GreySheet from their sponsor, who must

have 90 consecutive days of GreySheet abstinence (GreySheeters Anonymous, 2010).

27.2.9 Sex Addiction 12-Step Programs

There are three 12-step programs aimed at helping individuals overcome their addiction to sex or love: *Sexaholics Anonymous*, *Sex Addicts Anonymous*, and *Sex and Love Addicts Anonymous*. These programs have each adapted the 12 steps from Alcoholics Anonymous to treat sexual addiction; however, they differ in terms of the specific target behaviors and their definition of abstinence.

Sexaholics Anonymous views lust as the primary problem and targets lustful thoughts, feelings, and behaviors. This program has the strictest definition of abstinence, which involves ceasing any form of sex with oneself or partners other than one's spouse (Sexaholics Anonymous, 2013). Sex Addicts Anonymous views sexual thoughts and behaviors as the problem, and members define abstinence based on their own needs. In this program, members outline the specific behaviors that they want to abstain from, the behaviors that they are not sure about, and the behaviors that they view as healthy and acceptable (Sex Addicts Anonymous, 2013). Sex and Love Addicts Anonymous targets a wider range of behaviors than the other two 12-step programs for sexual behaviors. This program is directed at individuals who either compulsively engage in or avoid sexual behaviors or emotional attachments with others. Similar to Sex Addicts Anonymous, this program allows each individual member to define abstinence based on their own needs (Sex and Love Addicts Anonymous, 2012).

27.2.10 Debtors Anonymous

Debtors Anonymous (DA) is a 12-step program aimed at individuals who have excessive amounts of unsecured debt (Debtors Anonymous, 2011). According to DA, signs of compulsive "debting" include compulsive shopping, frequent borrowing, poor savings habits, being unclear about one's financial situation, and having difficulty meeting one's financial obligations (Debtors Anonymous, 2011). In addition to using the typical 12-step tools such as meeting attendance, the 12 steps, and sponsorship, DA members use additional tools aimed at improving their financial situation (Debtors Anonymous, 2011). Members maintain records of their daily spending habits, income, and repayment of any debts. Members of DA also organize "Pressure Relief Groups" in which other recovering DA members help review the individual's current financial situation and create a spending plan (budget) and an action plan outlining how the individual will pay off their debts and improve their financial situation (Debtors Anonymous, 2011).

27.3 Non-Twelve-Step Mutual-Help Groups

Out of dissatisfaction with certain tenets of the 12-step programs and a lack of diversity in addiction treatment options, several non-12-step mutual-help groups have been developed, primarily for SUD.

27.3.1 Rational Recovery

Rational Recovery (RR) was developed in 1986 out of disagreement with several of AA's basic tenets, such as being powerless, needing to surrender to a Higher Power in order to recover, the necessity of lifetime membership in a recovery group, and the need to label oneself "an addict" (Trimpey, Velten, & Dain, 1992). Originally, RR was based on the principles of Rational Emotive Behavior Therapy (REBT), a form of cognitive behavioral therapy. At that point, RR viewed irrational thoughts about addiction and sobriety as the cause of addiction, and treatment focused on identifying and disputing these thoughts (Bishop, 1995; Trimpey et al., 1992).

In a dramatic change occurring in 1994, RR moved away from REBT principles. The founder of RR, Jack Trimpey, has also developed a controversial structural model of addiction that posited that the human brain has two parts: the cortex and "the brain of The Beast" (Schmidt, 1996). According to Trimpey, the brain of The Beast is only concerned with obtaining substances, and the way to overcome addiction is to refuse to supply The Beast with what it wants. Abstinence is the goal of RR and the Addictive Voice Recognition Technique (AVRT) is the way to achieve this goal (Schmidt, 1996). AVRT involves personifying all thoughts that encourage relapse or express ambivalence about abstinence as "The Beast" in order to separate these thoughts from the self. The technique then requires members to track The Beast by identifying the frequency, intensity, and tone of The Beast, as well as the circumstances in which these thoughts occur. The current program does not offer meetings and is viewed as incompatible with any recovery group membership (Rational Recovery, 2013).

No information is available on how effective RR is in its current form. However, Schmidt, Carns, and Chandler (2001) investigated the efficacy of a professional substance abuse treatment program that was based on current RR principles. They found significant decreases in drug use severity in the group who received RR-based treatment; however, the study used an extremely small sample ($n = 10$ per group) and did not examine RR as a stand-alone treatment.

27.3.2 SMART Recovery

Psychologists and mental health professionals who were originally affiliated with RR when it was still based on REBT principles voted to separate from RR and create *SMART Recovery* (SMART Recovery, 2013). Most of the inspiration for SMART comes from REBT, cognitive behavioral therapy, and motivational

interviewing. The goal of SMART is abstinence from addiction, and the program is directed toward people suffering from both substance and behavioral addictions. This four-point program involves (1) building and maintaining motivation; (2) coping with urges; (3) managing thoughts, feelings, and behaviors; and (4) living a balanced life (Horvath & Yeterian, 2012). The primary techniques used are the stages of change model, cost-benefit analyses, coping with urges and managing emotional upsets, constructing a hierarchy of values, and role-playing and rehearsal. Meetings are small and involve active discussion among members. SMART encourages members to be involved in the program for months or years, but not for their whole lifetime (SMART Recovery, 2013).

There are currently over 690 SMART groups worldwide and multiple online meetings daily (Horvath & Yeterian, 2012). Although largely based on evidence-based principles, no research has examined its efficacy. According to a 2012 member survey, 60 % of members viewed SMART as an excellent resource for their recovery, and another 24 % viewed it as a very good resource (SMART Recovery, 2012).

27.3.3 Moderation Management

Moderation Management (MM) was developed in 1994 by Audrey Kishline out of dissatisfaction with her own participation in a 12-step-oriented treatment (Lembke & Humphreys, 2012a). The target population of MM is risky drinkers who do not have alcohol dependence. MM views alcohol *misuse* as a habit that can be alleviated using behavior modification techniques (Moderation Management, n.d.). As its name suggests, the goal in MM is moderation rather than abstinence, unlike AA and most other addiction treatments.

MM involves a nine-step cognitive behavioral (CB) program that involves self-monitoring, goal setting, building commitment to change, learning skills to both avoid and control drinking, identifying and managing personal triggers, identifying alternative nondrinking activities, and lapse prevention (Moderation Management, n.d.). Members are encouraged to attend MM meetings either face-to-face or online and to learn about the MM program. Members undertake a 30-day abstinence period and then resume drinking cautiously while adhering to MM guidelines for moderate drinking: for women, no more than nine standard drinks per week and a maximum of three drinks in a day; for men, no more than 14 drinks per week and a maximum of four in a day; and for both, having three to four nondrinking days per week (Moderation Management, n.d.).

Currently, there are 24 face-to-face meetings listed on the MM website, mostly in the United States (Moderation Management, n.d.). Despite the limited number of face-to-face meetings, MM is accessed frequently on the Internet (Lembke & Humphreys, 2012a). Little research has investigated the efficacy of MM. One longitudinal RCT compared MM to MM plus an additional web-based program called Moderatedrinking.com (Hester, Delaney, & Campbell, 2011). Both groups made clinically significant improvements in reducing their alcohol consumption

and alcohol-related problems; however, the group that used Moderatedrinking.com made greater improvements. In the absence of a control group, we cannot make conclusions regarding the effectiveness of MM as a stand-alone treatment.

27.4 Self-Help Programs for Eating Disorders

A large number of self-help books for ED have been published in the popular press, but only a few provide detailed, step-by-step instructions to assist one in carrying out an empirically tested program. Almost all self-help manuals for ED are CB in orientation. More details on CB self-help books and programs are provided below in approximate chronological order, as well as empirical research on each program.

It is also worth noting that one non-CBT self-help treatment approach has received some study. Preliminary research of a guided self-help version of dialectical behavior therapy for BED, which teaches individuals how to regulate their emotions, suggests it may offer a possible alternative to other, more extensively studied treatment approaches (Masson, von Ranson, Wallace, & Safer, 2013). Another treatment for ED, family-based treatment, also known as the Maudsley approach, has been developed and been studied (Lock & le Grange, 2012). Although the aim of family-based treatment is to support parents in helping their child in overcoming AN and BN, the manuals are not intended for use in self-help format (le Grange & Lock, 2007; Lock & le Grange, 2012).

27.4.1 Bulimia Nervosa, Binge Eating Disorder, and Recurrent Binge Eating

As BN and BED both involve recurrent binge eating, they are often grouped together in research studies as well as self-help manuals. Four CB self-help programs have been the focus of the majority of empirical research on the subject.

Getting Better Bit(e) by Bit(e) (Schmidt & Treasure, 1997). This well-studied self-help manual for BN and BED summarizes CBT for ED as provided at Maudsley Hospital in London (Schmidt & Treasure, 1997). It covers psychoeducation as well as practical strategies aimed at building skills and improving ED symptoms and quality of life. Each chapter includes vignettes illustrating how other patients have dealt with specific ED-related problems. It is intended to be read and the program followed over about 3 months. The first six chapters provide the core of the program and help readers to evaluate the severity of their bulimic symptoms; consider the pros and cons of changing those behaviors; monitor antecedents and consequences of their symptoms; learn a structured approach to problem solving; understand facts about weight and dieting, including effects of starvation and the ineffectiveness of purging behaviors for controlling weight; consider functions of bulimic symptoms; and learn strategies to improve body satisfaction. Subsequent chapters address the value of moderate exercise; preventing relapse; how childhood abuse might affect one as an adult; how to

identify and challenge automatic thoughts; developing assertiveness; identifying self-destructive behaviors, including alcohol and drug abuse, shoplifting, and overspending; how to improve relationships with others; how to handle work-related problems; and evaluating one's progress after completing the book.

In addition to providing basic CBT, *Getting Better Bit(e) by Bit(e)* also includes sections on improving body acceptance, planning a relapse, addressing childhood abuse, improving assertiveness, and handling work versus self-care. These additions make this manual more comprehensive than other CBT self-help manuals. The authors attempt to set realistic expectations for progress rather than a complete cure. A clinician's guide that is aimed at improving compliance and motivation is available to accompany the manual, allowing it to be used in guided self-help form (Schmidt & Treasure, 1997).

Bulimia Nervosa and Binge-Eating: A Guide to Recovery (Cooper, 1995). In this book's first part, Cooper defines and describes BN and binge eating and explains how they relate to other ED. In the second part, he presents a structured CBT self-help manual composed of six elements, all focused on interrupting factors that are maintaining the ED. They include self-monitoring of eating, symptoms, and context; establishing a meal plan; learning to intervene to prevent binge eating; problem solving; eliminating dieting; and changing unhelpful beliefs. Cooper recommends that a "helper" be recruited to assist the person who is using the manual.

Overcoming Binge Eating (Fairburn, 1995). Described as an extension of Cooper's 1995 manual, this book presents the most widely tested version of core concepts in CBT for BN, BED, and their variants. The book includes a summary of scientific knowledge about binge eating problems and a CBT self-help manual, which can be used alone (pure self-help) or with the assistance of a therapist (guided self-help). After walking readers through a preliminary consideration of whether self-help is appropriate for them, the following CB steps are presented: self-monitoring of food intake, cues, and context; weekly weighing; establishing a pattern of regular eating and ceasing purging behaviors; substituting alternate behaviors for binge eating; problem solving and reviewing progress; addressing dieting and food avoidance; and preventing relapse and coping with other problems, such as depression, low self-esteem, or anxiety.

A substantially expanded second edition of this book, including a new module on body image, is due to be published in August 2013. This revision of *Overcoming Binge Eating* presents a self-help form of enhanced CBT (i.e., CBT-E), a modification and extension of CBT (Fairburn et al., 2008). CBT-E takes a transdiagnostic approach that emphasizes common symptoms of various ED. Using a modular structure for the main part of treatment, it targets mechanisms theorized to maintain ED symptoms and includes updated approaches to capitalizing on early change, improving control over eating, targeting weight and shape concern, and relapse prevention (C. G. Fairburn, personal communication, June 4, 2013).

Overcoming Bulimia (Williams, Aubin, Cottrell, & Harkin, 1998). This CBT self-help program is available in CD-ROM form as well as online. It includes eight sessions, which cover information about bulimia, its consequences, and a vicious

cycle that keeps it going; why people develop ED and how keeping food diaries can help enact change; establishing healthy eating patterns; the role of thoughts in maintaining bulimia; increasing assertiveness and activity level; problem solving; facing fears; and review of progress.

27.4.2 Research Evidence

At least 24 controlled studies of CBT self-help treatments for BN, BED, and recurrent binge eating have been published (Wilson & Zandberg, 2012). Systematic reviews have consistently concluded that CBT self-help interventions, pure or guided, are better than no treatment for people with bulimic and binge eating symptoms (e.g., Stefano, Bacaltchuk, Blay, & Hay, 2006; Sysko & Walsh, 2008; Wilson & Zandberg, 2012). The evidence supporting the efficacy of CBT self-help treatments is more consistent for BED than for BN (Wilson & Zandberg, 2012). Based on these findings, treatment guidelines have recommended the use of evidence-based self-help programs for BN and BED as a possible first step in treatment (e.g., National Institute for Clinical Excellence, 2004). Outcome measures have shown positive effects on the frequency of, and not necessarily abstinence from, binge eating and purging behaviors, as well as other psychological symptoms and interpersonal functioning. Thus, for many individuals, a self-help intervention may be better viewed as a means to *improve* rather than *cure* an ED.

For treatment of BN, two small studies comparing CBT self-help to other credible self-help treatments—for assertion and perfectionism—found no difference between treatment groups, suggesting that the effects of self-help treatment may be nonspecific (Carter et al., 2003; Steele & Wade, 2008). In other words, self-help treatment may improve bulimic symptoms even if it does not target bulimic symptoms directly. If replicated in studies adequately powered to identify differences in treatment effects, these findings could have important implications for provision of self-help for BN. Interestingly, they are consistent with research that compared individual CBT to interpersonal psychotherapy and unexpectedly found comparable effects on bulimic symptoms at 1-year follow-up (Agras, Walsh, Fairburn, Wilson, & Kraemer, 2000).

27.4.3 Anorexia Nervosa

Treatment of AN presents different issues compared to BN or BED, given the characteristically low motivation to change among people with AN. For example, researchers have had difficulty conducting treatment outcome studies with AN patients due to very low retention rates (Halmi et al., 2005). In addition, some authors have argued that, because of the unique medical and psychological needs of those with AN, self-help approaches are contraindicated (Wilson & Zandberg, 2012). To date, only two studies have evaluated self-help interventions for AN.

Fichter, Cebulla, Quadflieg, and Naab (2008) developed and tested a CBT-guided self-help program for people with AN, binge/purge type. Following an introductory section providing psychoeducation about AN and its causes and consequences, six main topics were addressed over 6 weeks: stocktaking and objectives, developing healthy eating patterns, analysis of what sustains an individual's ED symptoms and identifying alternative ways to cope with problems, coping with negative thoughts, body image and acceptance, and dealing with feelings. Guided self-help involved supportive phone contact with a clinical psychologist for up to 30 min weekly. A wait-list controlled trial of this program was conducted with 102 adults waiting to receive inpatient ED treatment (Fichter et al., 2008). The program was effective in shortening subsequent inpatient stays and improving body image and bulimic symptoms. However, 11 of 68 participants (16 %) refused to complete the self-help program, believing there would be no benefit.

Rather than providing self-help to those with AN directly, another study evaluated an interactive, web-based program for carers of people with AN called *Overcoming Anorexia Online* (Grover et al., 2011). Carers may be a family member, friend, or partner of a person with AN. The OAO program uses a CBT and systemic framework and includes eight modules. The first three core modules address psychoeducation about factors affecting the willingness of AN sufferers to change, interrupting the vicious cycles that serve to maintain AN, and improving communication. Modules four to seven teach skills such as how to provide support at mealtimes, identify and manage medical risk in AN, and manage problem behaviors. Module eight provides additional information on treatment and services. An RCT of OAO provided with supportive guidance by phone or e-mail, compared to support from a community ED organization, with 64 carers showed reduced anxiety and depression symptoms among those receiving OAO (Grover et al., 2011).

27.5 Self-Help Programs for Substance Addictions

A number of different self-help interventions have been developed to help problem drinkers reduce their drinking to low-risk levels. These interventions exist in both paper and web-based formats and usually fall within one of two general categories: personalized feedback assessment or structured interventions based on CB and motivational strategies. Generally, investigations of self-help interventions have shown that they can be effective at reducing alcohol consumption and consequences associated with problematic alcohol use (Apodaca & Miller, 2003; Newman, Szkodny, Llera, & Przeworski, 2011).

Personalized feedback interventions provide users with an assessment of their current alcohol use and normative data to allow them to compare their drinking with those of other individuals. Combining personalized feedback with a self-help book can lead to reductions in alcohol consumption and alcohol-related consequences in the general population and in medical settings (Apodaca & Miller, 2003). However,

the effectiveness of either intervention on its own is questionable: one study found no support for the effectiveness of either intervention on its own (Cunningham, Koski-Jannes, Wild, & Cordingley, 2002), and in another, participants who only received personalized feedback reported less severe alcohol-related problems, but no differences in alcohol consumption or drinking-related consequences (Cunningham, Humphreys, Koski-Jannes, & Cordingley, 2005).

Web-based CB interventions to reduce problematic drinking vary but often include a self-assessment of drinking consumption, drinking patterns, and current motivation to change; goal setting; self-monitoring one's drinking; teaching strategies for coping with risky situations and for dealing with lapses; and access to an online forum to facilitate social support from peers who are also using the program.

Drinking Less is a web-based CB self-help intervention that is designed to be completed in 6 weeks (Riper et al., 2008). Community participants who received the *Drinking Less* intervention drank less and became more likely to drink within healthy limits relative to those who received a psychoeducational brochure on alcohol (Riper et al., 2009; Riper et al., 2008). The effectiveness of a television-based version of *Drinking Less* was also investigated (Kramer et al., 2009). This intervention included five 25-min episodes that encouraged viewers to use the self-help manual and access the *Drinking Less* website. The intervention group reduced their alcohol consumption and reported fewer alcohol-related problems than a wait-list control group. Limitations were that less than half of the participants used the website and that only 17–18 % of participants were actually drinking within healthy limits post-intervention.

Blankers, Koeter, and Schippers (2011) investigated the effectiveness of a web-based CBT and motivational intervention that included personalized feedback. Participants ($N = 205$) were randomly assigned to a self-help intervention, therapist intervention, or wait-list control group. Although some short-term improvements were seen for both interventions at 3 months and for the therapist intervention at 6 months, participants were still drinking at hazardous levels. Overall, investigations of web-based CBT self-help interventions suggest that while they help reduce alcohol consumption, the vast majority of participants continue to drink at hazardous levels after receiving treatment; limited effectiveness may be related to poor uptake of the intervention by users (Kramer et al., 2009; Riper et al., 2009; Riper et al., 2008).

Only one study has investigated the effectiveness of a web-based CBT self-help intervention for cocaine abuse. Schaub, Sullivan, Haug, and Stark (2012) randomly assigned 196 participants to receive their intervention *Snow Control* or a psychoeducational control condition. Few group differences were noted in cocaine-related outcomes despite reports of using less cocaine, and attrition rates were extremely high (93–96 %) at 6-month follow-up. Thus, although the implementation of a web-based self-help treatment program is feasible, it is not very effective with cocaine abusers.

27.6 Self-Help Approaches for Gambling Disorder

Only a small minority of problem gamblers attend formal treatment programs, including especially few women and people with less severe gambling problems (Hodgins, Currie, & el-Guebaly, 2001). Thus self-help is a promising form of treatment for gambling problems.

Hodgins et al. (2001) examined the effectiveness of a self-help book, *Becoming a Winner: Defeating Problem Gambling* (Hodgins & Makarchuk, 1998). They compared outcomes among participants who received the self-help book alone or with an additional motivational interview. Compared to a wait-list group, those who received the additional motivational interview showed greater improvements on the number of days gambled, money spent, and money lost during gambling. Although the motivational interview group also initially showed greater improvements on gambling outcomes relative to those who only received the self-help book, group differences were not maintained over the long term.

LaBrie et al. (2012) investigated the effectiveness of a self-help book, *Your First Step to Change: Gambling* (Division on Addiction, 2009), which focused on overcoming ambivalence to change and provided information about how to quit and strategies to help with relapse prevention. Compared to the wait-list group, both pure and guided self-help groups showed significant improvements on gambling-related outcomes including fewer days gambling, fewer cognitive distortions, and greater use of coping strategies. Rates of abstinence from gambling were 85 %, 67 %, and 52 % for the guided self-help, pure self-help, and wait-list groups, respectively.

Studies have investigated the use of personalized feedback at improving gambling outcomes, with mixed results. A pilot study found that personalized feedback yielded reductions in the amount of money spent on gambling after controlling for demographic factors and preexisting gambling symptoms (Cunningham, Hodgins, Toneatto, Rai, & Cordingley, 2009). A larger RCT, however, found that providing personalized feedback *without* normative comparison data was more effective at reducing gambling days when compared to providing personalized feedback *with* normative comparison data; however, both the feedback and wait-list groups spent less money on gambling (Cunningham, Hodgins, Toneatto, & Murphy, 2012). Taken together, existing research suggests self-help for problem gambling can be effective at improving gambling outcomes in the short and medium term; however, approximately 50 % of people appear to recover without receiving any form of treatment (Cunningham et al., 2012; Hodgins et al., 2001; LaBrie et al., 2012).

Conclusions and Recommendations

As we have illustrated, a variety of self-help programs and support groups exist for ED, SUD, and GD. The evidence-based self-help programs described above may be useful to selected individuals with ED, carers of those with AN, and individuals with addictions. In addition, groups like DTR and SMART Recovery may provide a means for those with comorbid ED and SUD to obtain support in

the community, although their utility has not been assessed specifically for this population.

To summarize research findings, selected evidence-based self-help interventions, such as CBT for recurrent binge eating and BN, can alleviate symptoms; more research is needed to ascertain whether self-help needs to address ED symptoms explicitly to be most effective. Self-help may not be suitable for (or acceptable to) most individuals with AN. Twelve-step participation in addition to traditional SUD or GD treatment tends to improve outcomes. However, as stand-alone treatments, support groups for ED, SUD, GD, sex addition, and compulsive shopping remain untested. Controlled research is needed evaluating the utility of these low-intensity treatments, which may be particularly appropriate for those with less severe symptoms. Certain specific questions remain unanswered. For example, it is important to evaluate the utility of OA in reducing ED symptoms because rigid dietary restriction, exemplified by the highly structured food plans often adopted by new members of OA, can trigger binge eating (Wilson, 2010). In addition, more RCTs are needed to evaluate questions such as when a pure versus guided self-help intervention is merited, what treatment moderators exist, and what professional "guidance" should include (e.g., Stefano et al., 2006; Wilson & Zandberg, 2012).

With respect to comorbidity, the little research that is available on the effect of support groups for individuals with comorbid SUD plus anxiety or depressive disorder suggests little, if any, benefit. To our knowledge, no research has been published on the merits of self-help or support groups for individuals with comorbid ED and addictions, most prominently SUD. Based on the assumption that more support is better, psychotherapists report often referring their ED clients to 12-step groups such as OA for adjunctive treatment (von Ranson, Wallace, & Stevenson, 2013). Although this appears to be a reasonable step, there is nevertheless a need for research examining the efficacy of self-help treatments and support groups, as well as identifying when they may be most suitable or contraindicated, for individuals with comorbid ED and SUD.

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Positive and Negative Aspects of Exercise in the Treatment of Eating Disorders and Substance Use Disorders

28

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Abstract

Engaging in regular physical activity is a fundamental part of good physical health and increasingly appreciated as essential to psychological well-being and improved mental health. Strong evidence supports that engaging in exercise and physical activity can transiently improve mood and decrease anxiety, reduce mortality rates particularly in men, and improve response to treatment for nicotine addiction. While less understood, physical fitness and exercise can positively impact on treatment response for patients with eating disorders and substance use disorders. Of interest is that problematic exercise can be in and of itself a primary behavioral addiction or associated with other problems such as eating disorders or anxiety disorders and secondary to concerns of body image, self-esteem, health concerns, or social function. Treatment strategies for problematic exercise include addiction and compulsive/anxiety models that are not mutually exclusive and can help clinicians work effectively with patients. This chapter will briefly review the literature underscoring the positive impact of physical activity and fitness on medical and mental health, identify the characteristics of problematic exercise in the eating disorder population, discuss treatment approaches for problematic exercise, and finally review the potential positive effects of physical activity in the treatment of patients with eating disorders and/or substance use disorders.

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28.1 Introduction

Poor diet and poor physical fitness are second to tobacco use as the leading cause of death in America and may in fact overtake tobacco use as the leading cause of death in the future (Mokdad, Marks, Stroup, & Gerberding, 2004). Data indicates that less than 50 % of the American adult population's level of physical activity meets the Centers for Disease Control and Prevention 2008 physical fitness guidelines (Centers for Disease Control and Prevention, 2012). Taken together these data support the need for regular leisure time physical activity as an important initiative to improve population health. In this chapter we will (1) discuss the general health and mental health benefits of exercise and physical fitness, (2) review the clinical characteristics and treatment approaches for problematic exercise in eating disorder (ED) patients, (3) discuss the benefits and problem exercise in ED and substance use disorder (SUD) populations, and (4) review the use of exercise as part of treatment in ED and SUD treatment including the circumstance of co-occurring ED and SUD.

28.2 Health and Mental Health Benefits of Physical Activity

The benefits of exercise and physical fitness in the area of medical problems are compelling, as regular physical activity is associated with reduced mortality rates (Lindsted, Tonstad, & Kuzma, 1991). Furthermore, in a study of Norwegian men ages 40–59, it was demonstrated that those who were in the highest quartile of physical fitness based on exercise testing of cardiovascular fitness had the lowest mortality rates (Sandvik et al., 1993).

In adult US populations, the most common mental illnesses are depression and anxiety. Regular physical activity is associated with a significantly decreased occurrence of anxiety disorders (panic disorder, agoraphobia, social phobia, and specific phobia) and current major depression (Goodwin, 2003). While the causal links between physical activity and reduced rates of anxiety and affective disorder are not fully understood (De Moor, Boomsma, Stubbe, Willemsen, & de Geus, 2008), potential modulators that have been discussed include changes in central nervous system monoamines and endogenous opiates, increases in core body temperature affecting improved physical and psychological sense of well-being, and changes in psychological modulators affecting change in social functioning, self-esteem, task performance and mastery, and stress reduction.

In animal models, high-intensity physical activity can activate opiate and serotonergic systems as a way of modulating pain (Mazzardo-Martins et al., 2010). In humans, exercise is also associated with an increase in endogenous opiates that can modulate metabolic functioning (Milman, Leu, Shamoon, Vele, & Gabriely, 2012). The positive effect of exercise on mood may also occur as a result of changes in body temperature. For example, one study found elevated mood ratings in humans exposed to cold environments when they exercised to the point of increasing body temperature (Muller et al., 2011). The effect of exercise on psychological factors has also been studied in humans. In a review of 23 studies, exercise was shown to convincingly be associated with transient improvement in self-esteem in children and young adults (Ekeland, Heian, Hagen, Abbott, & Nordheim, 2004). A study of adolescents found that improved self-esteem was associated with increased participation in sports and had a protective effect in terms of reduced risk of depression and suicidal ideation (Babiss & Gangwisch, 2009). Group exercise activities have been shown to increase social function outside of the exercise activity (Nadasen, 2008). Finally, physical activity in adolescence also is predictive of improved emotional well-being in adulthood (Sacker & Cable, 2006).

The impact on psychological health may relate to the intensity and frequency of engagement in physical activity rather than the type of exercise. In non-ill populations, exercise promotes better mood, less anger and stress, and more vigorous exercise, as opposed the less intense exercise, has a greater impact on mood ratings (Dunn, Trivedi, Kampert, Clark, & Chambliss, 2005). In another study, it was demonstrated that exercising five times a week with an energy expenditure of 17 kcal/kg/week was an effective dose of exercise that resulted in reducing depression as compared to exercising three times a week with an energy expenditure of 7 kcal/kg/week. For non-aerobic or resistant exercise, effort that is 80 % of maximum load is more effective than less effort in terms of decreasing depression in adults age 60 or over (Singh et al., 2005). While both aerobic and anaerobic exercises decrease depressive symptoms, it does not appear that improved fitness (i.e., cardiovascular fitness) modulates the effect of increased physical activity on depression.

28.3 What Is Problematic Exercise?

A review of problematic exercise has been previously covered in this volume (See Chap. 7). ED patients frequently engage in problematic exercise (Brewerton, Stellefson, Hibbs, Hodges, & Cochrane, 1995) with one study finding that as many as 84 % of ED individuals have a lifetime prevalence of problematic exercise (Davis, Kennedy, Relevski, & Dionne, 1994). Problematic exercise is typically characterized by preoccupation with physical activity, a sense of loss of control over whether or how much to exercise, difficulty stopping physical activity even when there is a negative impact on health, engaging in exercise in secret, attempts to hide physical activity levels from others, and reliably experience dysphoria and anxiety if unable to exercise. For some patients with ED, excessive exercise

becomes "addictive" and is characterized by repeated engagement in behaviors that have a negative impact on biopsychosocial functioning and appear to be modulated by central brain reward centers. Alternatively, problematic exercise in ED has also been characterized as a secondarily reinforced, compulsive behavior aimed at avoiding situations that are feared and associated with anxiety and distress.

There is empirical evidence that psychological factors associated with physical activity—such as guilt when missing an exercise session and irritation at a session being interrupted—have a higher correlation with eating dysfunction than quantity or intensity (Adkins & Keel, 2005). Further, measures of physical activity quantity when applied to community samples have found a bimodal distribution of psychological disturbance. That is, one cluster of individuals with a high level of physical activity also endorsed significant ED and other psychopathological symptoms, while another cluster with equally intense physical activity were the most psychologically healthy participants studied on measures of eating, depression, anxiety, self-esteem, and family connectedness (Ackard, Brehm, & Steffen, 2002). In sum, a recent review of the literature concluded that there is "no empirical support for a model defining *excessive* exercise purely in terms of the quantitative dimension" (Meyer & Taranis, 2011, p. 170).

28.4 Problematic Exercise: Treatment Approaches

There are significant challenges in treating ED when intense physical activity is a frequent and recurrent behavior. Interventions should first identify important behavioral manifestations of problematic exercise, including the frequency, intensity, and duration of physical activity; medical conditions that directly result from problematic exercise including physical injuries of overuse and abuse; and also the psychosocial impact on time management, job performance, and obligation and functioning in personal relationships. Since DSM-5 (American Psychiatric Association, 2013) identifies excessive exercise as an ED symptom, anyone with an ED, regardless of weight status, can exhibit excessive exercise as a symptom akin to food restriction, binging, or purging. Indicated treatment goals would be a more or less permanent cessation of problematic exercise, as extinction is certainly the goal of other eating disordered behaviors such as restriction, binging, and purging. Throughout treatment and recovery stages, amount of exercise should then be monitored, and if an individual were to return to some level of problematic exercise, this would be considered a relapse.

As previously reviewed in this volume (see Chap. 7), research would support two different treatment models aimed at reducing problematic exercise in ED patients. The first treatment model sees problematic exercise as representing a non-substance addiction; it is also considered a primary behavior in which exercise and its direct impact on physical and psychological functioning lead to reward-based reinforcement of behavior. The second treatment model sees problematic exercise as a compulsive symptom aimed at avoidance or modification of some feared state or situation, which in the case of ED most likely relates to body image

(most typically thinness in the case of females and leanness or muscularity in the case of males) and food and weight issues. Within the models there is significant conceptual overlap, and the presentation structure of this chapter does not imply these models are in opposition. Furthermore, while each treatment model has a unique emphasis and may suggest a differing treatment approach for a patient with an ED, our experience has been that they can be used together in working with patients with problematic exercise.

28.5 Problematic Exercise as a "Primary Behavioral Addiction"

Identifying running as a potential behavioral addiction was first proposed by Morgan (1979a, 1979b). This model suggests that extreme physical activity is essentially identical to a chemical addiction and shares qualities used to define addiction such as increasing amounts to achieve the same effect (tolerance), avoidance of life problems, and irritability if physical activity is prevented (withdrawal). Several measures such as the Exercise Dependence Scale (Hausenblas & Downs, 2000) have been developed and used in research to establish the existence of a condition of "exercise dependence" in populations of athletes and ED patients. A recent review promoting this model argues that individuals with intense and frequent physical activity, along with a psychological attachment to these activities, should be classified as having an "exercise addiction," even in the absence of ED symptoms (Berczik et al., 2012).

The addiction model is conceptually logical and as described has some empirical support as reviewed previously in this volume (see Chap. 7). Exercise and physical activity have been identified as behavioral symptoms of an ED aimed at expending calories consumed, producing weight loss, or managing body image concerns (Davis et al., 1994). As a result, ED patients entering treatment have been generally discouraged from engaging in any or allowed to engage in only limited levels of physical activity or exercise, irrespective of the presence, absence, or severity of problematic exercise and even when not significantly underweight or malnourished. Those adhering to an addiction model may then help the patients to understand distress associated with "abstinence from exercise" such as irritability, anxiety, depression, and/or urges to exercise as withdrawal symptoms. However, irritability, anxiety, and depression are also very common psychological states for individuals with ED, so it is not entirely clear how these symptoms would be ascribed to suspension of physical activity. Recovery from problematic exercise in ED would not target abstinence from exercise and physical activity, rather abstinence from problematic exercise, and help the patients distinguish between urges for physical activity that are stress reducing and normative vs. cravings associated with a formerly dependent status. For full recovery from the ED problematic exercise, one would need to be abstinent for a significant period of time assuming all other ED symptoms were in remission as well.

While compelling, it is important to emphasize that exercise, as a behavioral addiction, is not universally accepted within the ED population. It has been our

clinical experience that some ED patients and families, particularly ANR patients and their family members, may have a negative response to describing physical activity as an addiction. For example, even with severe and disabling ED symptoms, they often are unaware of increasing amounts of physical activity, continue to view physical activity as important to long-term good health and happiness, and are able to garner a great deal of evidence supporting this view. Characteristics of individuals with AN typically include high emotional control and perfectionism, and our group has observed that, right or wrong, these individuals often have very negative views about substance abuse or other behaviors that they consider to be socially deviant. Conversely, for non-ANR patients who engage in binge and/or purge behavior, the addiction model may be more accepted as there are increased rates of SUD (Root et al., 2010). Furthermore, it has been our clinical experience that ED patients who engage in these types of behaviors typically report a positive response to participation in support groups for substance and behavioral addictions.

28.6 Problematic Exercise as "Compulsive/Anxiety-Based Behaviors"

The compulsive/anxiety model of problematic exercise emphasizes the similarity between physical activity and other compulsive behaviors associated with anxiety disorders. While the term "compulsive" is sometimes also applied to chemical substance-seeking behavior, the current usage is intended to emphasize the analogy to ritualized compulsions such as hand washing in obsessive-compulsive disorder (OCD). Individuals with OCD have unwanted thoughts and fears (obsessions), which create anxiety, and they engage in related physical or mental rituals (compulsions).

The anxiety/compulsive model would suggest that *habituation* to anxiety-producing cues is the construct to measure treatment progress. Specifically for ED patients, this is done by creating a hierarchy of situations that create an anxiety response for which the patient would typically engage in a physical activity to neutralize their fears (Long & Hollin, 1995). To create the hierarchy, situations are rated (highest to lowest) by the patient according to the level of self-reported anxiety; then gradually and repeatedly they are exposed to the situation and not permitted to engage in the compulsive response. Any situation that increases the urge to engage in exercising to manage distress can be listed in the hierarchy (Hubbard, Gray, & Parker, 1998). For example, hierarchy items could include eating 100 % of prescribed meals and snacks with other patients, staff, and/or family; supervised meals; locked bathrooms and/or supervised bathroom privileges; and participation in emotionally distressing individual, group, or family sessions. Patients are encouraged to engage in these activities (i.e., exposure), but prevented (i.e., response prevention) from engaging in exercise to manage their escalating urges or anxiety.

Initially, patients complete exposure assignments determined to be challenging but manageable, and as they habituate to these stimuli (experience no or very little anxiety), they move on to more challenging stimuli. Progress is measured by percentage of the hierarchy that has been retired (i.e., number of anxiety-producing stimuli to which the patient has habituated). Urges for a compulsive response are tracked in terms of "submits" (submitted to the urge) and "resists" (had an urge, but resisted). In addition to exposure with response prevention (ERP) for anxiety habituation, efficacious treatment for anxiety disorders includes challenging thoughts or beliefs that maintain anxiety. Treatment progress can be measured in terms of patients' ability to challenge their own anxiety-producing thoughts, such as "If I miss my exercise routine today, I will be fat." In sum, ERP is a learning paradigm which offers patients a structured method for approaching moderate and healthy physical activity and does not identify physical activity as a fundamentally unhealthy behavior.

Regardless of whether the characteristics of problematic exercise for the individual best fit the addiction model, compulsive/anxiety model, or in some cases characteristics of both models, the challenge remains that patients must appreciate the context in which their physical activity is part of their ED, commit to the rationale of the model, and be motivated to move from problematic to healthy exercise and physical activity.

28.7 Starvation and Problematic Exercise

In patients with AN, it is important to recognize that physical activity and fear of food consumption may have a significant non-volitional aspect when patients are severely underweight. Additionally, food restriction in a malnourished state may result in higher drive for activity. Because this cycle is so demonstrably dangerous, the model would suggest that the primary immediate treatment goal for anyone under a given weight threshold is suspension of at least all planned physical activity until weight is restored beyond the threshold. It would further suggest that this combined state of low weight, food restriction, and hyperactivity is a physical symptom of AN, akin to lanugo or bradycardia.

If animal models hold in humans, a body weight lower than 25 % of expected weight would generate a reciprocal cycle of food restriction and activity, regardless of the patients' cognitive intent. Data from animal models suggest that males would be at risk of reaching this threshold faster than females, although how much faster is unknown in humans. Additionally, animal models suggest that patients in this state would benefit from having a higher ambient body temperature; however, in a controlled study, increasing body temperature in AN patients was not associated with improved treatment response (Birmingham, Gutierrez, Jonat, & Beumont, 2004).

The lack of cognitive control over physical activity suggests a nonpunitive and noncognitive approach to behaviors such as standing, restless movement, or planned activity. Interventions such as "thought challenging" the purpose of

activity, processing emotional need for activity, or negative contingencies for engaging in activity when the patient is significantly underweight would be unnecessary and unhelpful and may imply to patients a level of control that they simply do not have while in this state. As we have noted, research has demonstrated that many individuals with ED do return to intense physical activity, often proceeding to relapse into other ED symptoms, even after weight status is restored (Strober, Freeman, & Morrell, 1997). This indicates that intense physical activity continues to be extremely compelling for some individuals even when at a normal weight.

28.8 Steroids, Body Image, and Drugs of Abuse

Although not typically associated with problematic exercise or, for that matter, ED, the increasing focus on muscle-enhancing behavior and use and abuse of anabolic-androgenic steroids (AAS) and related compounds has many behavioral and treatment issues similar to both SUD and ED (See Chap. 20). Furthermore, for treatment of AAS and other muscle-enhancing drugs and behaviors, limited research would suggest incorporating treatment approaches common to both ED and SUD (Corcoran & Longo, 1992).

Numerous studies suggest AAS abuse is linked to SUD. In a study of over 400 elite athletes, 90 % held the belief that banned substances and methods aimed at performance enhancement had the desired effect (Alaranta et al., 2006). Furthermore, these researchers found the athletes involved in speed and power sports (21 %) and team sports (14 %) were offered banned substances (most commonly stimulants and AAS). In a study of almost 3,000 urban middle school students, 35 % used protein powders or shakes and 6 % reported steroid abuse (Eisenberg, Wall, & Neumark-Sztainer, 2012). Behavioral manifestations of AAS use include aggression, violence, and mania, and long-term AAS use may lead to the development of dependence and withdrawal (Trenton & Currier, 2005). Human growth hormone (HGH) was reportedly used in 12 % of young male weight lifters. The illegal use of HGH has become a common form of substance abuse and linked to AAS dependence and abuse of other nonperformance-enhancing substances, such as opiates, cocaine, and ecstasy (Brennan, Kanayama, Hudson, & Pope, 2011). In a survey of dependent vs. nondependent AAS users, AAS dependent users had higher rates of heroin use as well as increased rates of anxiety and depression (Ip et al., 2012).

Distress and anxiety concerning body image also are related to the problem of AAS abuse. In a study of Polish adolescents, risk factors for using AAS for males most often included concerns with physical appearance (Rachon, Pokrywka, & Suchecka-Rachon, 2006). In male weight lifters, AAS use was associated with conduct disorder and body image concerns (Pope, Kanayama, & Hudson, 2012). When comparing males with ED to males who recently stopped using AAS, groups were similar for the occurrence of serious psychiatric illness; however, the ED group had more severe concerns with self-image (Bjork, Skargberg, & Engstrom, 2013).

28.9 Benefits and Problems with Exercise in ED

Eating disorders, as described in DSM-5 (American Psychiatric Association, 2013), are characterized by persistent disturbances of eating or eating-related behaviors that result in significant impairment in physical health or psychosocial functioning. In addition, the diagnostic categories of AN and BN, which are also characterized in part by preoccupations with food intake, body weight, and body shape are the only psychiatric diagnosis in the DSM-5 for which physical activity or exercise is included as a contributing and problematic symptom. Changes in ED classification in DSM-5 have resulted in a new category, Other Specified Feeding and Eating Disorders (OSFED), that includes subclinical AN, BN, and binge eating disorder (BED), as well as purging disorder and night eating syndrome. Research into problematic exercise in this new diagnostic group is not available; however, patients in this group can be as severely compromised by problematic exercise as patients that meet full diagnostic criteria for an ED.

As mentioned previously, ED patients for whom exercise is a core feature of their illness present treatment providers with significant challenges. Physical fitness and participation in exercise has been shown to improve symptom reduction and treatment response in ED patients. One study found that past physical activity or commitment to athletics in AN patients supports positive self-esteem, even after becoming ill (Bewell-Weiss & Carter, 2010). In the case of binge eating behavior in BN and BED and the treatment goals of reduced weight in BED, regular fitness and exercise is much more likely to be considered an essential as a part of their recovery.

In a small study, patients with BN were randomized to exercise, CBT, nutrition, or wait list. The exercise group was shown to have more improvement in body image and bulimic symptoms than the CBT group (Sundgot-Borgen, Rosenvinge, Bahr, & Schneider, 2002). Even more compelling are studies showing the benefits of exercise and fitness in BED. Physical fitness and nutritional counseling when added to CBT have been shown to reduce depression and anxiety and facilitate weight loss in BED (Fossati et al., 2004). In another study in which BED patients received CBT with and without exercise, the group with the highest abstinence rates at the end of treatment was the combined CBT and exercise group (61 %) compared to the non-exerciser group (30 %) (Pendleton, Goodrick, Poston, Reeves, & Foreyt, 2002).

28.10 Benefits and Problems with Exercise in SUD

There is strong evidence that exercise can be an effective adjunctive treatment for nicotine addiction (Zschucke, Heinz, & Strohle, 2012). While the efficacy of physical activity as an intervention for alcohol and drug treatment is less compelling (Dakwar et al., 2012), data suggests that improved fitness can reduce the risk for developing an SUD and can impact positively on substance use patterns (Collingwood, Sunderlin, Reynolds, & Kohl, 2000). One study found that regular

physical activity has a protective effect on substance abuse in youth (Collingwood et al., 2000; Ströhle et al., 2007). Elite student athletes also had significantly lower levels of alcohol and cannabis use than controls, although some differences were found based on different sports (Peretti-Watel et al., 2003). The impact of exercise and fitness has been shown to reduce craving for patients treated in an inpatient alcohol treatment setting (Ermalinski, Hanson, Lubin, Thornby, & Nahormek, 1997) and also improve abstinence rates in an outpatient sample undergoing substance use treatment (Sinyor, Schwartz, Peronnet, Brisson, & Serganian, 1983; Weinstock, Barry, & Petry, 2008). Participation in running was also shown to reduce alcohol consumption while on a wait list for treatment in problem drinkers (Murphy, Pagano, & Marlatt, 1986). Physical activity has long been promoted as a treatment intervention to facilitate recovery from substance abuse in prison (Barthwell et al., 1995) and psychiatric settings (Stathopoulou, Powers, Berry, Smits, & Otto, 2006).

Unfortunately, participation in organized athletics, as opposed to physical activity, may increase the risk of substance use. In adolescence, while athletic team participation is associated with higher alcohol use, the combination of team and other physical activities, or physical activities outside of team sports, is associated with lower levels of alcohol, cigarette, and cannabis use (Terry-McElrath, O'Malley, & Johnston, 2013). In adolescent athletes, participation in team sports may also increase the risk of smokeless tobacco, alcohol, and performance-enhancing drugs such as steroids (Terry-McElrath, O'Malley, & Johnston, 2011); however, this association may be moderated by team education initiatives (Goldberg et al., 2000).

The positive effect of exercise on symptoms in ED and alcohol use disorders (AUD) is not surprising, as strong links are present between these two disorders (Dansky, Brewerton, & Kilpatrick, 2000). Approximately 40 % of individuals with BN are diagnosed with a co-occurring SUD (Wiederman & Pryor, 1996), which has been linked to impulsivity characteristics. Individuals with AN (both ANR and ANBP) are likely to engage in excessive exercise (Shroff et al., 2006) and about one in four AN individuals have SUD (Hudson, Hiripi, Pope, & Kessler, 2007). Of interest is that subgroups of individuals with AN differ in terms of risk for SUD. In one study, 14 % of ANR individuals were found to have AUD as compared to 35 % of individuals with ANBP (Root et al., 2010). The difference in rates of SUD in AN subgroups suggests that perfectionism, high harm avoidance, and low novelty seeking may represent protective factors in ANR (Holderness, Brooks-Gunn, & Warren, 1994).

28.11 The Use of Exercise in the Treatment of ED and SUD

Both SUD and ED are characterized by repetitive compulsive behaviors that become increasingly destructive and are reinforced through neurobiological mechanisms. For both disorders, exercise can play an important role in treatment, recovery, and relapse prevention. One study found evidence that athletic

participation had a positive impact on substance use and disordered eating in youth (Elliot et al., 2004).

As previously reviewed, the benefits of physical activity for ED and SUD are increasingly apparent. Thus, limiting physical activity during treatment for patients with ED and SUD may negatively impact on treatment effectiveness. For example, it is not uncommon for ED treatment providers to allow little or no physical activity, based on the belief that any activity would interfere with weight gain or would reinforce a pathological symptom of the ED. A recent review of the treatment literature found significant variance among ED treatment providers in how they manage physical activity and few if any written guidelines (Zunker, Mitchell, & Wonderlich, 2011). While variance in treatment approaches is likely the rule for most psychological disorders, it does create particular difficulties for patients with ED who may move between facilities and levels of care. For example, a patient may see an outpatient provider who conceptualizes physical activity as an "addiction" and may then be at a residential treatment center that utilizes physical activity as part of a treatment protocol. At minimum, it is important for treatment team members to have a consistent conceptualization and treatment approach to exercise, to avoid having patients and family members receive mixed or conflicting messages. We have found that identifying specific members or the treatment team, often with input from certified fitness trainers and medical staff, that are comfortable recommending individualized non-excessive exercise as part of the treatment can noticeably increase a patient's physical and emotional well-being in treatment. Still, the primary challenge with this approach has been discussed extensively in the literature and here; how much exercise is "excessive" in the recovering ED patient, and how should it be measured? The US Centers for Disease Control and Prevention (CDC) recommends all adults engage in 150 min of "moderate" cardiovascular exercise or 75 min of "intense" cardiovascular exercise as well as strength training twice weekly (Centers for Disease Control, 2013).

It has not been verified whether these guidelines are appropriate for ED individuals who are moderately or mildly underweight and those who are normal weight but have been treated for AN or excessive exercise in the past. To our knowledge, there have been no controlled studies comparing short- or long-term outcomes of specific treatment approaches to physical activity in ED; however, we do know that caution must be exercised as to prescribing physical activity for patients with AN, as the presence of behaviors indicative of problematic exercise may negatively impact on the course of illness (Strober et al., 1997). However, a few studies have demonstrated that moderate physical activity paradoxically enhances weight gain in underweight patients with AN (Thien, Thomas, Markin, & Birmingham, 2000). One controlled study reported that the physically active group gained one-third *more* than the inactive control group (Calogero & Pedrotty, 2004). Mechanisms are not fully explicated; however, it is possible that moderate physical activity can facilitate weight gain by improved emotional well-being, by increased appetite, and by reducing distress around body image and appearance and that particularly for AN patients with co-occurring SUD participation in physical activity and exercise may improve treatment response.

Conclusions

Evidence strongly supports the positive impact physical activity has on reducing the risk of developing a serious mental illness, enhancing treatment response, and reducing relapse rates. Improved education on the importance of physical fitness as an adjunct to primary treatment interventions such as pharmacotherapy and psychotherapy is needed. This is particularly true for illnesses such as SUD and ED where positive behavioral changes and the use of healthy coping skills are a primary focus of treatment and recovery. Identifying effective strategies for integrating physical activity at an intensity and frequency that is effective in psychiatrically ill populations in general and within the ED field needs further study. In particular, designing studies that follow ED patients with problematic exercise through multiple levels of care that evaluate the effectiveness of treatment approaches (i.e., the addiction model approach vs. the compulsive/anxiety model) could significantly assist providers in determining to what extent physical activity during treatment including weight restoration can facilitate treatment response for patients with ED and SUD.

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Alternative and Complementary Therapies in the Treatment of Eating Disorders, Addictions, and Substance Use Disorders

29

Sloane Madden, Sarah Fogarty, and Caroline Smith

Abstract

Research over the past decade into eating disorders and substance use disorders has been considerable. Despite this, response to treatment for many remains poor. New adjunctive therapies that improve the effectiveness of existing treatments and lead to improved mental health outcomes for these patient groups are highly desirable. Recent research into the role of complementary and alternative medicine in eating disorders and substance use disorders has responded not only to the need to improve treatment outcomes but also to the common usage of complimentary therapies by individuals with these disorders. While still in its early stages, research into different types of complimentary therapies including yoga, acupuncture, therapeutic massage, hypnosis, herbal medicine, light therapy, spiritual healing, and art therapy has shown promise both in addressing not only comorbid anxiety and depression but also core disorder symptoms. It is therefore critical that these therapies are rigorously evaluated so we utilize any potential benefits from these treatments and respond to our patients' considerable interest in and desire to utilize complementary and alternative medicine in their recovery.

Keywords

Acupuncture • Alternative medicine • Complimentary medicine • Eating disorders • Herbal medicine • Hypnosis • Spiritual healing • Substance use disorders • Therapeutic massage • Yoga

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29.1 Introduction

Complementary and alternative medicines (CAM) refer to a broad range of health practices (National Centre for Complementary and Alternative Medicine, 2012), though there is no widely accepted definition of CAM. Nomenclature varies with terms including complementary (health and medical practices not recognized as part of mainstream Western medicine), alternative (complementary medicine used in place of mainstream Western medicine), traditional (indigenous medicines and practices), and integrative medicine (complementary and mainstream medicines used together) (NICM, 2011). An operational definition of CAM proposed by Wieland and colleagues defines CAM based on (1) therapies that rely upon non-allopathic models of health, (2) exclusion from standard treatment within the dominant medical system, and (3) self-care or care delivered by alternative practitioners (Wieland, Manheimer, & Berman, 2011).

There are a diverse range of CAM modalities used to promote, treat, and maintain health and well-being. These modalities are categorized into four main domains and medical systems, developed by the US National Center for Complementary and Alternative Medicine:

1. **Mind-body medicine** uses a variety of techniques designed to enhance the mind's capacity to affect bodily function and symptoms. These include meditation and therapies that use creative outlets such as art, music, or dance.
2. **Biologically based practices** in CAM use substances found in nature, such as herbs, foods, and vitamins.
3. **Manipulative and body-based practice** is based on tactile therapies and structured exercise regimes. Manipulation may be performed as a part of other therapies or whole medical systems, including chiropractic medicine, osteopathic medicine, massage, and naturopathic medicine. Structured exercise regimes include yoga, tai chi, and specific exercise programs.
4. **Energy medicine** involves the use of energy fields. There are two types of energy medicine: biofield therapies and bioelectromagnetic-based therapies. Biofield therapies are intended to affect energy fields that purportedly surround and penetrate the human body. Some forms of energy therapy purportedly manipulate biofields by applying pressure and/or manipulating the body by placing the hands in, or through, these fields. Examples include Qigong, a practice that combines movement, meditation, and controlled breathing; reiki, a therapy in which practitioners seek to transmit a universal energy to a person, either from a distance or by placing their hands on or near that person; therapeutic touch, which involves practitioners passing their hands over another person's body with the intent to use their own perceived healing energy to promote health; and bioelectromagnetic-based therapies which involve the unconventional use of electromagnetic fields, such as pulsed or magnetic fields.
5. **Whole medical systems** are built upon complete systems of theory and practice. These systems have evolved apart from and earlier than the conventional medical approach. Examples of whole medical systems include homeopathic medicine, naturopathic medicine, Traditional Chinese Medicine (TCM), and

Ayurvedic medicine. Meditation is often included within the CAM mind-body domain; however, meditation frequently contains an element of cognitive behavioral therapy (CBT) and mindfulness both of which are addressed in Chaps. 24 and 25.

29.2 Eating Disorders, Substance Use Disorders, and Complementary Medicines and Therapies

Research over the last decade into ED has been considerable and while evidence-based treatments have begun to emerge, treatment outcomes for many remain poor. Similarly, while there has been a growing understanding of the neurobiology of SUD, this is yet to translate into more effective interventions (Behere, Muralidharan, & Benegal, 2009). New adjunctive therapies that improve the effectiveness of these treatments and lead to improved mental health outcomes for these patient groups are highly desirable. Research into the role of CAM in ED and SUD has responded not only to the need to improve treatment outcomes but also to the common usage of CAM therapies by sufferers. Research has been most pronounced in acupuncture and herbal medicine, though in recent years research has also looked at yoga, massage, and spiritual healing (Behere et al., 2009).

29.2.1 Yoga

Yoga is a practice that incorporates physical postures, controlled breathing, deep relaxation, and meditation. It has its origins as an ancient Indian practice (Balasubramaniam, Telles, & Doraiswamy, 2013) and has been used to manage stress, sleep, anxiety, and depression (Carei, Fyfe-Johnson, Breuner, & Brown, 2010). It is believed that yoga creates changes in the neurophysiology of the body through the combination of self-control, self-regulation, exercise, controlled breathing, controlled senses, concentration, meditation, and self-realization (Balasubramaniam et al., 2013; Douglass, 2009). These changes are proposed to affect the hypothalamic-pituitary axis (decreasing cortisol), neurotransmitters (increasing GABA, glutamate, serotonin, and acetylcholine and decreasing dopamine and norepinephrine), neurotropic factors (increase BDNF), and the immune system (Balasubramaniam et al., 2013).

29.2.1.1 Yoga and Eating Disorders

A mixed method study including a RCT and a qualitative narrative-based study was conducted by McIver, McGartland, and O'Halloran (2009) and McIver, O'Halloran, and McGartland (2009). Ninety overweight or obese women with BED were randomized to either a 12-week yoga program or a wait-list control. Fifty women (25 in each group) completed the study. The yoga sessions were 60 min in duration. The yoga practice encouraged physical awareness through

movement (*asana*), breath awareness (*pranayama*), and concentrative meditation (*dharana* and *dhyana*) including an "eating mindfully" meditation.

A benefit was found for yoga with a significant reduction in binge eating and a small but significant reduction in BMI and hip and waist measurements. Binge eating scores improved by approximately 50 % compared to the wait-list control. These improvements were maintained at the 3-month follow-up.

The RCT included a nested qualitative study (McIver, McGartland, et al., 2009; McIver, O'Halloran, et al., 2009). Yoga participants were invited to complete a personal self-reflective journal with no specific instruction given on what to record; 20 journals were completed. The journals were analyzed using an existential-phenomenological inquiry. The study found participants experienced a healthy reconnection to food, the development of physical self-empowerment, a reduction in the quantity of food consumed, decreased eating speed, and improved food choices. Together these studies suggest a benefit from the intervention; however, there was no attempt to control for the effects of time and attention given to the treatment group. Subjects were not blind to their group allocation and this may have impacted on outcomes. The yoga practice involved many different components including an "eating mindfully" meditation which focused participants' minds on their eating habits, speed of eating, amount consumed, and eating without distraction. The focus on eating and food may have also contributed to the beneficial effects of the yoga program, and it is unclear which aspects of the intervention may have been most beneficial.

The evidence for yoga in other ED is informed by one clinical trial and an observational study. Carei et al. (2010) undertook a pilot RCT to examine the effect of yoga on ED outcomes at an inpatient facility in the USA. Fifty females and four males aged 11–21 years with diagnoses of AN, BN, or EDNOS were recruited to the study. Subjects were randomized to yoga plus treatment as usual (TAU) or to a wait-list group. The yoga group received a one-on-one, 60-min yoga session twice a week for 8 consecutive weeks, with follow-up at 12 weeks. The wait-list group received TAU during the study and then received the yoga intervention following completion of the study. Both groups experienced changes over time. Yoga participants demonstrated greater decreases in ED symptoms at 12 weeks compared to the control and significantly reduced food preoccupation immediately after the yoga session ($p = 0.01$). Both groups maintained BMI and experienced decreased anxiety and depression.

The effect of yoga on disordered eating is informed by one clinical trial (Mitchell, Mazzeo, Rausch, & Cooke, 2007). This community-based study in the USA recruited female undergraduate volunteers who were dissatisfied with their bodies. The RCT evaluated the effectiveness of two new interventions: yoga and a cognitive dissonance group designed to reduce the symptomology of disordered eating and the risk factors for ED. Ninety female undergraduate students were randomly assigned to either a cognitive dissonance-based intervention ($n = 30$), a yoga and meditation intervention ($n = 30$), or a control group (details not stated) ($n = 30$). The yoga and dissonance groups met once a week for 45 min over a 6-week period. The dissonance therapy specifically addressed the thin ideal and its

negative consequences, "fattism," self-objectification, and the oppression of women. The yoga intervention did not specifically address body satisfaction or mind-body awareness pertaining to disordered eating. The control group received no treatment. At baseline 73 % of participants had no or mild binge eating, 65 % feared gaining weight or getting fat, 30 % reported a loss of control when binge eating, and only a small number reported purging, laxative use, or excessive exercise. The cognitive dissonance group demonstrated decreased disordered eating symptomology, drive for thinness, body dissatisfaction, alexithymia, and trait anxiety compared with yoga and the control group (Mitchell et al., 2007). The dose of the yoga intervention has been identified as a limitation of this study with the yoga described as lacking both intensity and frequency.

29.2.1.2 Yoga and Substance Use Disorders

Yoga and meditation has been proposed as a treatment for SUD because of their potential to reduce stress, improve mood and anxiety, and improve self-awareness and self-esteem and their ability to induce a higher state of consciousness, thereby removing the need for individuals to experience a drug-induced high. Despite this there have been only a handful of small cohort studies and RCTs in this area (Khalsa, Khalsa, Khalsa, & Khalsa, 2008).

In one small RCT alcohol-dependent subjects showed a statistically greater reduction in their substance dependence following an 8-week yoga intervention compared to physical exercise control group, while in a second RCT clients in a methadone maintenance outpatient clinic showed similar physical, psychological, and social benefits following a 6-month yoga intervention compared with group psychotherapy (Khalsa et al., 2008). While these findings show potential promise for yoga, the small number of subjects in these trials and the small number of trials in this field limit any recommendations for the use of Yoga in SUD.

29.2.2 Acupuncture

Acupuncture is characterized by the insertion of needles into specific body points to impact the flow of qi (vital energy), a therapeutic relationship, individualized treatment, and active engagement of patients in self-care or management (National Centre for Complementary and Alternative Medicine, 2012). The acupuncture consultation is a complex therapeutic intervention involving not just needling but a more holistic experience (Shi, Yang, Liu, & Wang, 2012). There is emerging evidence identifying an adjunctive role for the use of acupuncture to treat some mental health disorders (Pilkington, Kirkwood, Rampes, Cummings, & Richardson, 2007; Smith, Hay, & MacPherson, 2010); however, research evaluating the effect of acupuncture as an adjunctive treatment for ED is sparse. Acupuncture has been identified as being used in ED for the treatment of anxiety and quality of life (Clarke, 2009; Fogarty, Harris, Zaslawski, McAinch, & Stojanovska, 2010), emotional support, stress, menstrual irregularities, depression, and digestive complaints (Clarke, 2009).

Conversely there has been extensive research into the role of acupuncture in SUD in particular, alcohol, cocaine, and opiate abuse and dependence. Some of the largest treatment trials in SUD treatment have been carried out using acupuncture interventions. Disappointingly the results from these studies have been largely negative (Behere et al., 2009; Samuels, Gropp, Singer, & Oberbaum, 2008).

29.2.2.1 Acupuncture and Eating Disorders

Fogarty et al. (2013) conducted a mixed methods pilot RCT and a qualitative study with a clinical inpatient population with AN. Twenty-five women and one man were randomized to acupuncture plus TAU or acupressure plus TAU. Treatment was administered twice a week for the first 3 weeks followed by weekly treatment for 3 weeks. At the end of the intervention, differences were found for one item on a subscale of the Eating Disorder Examination Questionnaire (EDE-Q) outcome. Participants in the control group demonstrated reduced eating concerns on the EDE ($p < 0.05$). Over time, both groups reported changes for EDI-3 Bulimia and total quality of life and in the physical/cognitive domain of the Eating Disorder Quality of Life Scale (EDQoL). Within-group comparisons found significant improvement in symptomology for the acupuncture group in relation to EDQoL psychological, EDE shape, and EDE weight. A similar improvement was found for the control group with improvements over time for the EDE including total score, restraint, and eating concern (Fogarty et al., 2013).

The qualitative study involved patient interviews following their completion of the trial (Fogarty et al., 2013). Both groups reported positive experiences from treatment and the interventions were viewed as a welcome supplementary activity from usual care. An improvement in mood and feeling relaxed and calm were the most common reported benefits for those receiving acupressure. Those receiving acupuncture reported a broader range of outcomes including an improvement in mood, feeling relaxed and calm, sleeping better, less "churning of thoughts," less anxiety and stress, and being more communicative.

Fogarty et al. (2010) conducted an open-label randomized crossover design pilot study to examine the role of acupuncture in the treatment of ED. The two interventions were TAU and TAU plus acupuncture with a 2-week washout period in between phases. Nine women enrolled in the study and received ten 60-min sessions of acupuncture. Significant improvements were found for state anxiety using the State Trait Anxiety Inventory (STAI) ($p = 0.017$), an improvement in quality of life as measured by the EDQoL ($p = 0.007$), and a significant change in the Physical/Cognitive domain of the EDQoL ($p = 0.0009$). There was a trend for an improvement in the EDI-3 (Garner, 2004) subscale of Perfectionism ($p = 0.0597$) and for the Psychological domain of the EDQoL ($p = 0.0557$).

Views on the role of acupuncture on ED were reported by acupuncturists and ED patients in a treatment study from the UK (Clarke, 2009). Acupuncturists reported their belief that acupuncture was helpful, while patients reported feeling better; had decreased signs and symptoms associated with their ED and less menstrual irregularities, stress, and depression; and had improved self-esteem. Interviews with patients identified that acupuncture was not sought to specifically help their

ED but to assist with co-occurring symptoms. Patients reported feeling relaxed and generally better following treatment. Acupuncture was not regarded as a cure for ED by either patients or practitioners, but viewed as a useful adjunct and as a resource to manage their life and their ED (Clarke, 2009).

29.2.2.2 Acupuncture and Substance Use Disorders

A recent systematic review identified 11 RCTs, involving 1,110 participants, which had looked at the role of acupuncture in alcohol dependence. In this review acupuncture was compared to conventional therapies, sham acupuncture, nonspecific acupuncture, symptom-based acupuncture, and aromatherapy (1 RCT). The type of acupuncture used included auricular acupuncture and whole body acupuncture though the small number of studies did not allow for the type of acupuncture to be separately reviewed. While some individual trials showed significant findings in favor of acupuncture, a pooling of the data did not show that acupuncture was superior to either sham acupuncture or conventional therapies in reducing alcohol cravings and withdrawal symptoms or in treatment completion (Cho & Whang, 2009).

The largest RCT involved 503 participants with alcohol dependence, in a residential treatment setting. Participants were randomized to receive auricular acupuncture and conventional therapies, conventional therapies alone, nonspecific acupuncture and conventional therapies, or symptom-based acupuncture and conventional therapies. Acupuncture was provided on a daily basis for 6 days of each week for a total of 3 weeks. Assessments were taken at the beginning and end of treatment and at 3-, 6-, and 12-month follow-up. While all study participants experienced improvement across a range of measures, there were no significant differences between groups in terms of alcohol use, alcohol cravings, mood symptoms, and measures of treatment completion or treatment preference. It was the authors' conclusion that the addition of acupuncture did not offer any advantages to conventional therapy alone in treating alcohol dependence (Bullock et al., 2002).

The use of auricular acupuncture as a treatment for cocaine dependence is common with over 400 centers in Europe and the USA identified as offering the treatment (Gates, Smith, & Foxcroft, 2006). A recent systemic review of the use of acupuncture in cocaine dependence identified a total of seven RCTs involving a total of 1,433 subjects. In these studies acupuncture was compared with either sham acupuncture or no acupuncture. Outcome measures included cocaine use, measured either biochemically or by self-report, cocaine craving, severity of dependence, and treatment dropout. When data was pooled no significant differences were found between auricular acupuncture and sham acupuncture or no acupuncture in treatment outcomes. While this finding would suggest no benefit from auricular acupuncture in the treatment of cocaine dependence, major methodological flaws in all studies including high dropout rates, lack of long-term follow-up in many studies, and a lack of consistency in treatment and placebo interventions make conclusive findings about the role of acupuncture impossible (Gates et al., 2006).

Acupuncture has a long history in the management of opiate withdrawal and dependence. Interest in the role of acupuncture in opiate dependence arose in the context of early research examining the role of acupuncture and analgesia. The first studies of acupuncture in opiate addicted patients were carried out in Hong Kong and involved the non-blinded application of acupuncture to assist in opiate withdrawal. Outcomes in the original eight patients treated were limited with only five of the eight reporting any benefits from treatment (Wen & Cheung, 1973).

Since this initial trial there have been a number of randomized controlled trials primarily looking at the role of acupuncture in opiate withdrawal. In a recent systematic review, six RCTs of auricular acupuncture were identified. While several studies noted reduced withdrawal symptoms in those treated with auricular acupuncture, the largest and most methodologically rigorous studies showed no benefit of acupuncture compared to sham acupuncture or relaxation techniques. It was the authors' conclusion that current evidence does not support the role of acupuncture in the treatment of opiate withdrawal (Jordan, 2006).

29.2.3 Therapeutic Massage

Therapeutic massage is the "therapeutic friction, stroking or kneading of the body" for clinical benefit or the "manipulation of soft tissue by trained therapists for therapeutic purposes" (Field, 1998). Therapeutic massage has been used to reduce arousal associated with substance use withdrawal, and anxiety and depression for those with eating and SUD (Field et al., 1998; Hart et al., 2001). Greater parasympathetic arousal (reduction in cortisol stress hormones and catecholamines and an increase in vagal tone) is one mechanism proposed to explain the effects from massage (Hart et al., 2001). Massage has also been associated with an increase in serotonin and dopamine, which may account for improved mood (Hart et al., 2001). Included under the category of massage is Mindful Awareness in Body-Oriented Therapy (MABT).

MABT is a combination of manual (massage) and mind-body therapy (body awareness techniques and body awareness skills and mindful practice). This combination of manual hands-on approach and an individual instead of group delivery makes it different from other mindfulness approaches (Price, Wells, Donovan, & Rue, 2012). MABT was developed by Cynthia Price with the primary components addressing awareness, interoception, and regulation which may be associated with behavioral control and brain function abnormalities seen in those with addictions (Price et al., 2012). MABT may regulate the mechanism of interoception, which is disrupted by drug dependence (Price et al., 2012). MABT was used as an adjunct therapy to help prevent SUD relapse and for psychological and physical distress including ED symptoms and stress (Price et al., 2012).

29.2.3.1 Therapeutic Massage and Eating Disorders

One small parallel RCT has investigated the effect of massage with managing symptoms of BN (Field et al., 1998). Twenty-four female inpatients with BN

were randomized to standard care or massage therapy plus standard care. Study participants received two massages (30-min duration) a week for 5 weeks. The massage treatment followed a protocol involving massage treatment strokes (traction, rocking, stretching, smooth long stroke, and squeezing and friction), treatment duration (15 min prone and supine), and areas of the body to be worked on (head, face, neck, shoulders, arms, legs, and back). Participants received their massage fully clothed. Findings reported a benefit from massage with patients significantly less depressed and anxious following each massage from the initiation to the end of the study ($p = 0.001$). Significant decreases in Eating Disorder Inventory (EDI) scores were found for the massage group including drive for thinness, bulimia, body dissatisfaction, ineffectiveness, perfectionism, interpersonal distrust, interoceptive awareness, and maturity fears ($p = 0.001$) (Field et al., 1998).

One small RCT has evaluated the role of massage on outcomes for individuals with AN (Hart et al., 2001). Nineteen women with AN were recruited from both an inpatient and outpatient setting in the USA. Participants were randomized to standard care alone or standard care plus massage. Participants received two massages (30-min duration) a week for 5 weeks. The massage therapists were instructed not to talk during the massage and to discourage the participant from talking.

Study findings reported less anxiety ($p < 0.01$) and improved mood ($p < 0.05$) for participants following their first and their last massage. The massage participants also had significantly lower EDI scores at the end of the 5-week intervention ($p < 0.05$) and higher dopamine and norepinephrine levels ($p < 0.05$). There were no significant differences in depression scores between groups (Hart et al., 2001).

29.3 Mindful Awareness in Body-Oriented Therapy, Eating Disorders, and Substance Abuse

One small RCT investigated MABT, ED, and SUD (Price et al., 2012). Forty-six women were randomized to either MABT plus TAU or TAU only. Eight 90-min weekly sessions were offered. MABT was delivered through clothing with the massage portion varying between sessions: maximum time 45 min, minimum time 15 min. MABT was found to be significantly beneficial for reducing both alcohol and ED problems. There were significantly fewer days of substance use for the MABT + TAU group at the end of the intervention ($p < 0.02$) and at 6- and 9-month follow-up ($p = 0.01$). EDE-Q scores, depression, and anxiety were significantly lower at the 6- and 9-month follow-up ($p < 0.02$). At 9-month follow-up perceived stress was significantly decreased for the MABT group. This small study lacked a control for time and attention received by the MABT group.

29.4 Therapeutic Massage and Substance Use Disorders

A number of studies including three published RCTs have demonstrated significant benefits for the use of massage as an adjunct therapy in substance withdrawal (Hernandez-Reif, Field, & Hart, 1999; Reader, Young, & Connor, 2005; Scafidi et al., 1996; Wheeden et al., 1993). In the first of the largest of these trials, 50 consecutive admissions to a hospital-based drug and alcohol detoxification clinic (41 male and 9 female) with alcohol dependence were randomized to receive either a daily 15-min massage intervention or 15 min of bed rest. The massage intervention involved a seated massage of the shoulders, neck, and head while fully clothed on the first 4 days of the substance detoxification program. Subjects in the massage group demonstrated less physiological arousal and significantly lower withdrawal scores than the control group on day 1 ($p < 0.05$), with lower pulse rates on 3 of the 4 days ($p < 0.05$) and lower respiratory rates on day 4 compared to controls ($p < 0.01$). Higher rates of satisfaction were reported in the massage group (Reader et al., 2005).

In the second RCT, 30 cocaine-exposed, preterm infants were randomized once medically stable to TAU or TAU and massage. The massage intervention consisted of three 15-min periods, during 3 consecutive hours a day, for 10 days. The first and last 5 min of the intervention involved stroking of different body parts, while the middle 5-min period involved flexing and extending the infants' limbs. Infants in the massage intervention showed an average of 28 % greater weight gain a day ($p < 0.01$), decreased postnatal complications and stress behaviors ($p < 0.03$), and more mature motor behaviors ($p < 0.02$) than the control group (Wheeden et al., 1993).

In the final RCT 20 individuals were randomized to a self-massage intervention or a control group. Those in the self-massage group were taught and given written instructions on self-massage for the hand or ear. They were encouraged to use the massage three times a day for 5 min when experiencing withdrawal symptoms for a 30-day period. Those in the massage group experienced significantly lower levels of depression, anxiety, and withdrawal symptoms ($p < 0.05$) and were smoking significantly fewer cigarettes a day by the end of the intervention ($p < 0.05$) (Hernandez-Reif et al., 1999).

29.4.1 Herbal Medicine

"Herbal medicine—also called botanical medicine or phytomedicine—refers to the use of a plant's seeds, berries, roots, leaves, bark, or flowers for medicinal purposes" (University of Maryland Medical Center (UMMC), 2011). Plants have been part of indigenous cultures, traditional medicine systems, and ancient Chinese and Egyptian civilizations for medicinal purposes as early as 3,000 BC (University of Maryland Medical Center (UMMC), 2011). While herbal medicines are widely used by individuals with eating and SUD, clinical studies are limited with most evidence coming from animal studies or small case series.

Evidence for a possible role of herbal medicine in the treatment of ED and SUD comes almost exclusively from animal studies, primarily studies of mice. In the few studies that have looked at herbal medicine in humans, results have been equivocal or derived from noncontrolled trials.

29.4.1.1 Herbal Medicine and Eating Disorders

Only one herb, *Rhodiola rosea* (and its active principle, salidroside) has been investigated for its use in treating ED. It has been researched for its therapeutic effect as an individual herb and in combination with other herbs. The plant *R. rosea* is used in traditional medicine in Eastern Europe and Asia (Cifani et al., 2010). *R. rosea* is grown at high altitudes and northern latitudes (Ishaque, Shamseer, Bukutu, & Vohra, 2012). The herb is reputed to have adaptogenic properties, which is the capacity to decrease cellular sensitivity to stress (Cifani et al., 2010; Ishaque et al., 2012). It is hypothesized that *R. rosea's* effect on BED might come from its influence on the hypothalamic-pituitary-adrenal axis, reducing serum cortisol levels (Cifani et al., 2010). The exact mechanisms underlying the anti-anorectic effects of *R. rosea* are unknown, but it is hypothesized that it could be attributed to *R. rosea's* ability to modulate the activation of several components of the stress-response system (Mattioli & Perfumi, 2007). Cifani et al. (2010) examined the effect of *R. rosea* with reducing or abolishing binge eating episodes in rats. Binge eating in 120 female rats was evoked by three 8-day cycles of food restriction and refeeding (4 days of 66 % of usual chow intake; 4 days food ad libitum), followed by acute stress on the test day to evoke binge eating. The stress procedure involved placing highly palatable food in the rat's cage just out of reach of the rat, where it could see and smell the food. The rats were divided into four groups (30 in each group) (1) nonrestricted and no stress, (2) restricted and no stress, (3) nonrestricted and stress, and (4) restricted and stress. *R. rosea* extract was given 60 min before access to the highly palatable food, and rats were given 60 min access to the highly palatable food. The study found 10 mg/kg of *R. rosea* significantly reduced binging in stressed and food-restricted rats, and a 20 mg/kg dose completely abolished binging in the stressed and food-restricted rats. *R. rosea* did not affect the food intake of rats in conditions where binge eating was not expressed, and no adverse events were indicated (Cifani et al., 2010).

A second animal study investigated the use of *R. rosea* to prevent stress-induced anorexia in rats. Rats were allocated to six experimental groups (1) freely feeding rats ($n = 44$); (2) restraint stress-induced anorexia rats ($n = 41$) (rats were food deprived for 20 h and then received *R. rosea*; 60 min later restraint stress was applied (restrained in cylindrical Plexiglas tube) and then food was made available ad libitum); (3) corticotrophin-releasing-factor (CRF)-induced anorexia ($n = 32$) (20-h food-deprived rats received *R. rosea*, and then 60 min later they received an injection of CRF; rats were given free access to food for 20 min post CRF injection); (4) lipopolysaccharide (LPS)-induced anorexia ($n = 31$) (food-deprived rats were injected with LPS; 4 h later they received *R. rosea*, and *then* 60 min later rats were offered access to food); (5) fluoxetine-induced anorexia ($n = 29$) (food-deprived rats received *R. rosea*; 60 min later they were injected with fluoxetine, and

then 30 min later they were allowed free access to food); and (6) food intake in food-deprived rats ($n = 46$) (rats deprived of food for 20 h and then rats received *R. rosea*; then 60 min later rats were given free access to food). The study results showed that *R. rosea* significantly reduced the stress-induced anorexia in two experimental groups only: restraint stress-induced ($p < 0.01$) and CRF-induced anorexia in rats ($p < 0.01$). No adverse effects were reported and food intake was not modified in rats who were freely feeding or those who were not stressed (Mattioli & Perfumi, 2007).

The effect of the herbal supplement in humans has been reported in a single clinical study. Ross, Herman, Rocklin, and Rojas (2008) retrospectively analyzed data from two cohorts who were treated for insomnia and constipation by conventional medicine ($n = 27$) or integrative medicine ($n = 38$) (received integrative medicines and conventional treatment if needed). Data was obtained for a 6-month period in consecutive years (1 June 2004 to 30 November 2005). For patients in the integrative care group, they received a herbal medicine containing *R. rosea*, valerian root, hops strobiles, passionflower, and German chamomile flower. The medication group was treated with trazodone. Patients with insomnia only were included in the integrative medicine group. The study reported both groups slept well; however, there was significantly less medication use among patients using the herbal extract compared to those medicated pharmaceutically ($p = 0.001$) (Ross et al., 2008).

29.4.1.2 Herbal Medicine and Substance Use Disorders

A number of herbs and their extracts have been identified as potentially having a role in SUD. Despite this, there are only a handful of clinical trials that have examined this issue with most evidence arising from animal studies. Herbal products that have been investigated in SUD include Radix Puerariae (Kudzu), Tabernanthe iboga, and ginseng. Of these, only Radix Puerariae has been evaluated in controlled clinical trials.

Radix Puerariae, also called Kudzu, comes from the root of a leguminous plant Puerariae that is native to Eastern Asia. It was first used to treat alcohol-related problems almost 1,000 years ago including alcohol intoxication and abuse (Lu et al., 2009). Its major extract daidzin is a potent, selective, reversible inhibitor of acetaldehyde dehydrogenase, an enzyme that is essential to the breakdown of acetaldehyde, a toxic compound arising from ethanol metabolism. Blockage of this enzyme leads to severe nausea, similar to the action of disulfiram.

There have been two RCTs of Radix Puerariae in alcohol use, but with contradictory results. In the first of these trials, 38 subjects with alcohol dependence were given either 1.2 g of kudzu root extract or placebo for 1 month and asked to fill in questionnaires of alcohol craving and alcohol consumption. At the end of 1 month, no significant difference was seen between the two groups (Shebek & Rindone, 2000). In the second trial 14 volunteers (11 male) with heavy alcohol consumption were randomly assigned to receive either 500 mg of kudzu extract or placebo for 7 days. Subjects were then allowed to freely drink their preferred beer. Those who were given kudzu extract drank significantly less alcohol than the placebo group

(Lukas et al., 2005). Further evidence to support a possible role for Radix Puerariae comes from a series of animal studies that have shown that kudzu root extract reduces alcohol consumption in alcohol-preferring rats (Overstreet, Keung, Rezvani, Massi, & Lee, 2003; Overstreet, Knapp, Moy, & Breese, 2003; Rezvani, Overstreet, Perfumi, & Massi, 2003).

Panax ginseng (Asian ginseng) has been used in Chinese traditional herbal medicine for many centuries and is widely known in the West. Its main active components are called ginsenosides of which more than 20 have been isolated. While there have been no controlled clinical trials of ginseng, animal studies have demonstrated reduced withdrawal symptoms in opiate-dependent mice and reduced behavioral effects from methamphetamine intoxication in mice (Lu et al., 2009).

Tabernanthe iboga and Voacanga africana are two African shrubs that are used widely in traditional African medicine. Its active component ibogaine has been anecdotally reported to reduce opiate withdrawal and cravings and in one small uncontrolled trial was seen to reduce withdrawal symptoms and drug use in seven opiate-dependent subjects (Lu et al., 2009). In animal studies ibogaine has been shown to reduce self-administration of morphine and cocaine and reduce the behavioral effects of cocaine, nicotine, and methamphetamine in mice (Lu et al., 2009).

29.4.2 Hypnosis

The American Psychological Association defined hypnosis as typically involving three aspects: an introduction during which the subject is told that suggestions for imaginative experiences will be presented, a hypnotic induction involving an extended initial suggestion for using one's imagination that may contain further elaborations of the introduction, and a hypnotic procedure to encourage and evaluate responses to suggestions (Green, Barabasz, Barrett, & Montgomery, 2005). These techniques are used to guide a person to respond to suggestions to change their "subjective experience, alterations in perception, sensation, emotion, thought or behavior" (Green et al., 2005).

Hypnosis has been used in the treatment of individuals with SUD, AN, BN, BED, and distorted body image (Anbar & Savedoff, 2005; Barabasz, 2007; Mantle, 2003; Túry, Wildmann, & Szentes, 2011; Walsh, 2008, 2010). It has, however, been noted that severely ill AN sufferers may have issues with cognitive processing due to malnutrition and that hypnosis may not be appropriate for these individuals (Mantle, 2003). There are a number of different techniques used in hypnosis including a CBT aspect, ego therapy, and age regression (Mantle, 2003). These techniques have their origins in psychology.

29.4.2.1 Hypnosis and Eating Disorders

A literature review of CAM reported hypnosis was the most widely cited and popular CAM treatment for AN, though no RCTs of hypnosis in the treatment of AN were found (Birmingham & Sidhu, 2007). Similarly research into the use of

hypnosis in BN is characterized by a small number of clinical studies, mostly case and anecdotal reports, and narrative reviews discussing methodological issues including the capacity of individuals to respond to hypnosis and different clinical approaches to the use of hypnosis (Anbar & Savedoff, 2005; Barabasz, 2007, 2012; Hutchinson-Phillips, Gow, & Jamieson, 2007; Mantle, 2003; Torem, 1992; Túry et al., 2011; Walsh, 2008, 2010).

The capacity for hypnotic responses varies between individuals. This has been investigated in two studies. Kranhold, Baumann, and Fichter (1992) conducted a pilot nonrandomized comparison study of an unspecified form of hypnosis investigating hypnotizability in 15 ED inpatients. Hypnotizability as measured by the Harvard Group Scale of Hypnotic Susceptibility demonstrated patients diagnosed with BN were more hypnotizable than those with AN ($p < 0.05$) (Kranhold et al., 1992).

In a second study of patients with AN and BN, Vanderlinden, Spinhoven, Vandereycken, and van Dyck (1995) conducted a comparison study investigating hypnotizability in 53 inpatients with ED and 86 control subjects. ED participants demonstrated significantly higher levels of hypnotizability and dissection compared with controls ($p < 0.0001$). Significant differences were found between ED subgroups, with AN binge purge subtype and BN participants scoring higher on measurement scales indicating greater hypnotizability ($p < 0.0001$) (Vanderlinden et al., 1995). Both studies confirm ED patients have high hypnotizability and consequently hypnosis may be a valuable adjunctive therapy.

The clinical effects of hypnosis in BN have been evaluated in three trials (Griffiths, 1995; Griffiths, Hadzi-Pavlovic, & Channon-Little, 1994, 1996). In the first RCT to look at hypnosis in EDs, subjects were randomized to hypno-behavioral treatment (HBT), CBT, or a wait-list control group (Griffiths et al., 1994, 1996). Seventy-eight participants were randomized to HBT, CBT, or a wait-list control. Seven CBT or HBT treatments (60-min duration) were administered over an 8-week period. Significant differences were found: Eating Attitudes Test (EAT) total score ($p < 0.001$), the EAT-dieting subscale ($p < 0.001$), EAT-bulimia and food preoccupation subscale ($p < 0.001$), and the EDI-3 bulimia subscale ($p < 0.001$) for the two treatment groups compared to the wait-list control. No differences were found between the two active groups (CBT and HBT).

In a second RCT involving the wait-list controls being allocated to either HBT or CBT after the completion of their waiting period, no differences were found between HBT and CBT for ED pathology, bulimic behaviors, or binging and purging outcomes (Griffiths et al., 1994, 1996).

Griffiths (1995) also reported on an uncontrolled 2-year follow-up study on 14 women (9 from the above studies and an additional 5 subjects). All participants received 7 sessions of hypno-behavioral hypnosis for BN over an 8-week period. Follow-up at 2 years involved completing psychological questionnaires and ED health questions about binge eating and compensatory behavior over the previous 3-month period. Reductions in binge eating and vomiting compared to pretreatment baseline were seen post treatment (binging $p < 0.02$, vomiting $p < 0.01$), at 6 weeks follow-up (binging $p < 0.01$ and vomiting $p < 0.01$), and at 2-year follow-up

(binging $p < 0.01$, vomiting $p < 0.01$). There was also a significant reduction in ED symptomology comparing pretreatment and 2-year follow-up: drive for thinness ($p < 0.0001$), bulimia ($p < 0.003$), body dissatisfaction ($p < 0.003$), ineffectiveness ($p < 0.01$), and interceptive awareness ($p < 0.0001$).

Other clinical evidence suggesting a benefit for hypnosis has been supported in a series of case presentations and hypnosis approaches to ED conditions including AN (Walsh, 2010), BN (Túry et al., 2011; Walsh, 2010), BED (Anbar & Savedoff, 2005), and distorted body image (Walsh, 2008).

29.4.2.2 Hypnosis and Substance Use Disorders

As in ED, hypnosis is frequently promoted and used as a treatment for SUD. While the use of hypnosis has been investigated in a large number of RCTs in smoking cessation, only one RCT of its use in abstinence from substance use has been published (Pekala et al., 2004). Otherwise, evidence for the use of hypnosis is limited to a number of case studies (Potter, 2004).

A recent systematic review identified 11 RCTs for smoking cessation in which hypnosis was compared to 18 other interventions including no treatment, rapid smoking, or other psychological interventions. The results from these trials were heterogeneous and often conflicting. Based on their review the authors concluded that there was no evidence to support increased effectiveness of hypnosis compared with other treatments or no treatment (Barnes et al., 2010).

One RCT has compared self-hypnosis to two other psychological interventions, transtheoretical cognitive behavioral and stress management, and TAU in 261 patients with dual diagnosis admitted to a 21- or 28-day substance abuse residential rehabilitation program. Study interventions involved the provision of four 1-h sessions. In the hypnosis group participants experienced four hypnotherapy protocols: self-esteem enhancement, serenity enhancement, relapse prevention, and anger and anxiety management/reduction. Participants were given four audiocassettes to listen to upon discharge. Relapse was assessed at the end of the intervention and again at 7 weeks where no difference was found between groups (Pekala et al., 2004).

29.4.3 Spiritual Healing

Spiritual healing is the use of spirituality to assist in healing. Spirituality is defined as experiencing "compassion, love, acceptance of love, hope, being inspired, feeling enlightened, honesty, congruence, gratitude, and a sense of meaning and purpose." Spirituality is nondenominational and can cover all religions and those who are nonreligious. Spiritual healing is used in ED as an adjunct therapy for enhanced treatment outcomes (Berrett, Hardman, & Richards, 2010). Integration of spirituality appears common in some settings. In the USA 150 ED treatment programs were identified as addressing religious or spiritual issues (Berrett et al., 2010).

29.4.3.1 Spiritual Healing and Eating Disorders

One treatment approach to how spirituality may help with treatment recovery has been outlined by Berrett et al. (2010). This approach utilizes six spiritual pathways to recovery including listening to and following the heart, learning a language of spirituality, mindfulness and spiritual mindedness, principled living, giving and receiving gifts of love, and holding up a therapeutic mirror that reflects spiritual identity. This has been examined in one RCT (Scott Richards, Berrett, Hardman, & Eggett, 2006). The study included 122 women diagnosed with AN, BN, or EDNOS in an inpatient setting. Participants all received TAU and were randomized to a spirituality intervention, a cognitive intervention, or an emotional support group. The spirituality intervention involved the reading of *Spiritual Renewal: A Journey of Faith and Healing*, a self-help nondenominational spiritual workbook, and participation in weekly 60-min groups addressing key concepts from the workbook. The cognitive intervention included reading *Mind Over Mood: Change How You Feel by Changing the Way You Think*, a self-help book including cognitive-based interventions, plus weekly 60-min groups discussing important concepts from the workbook. The emotional support group intervention involved attending a weekly 60-min "open topic" support group. All groups improved over time. Participants in the spirituality group scored significantly lower on measures of ED symptoms ($p < 0.05$) at the end of the treatment compared to the other groups.

29.4.3.2 Spiritual Healing and Substance Use Disorders

Spirituality is a common part of many substance use interventions including Alcoholics Anonymous and Narcotics Anonymous. The 12-step facilitation approach is reviewed in detail in Chaps. 12 and 27 and for this reason will not be reviewed here. In summary, however, studies have demonstrated a positive relationship between 12-step involvement and improvement in substance use outcomes for both alcohol- and drug-dependent individuals for periods ranging up to 16 years (Donovan et al., 2012). The largest and longest standing study of 12-step facilitation is Project MATCH which randomized 952 alcohol-dependent outpatients and 774 alcohol-dependent adults following inpatient admission to 12-step facilitation, motivational interviewing, or CBT. The study found improvements in all arms with 12-step facilitation superior to CBT in individuals with low levels of psychiatric comorbidity but no difference between treatments in individuals with high levels of psychiatric comorbidity (Anonymous, 1997). Excluding 12-step facilitation the literature reveals only one RCT and a follow-up cohort study looking at spirituality and substance use.

In this RCT to examine the role of spiritual healing in SUD, 60 poly-substance users (36 male and 24 female) admitted to a public hospital inpatient program for substance dependence were randomly assigned to TAU or TAU and a manualized spiritual intervention. The spiritual intervention involved 12 manualized, individual sessions on spirituality provided by a "certified spiritual director" over the course of the patients' admission. The treatment manual was based on Judeo-Christian concepts of spirituality. TAU consisted of behavioral counseling and educational interventions. The average length of inpatient treatment was 25 days. All patients

were then assessed at baseline and 4, 8, and 12 months after baseline. This study did not demonstrate any significant differences between the two groups with regard to substance use outcomes or spiritual practices at any assessment point. Interestingly higher rates of depression and anxiety were seen in the group who received the spiritual intervention 4 months post intervention (Miller, Forcehimes, O'Leary, & LaNoue, 2008).

As a follow-up to this study, a further 80 patients were enrolled in two cohorts of 40 to receive either TAU or the manualized spiritual intervention, though this time the intervention was provided by all treatment staff. Again no differences in substance use outcomes or spiritual practices were seen between groups (Miller et al., 2008).

Despite these findings spirituality is consistently reported as an important component of treatment and recovery by both staff and patients involved in substance use treatment (Brown et al., 2007; Galanter et al., 2007; Heinz, Epstein, & Preston, 2007; Mason, Deane, Kelly, & Crowe, 2009; Novins et al., 2012). This is particularly so in culturally specific programs (Novins et al., 2012). Finally spirituality has been shown to be protective for the development of SUD (Coyle, Crum, & Ford, 2006; Miller et al., 2008).

29.4.4 Phototherapy

Phototherapy is the substitution of sunlight with full-spectrum artificial light for medical benefit. Phototherapy is used predominately to treat depression (Blouin et al., 1996; Braun, Sunday, Fornari, & Halmi, 1999; Janas-Kozik et al., 2011; Lam, Goldner, Solyom, & Remick, 1994). Possible theories of the action include phototherapy's "modulatory effect on serotoninergic systems, which may be dysregulated in ED and SUD" (Blouin et al., 1996; Krysta, Krzystaneck, Janas-Kozik, & Krupka-Matuszczyk, 2012). It is also hypothesized that phototherapy modulates the circadian rhythm, normalizing body temperature and hunger, both of which are affected in AN and BN (Yamamotova, Papezova, & Vevera, 2008).

While there are no studies that have specifically looked at the impact of phototherapy on SUD, high rates of comorbid depression and anxiety (Hides et al., 2011) and the negative impacts these disorders have on treatment outcomes suggest a possible role for phototherapy in this group of patients.

29.4.4.1 Phototherapy and Eating Disorders

Three studies have investigated the effect of phototherapy on the management of BN (Blouin et al., 1996; Braun et al., 1999; Lam et al., 1994). Lam et al. (1994) undertook a blinded randomized crossover trial of 17 female patients with BN. The two phases were light therapy (10,000 lx for 30 min) or controlled light condition (500-lx red light for 30 min). Light exposure was administered daily for 2 weeks in the early hours of the morning between 7 and 8 am (Lam et al., 1994). There was no washout period. Treatment was given during the winter months (October–March). The study found phototherapy was significantly more beneficial than dim red light

at reducing binging and purging ($p < 0.05$) and reducing depression ($p < 0.05$) (Lam et al., 1994). The authors found those with a seasonal pattern of symptoms had greater improvement on total binge eating episodes ($p < 0.05$), total purging episodes ($p < 0.04$), and depression ($p < 0.0002$).

In a RCT Blouin and colleagues recruited 18 women with BN. Ten of the eighteen subjects met the criteria for SAD. Participants were allocated to either phototherapy (2,500 lx 120 min) or dim light therapy (placebo <500 lx) for 7 days, receiving treatment from 17:00 till 19:00 h. The phototherapy group demonstrated significantly reduced depression ($p < 0.05$), but no differences were found for binge eating or purging (Blouin et al., 1996).

Braun and coworkers conducted a double-blinded placebo-controlled RCT of 34 females with BN (Braun et al., 1999). The study participants were randomly allocated to receive either phototherapy (10,000 lx 60 min) or dim red light (placebo 500 lx 60 min). Treatment was administered every morning between 06:00 and 09:00 h for 3 weeks in November–December or January–March. Fifteen subjects met the criteria for SAD. A significant benefit was found for the phototherapy group with a decreased binge eating frequency ($p = 0.017$) compared to the placebo.

A RCT investigated phototherapy in 24 restrictive AN subjects (Janas-Kozik et al., 2011). The study interventions, CBT plus phototherapy (10, 000 lx, 30 min daily) or CBT only, were administered for 6 weeks. Phototherapy was administered in the morning. The study showed a significant improvement in depressive symptoms for the phototherapy + CBT group ($p < 0.001$) (Janas-Kozik et al., 2011).

29.4.5 Art Therapy

Art therapy employs creative arts such as drawing, dance, music, and drama to address psychological distress and disorders (Frisch, Franko, & Herzog, 2006; Holmqvist & Persson, 2012). Art therapy utilizes psychological and developmental theories integrated in the art form to try to bring about personal growth and a positive change (Frisch et al., 2006). In art therapy similar modalities can be used for different outcomes. Music, for example, can be used as a tool for self-discovery or a method of relaxation, while dance can be used to impact the mind through work with the body or as an exploration of feelings (Frisch et al., 2006).

In a survey undertaken in 2006, of 22 residential treatment programs based in the USA, 19 reported offering art-based therapy at least once a week. In these settings therapy was provided as an outlet for self-discovery and self-expression and a nonthreatening outlet for patients who had difficulty with talk-oriented therapies (Frisch et al., 2006).

A review of the literature identified no RCTs evaluating the use of art therapy in the treatment of eating or SUD. A recent literature review identified a small number of studies of art therapies used in the treatment of ED (Holmqvist & Persson, 2012).

The review concluded that the study quality was poor and there was no evidence for the use of art therapy in the treatment of ED.

29.5 Summary

Despite the many studies reporting improvements in outcomes, no CAM intervention has been identified as an effective, evidence-based treatment in ED and SUD. Research into CAM therapies is limited to a small number of studies often with poor quality designs. Studies are often small, frequently lack appropriate controls, interventions are potentially underdosed, and studies include a mix of patient diagnoses.

With the exception of acupuncture and SUD, much of the research for CAM therapies in eating and SUD is in its infancy, though research capacity is developing. Findings from acupuncture have been disappointing with large RCTs not demonstrating efficacy in SUD. Preliminary findings from evaluations of yoga, hypnosis, and massage show promise. Findings from studies using phototherapy are mixed and confined primarily to eating and mood disorders, while the effectiveness of herbal medicine, art therapy, and spirituality has yet to be demonstrated. As such there is a need to build research capacity and undertake evidence-based research.

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Civil Commitment in the Treatment of Eating Disorders and Substance Abuse: Empirical Status and Ethical Considerations

30

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Abstract

Civil commitment has evolved over the years to reflect the ideas of community, mental health professionals, and law. Individuals with an eating disorder or substance abuse problems can show a high degree of reluctance for treatment while displaying an inability to assess the outcome of their actions. When the safety of the individual clashes with their desire for maintaining the status quo or the individual is incapacitated due to the consequences of an eating disorder or substance use disorder, a discussion of involuntary treatment must be considered. The consequence of those behaviors directs the healthcare provider or family toward coercion. The perception of coercion in civil commitment is complex and not necessarily related to the degree of restriction of freedom. Civil commitment is a legitimate tool in emergent situations when an eating disorder or substance use disorder becomes life threatening. Compulsory treatment can be viewed as being in the best interest of the patient, family, and care provider. Civil commitment as a method to providing treatment is not without its critics or controversies, and a host of ethical concerns accompanies the use of this approach. Although controversial, there is a role for civil commitment in the treatment of eating disorders and substance use disorders.

Keywords

Civil commitment • Eating disorders • Ethics • Substance use disorders • Psychiatric advance directive

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30.1 Introduction

Civil commitment or compulsory treatment for psychiatric disorders has been in existence for over 150 years (Testa & West, 2010). How and when civil commitment has been implemented has evolved over the years to reflect the ideas of community, mental health professionals, and law. The use of involuntary hospitalization has been argued as a critical first step in psychiatric care and has been seen as a mainstay to the initiation of psychiatric care (Testa & West, 2010). Patients who lack the capacity to assist in their own treatment, who deny the illness, or who refuse treatment that leads to life-threatening or life-impeding outcomes are often embroiled in civil commitment.

Balancing the treatment needs of the mentally ill with protection of personal freedom and civil liberty is one of the greatest challenges of civil commitment law. The first European institutions specifically for people with mental illness were established in 1403 in London, England, and Valencia, Spain, in 1407. Unfortunately, Europeans increasingly isolated mentally ill people, often housing them with handicapped people, vagrants, and delinquents. Additionally, those considered insane were treated inhumanely, often chained to walls and kept in dungeons. Concern about the treatment of mentally ill people grew to the point that occasional reforms were instituted. After the French Revolution, French physician Philippe Pinel took over the Bicêtre hospital and stopped the use of chains and shackles. He also removed patients from dungeons, provided sunny rooms, and allowed exercise.

In England in the nineteenth century, individuals with mental illness were often treated as subhuman and were housed in prisons. The authority of two justices of peace could involuntarily detain an individual with mental illness. During the Victorian Era, there was a movement to provide more positive structure and compassionate care. Early asylums, although a step forward, were still patterned after penitentiaries. More humane hospital environments were eventually legislated into existence and included inspection of private and public hospitals with the intent of balancing the protection of society while protecting patients against abuse. At this time in history, civil commitment was usually instituted by a relative or other person supported by two medical doctors. Mental health legislation relied upon the family as protector of patients but left patients unprotected from improper detention at the instigation of relatives. By the 1890s, civil commitment was now in the hands of a magistrate or county judge (Fennell & Goldstein, 2006; Jacobsen, 2012).

In the United States, prior to the Revolutionary War, mentally ill individuals who could not care for themselves and lacked family supervision or support were typically ignored or managed in jails or houses for the poor. Philadelphia created the first psychiatric hospital in 1752, and by the early decades of the nineteenth century, a few small private and public facilities had developed across the states. Between 1817 and 1824, four privately funded asylums were established in the northeastern states of Connecticut, New York, Massachusetts, and Pennsylvania. Admissions were always involuntary, typically initiated by family or friends, and the length of stay was linked to ongoing private financial support. Hospital treatment was coerced, since it was presumed that mentally ill patients were too disabled

to request (or refuse) care on their own behalf. These early commitment laws focused more on the need for treatment with the state acting in its parens patriae function, the traditional power to care for those incapable of caring for themselves (Anfang & Appelbaum, 2006; Appelbaum, 2006; Fennell & Goldstein, 2006).

In the early twentieth century, "psychopathic hospitals" were developed in major cities, dedicated to caring for acute cases, in the hope that early intervention and treatment would have greater therapeutic impact. States developed special emergency commitment procedures that would bypass time-consuming judicial hearings, allowing physicians (and sometimes police) to hospitalize patients emergently for brief periods of time without court review.

By the 1950s, care of all mental health problems was operating under a single legal framework. Also, tribunals were established to oversee the legality of civil commitment and its continuation. The tribunals consisted of a legally qualified president, a psychiatrically qualified medical member, and a third member with expertise in the operation of the mental health system. To be civilly committed, a patient had to be suffering from a mental disorder of a nature or degree warranting detention for assessment and/or treatment. When treatment was necessary, it had to be in the interest of the patient's health or safety or for the protection of others (Fennell & Goldstein, 2006; Jacobsen, 2012).

In 1951, the newly established National Institute of Mental Health (NIMH) issued the landmark "Draft Act Governing Hospitalization of the Mentally Ill" which proposed streamlining commitment procedures, including a certification process that was entirely in medical hands (Anfang & Appelbaum, 2006). The NIMH Draft Act proposed a modified version of the traditional "need for treatment" formula. To involuntarily hospitalize a person, there must be proof that the individual is in need of care or treatment in a mental hospital. Additionally, the individual must lack sufficient insight or capacity to make responsible decisions (Anfang & Appelbaum, 2006; Fennell & Goldstein, 2006).

Dangerousness as the sole ground for civil commitment was first adopted by the District of Columbia in 1964 and then by California. California's 1969 Lanterman-Petris-Short Act permitted civil commitment only for those who were imminently dangerous to themselves or to others or who were so "gravely disabled" as to be unable to meet their minimal needs for survival (Anfang & Appelbaum, 2006). This approach to involuntary hospitalization quickly became the model for many US states. The need for treatment was no longer a substantive factor for civil commitment. By the end of the 1970s, nearly every state had revised its commitment statutes to conform to the dangerousness criteria (http://www.treatmentadvocacycenter.org/). Over the past 20 years, several states have broadened the definition of "grave disability" for inpatient commitment to include the prospect of severe deterioration, disabling illness, or general inability to care for oneself (Anfang & Appelbaum, 2006; Testa & West, 2010).

The evolution of modern civil commitment is, over time, the story of interactions among state legislatures, lay and professional interest groups, and courts. Civil commitment in the USA has been shaped by multiple factors, including sensitivity to civil rights, public perception of psychiatry, availability of resources, and larger

economic pressures. With the intent of balancing the desire to help the "gravely disabled" and the fear of restricting personal liberty, American civil commitment law has evolved but has not yet developed statutes that are consistently fair, reasonable, and compassionate. Perhaps we can create a consistent view of civil commitment if we merge our experience with the experience and the experiments of other nations (Anfang & Appelbaum, 2006; Appelbaum, 2006; Bloom, 2004)

30.2 Civil Commitment with Eating and Substance Use Disorders

Civil commitment as an approach to providing treatment is not without its critics or controversies, and a host of ethical concerns accompany the use of civil commitment. Among the most debated ethical concerns are showing respect for the patients' autonomy (allowing patients to make their own decisions), non-maleficence ("do no harm"), beneficence (providing care that will benefit the patient), and paternalism (interfering with a person's freedom for his or her own good). Each of these principles adds a confounding dimension to the idea of involuntary treatment. This is especially complicated when treating individuals with eating disorders (ED) and substance use disorders (SUD) which do not obviously or grossly impact the individual's reality testing (Testa & West, 2010). Along with ethical considerations are the legal aspects of civil commitment. In the USA, the prevailing standard for civil commitment is the presence of dangerousness as a result of a mental disease. The standard that most states invoke when considering civil commitment is based on the Supreme Court's criteria for holding an individual in a hospital against their will. Basically, an individual needs to have a diagnosed mental illness and a known risk of self-harm or harm to others (Ferris, 2008; Grace & Hardt, 2008).

Civil commitment for psychiatric disorders occurs in all states, but the standards vary from state to state. In eight states, the sole grounds for civil commitment are "dangerousness" which means an individual must demonstrate an immediate, physical danger to self or others before a court can intervene and order treatment. In the remaining 42 states, laws permit intervention based on additional criteria that are broader than "dangerousness to self or others" and generally include a second standard, referred to as "grave disability," which typically focuses on the person's inability to meet his or her basic survival needs. In 26 states, there is a third provision under which a court can intervene in a mental health crisis, that is, the "need-for-treatment" standard. Need-for-treatment standards typically include qualification for care based on at least one of the following conditions (1) the person's inability to provide for needed psychiatric care, (2) the person's inability to make an informed medical decision, and (3) the person's need for intervention to prevent further psychiatric or emotional deterioration (The Treatment Advocacy Center, 2013).

Forty-four states permit the use of assisted outpatient treatment (AOT), also known as outpatient commitment or court-ordered outpatient treatment. AOT is

court-ordered treatment (including medication) for individuals with symptoms of severe mental illness who meet strict legal criteria (e.g., they have a history of medication noncompliance). Studies and data from states using AOT have found that AOT is effective in reducing the incidents and duration of hospitalization, homelessness, arrests and incarcerations, victimization, violent episodes, and other consequences of nontreatment. AOT can increase treatment compliance and promotes long-term voluntary compliance. Typically, violation of the court-ordered conditions can result in the individual being hospitalized for further treatment (Geller, 2006; Honig & Stefan, 2005).

By contrast, only 38 states allow outpatient or inpatient civil commitment of individuals with SUD in the absence of criminal conduct, while four states allow police pickup or emergency hospitalization. Like involuntary treatment for psychiatric disorders, most of the 38 states require dangerousness to self or grave disability to be present to seek civil commitment for SUD, while others require a combination of criteria (Pinals & Mossman, 2011).

Two populations that can be involved in civil commitment are those individuals with an ED and those with SUD. Eating disorders, especially anorexia nervosa (AN), can pose serious physical and mental health risks, especially for untreated patients who fail to understand the seriousness of the disorder. The health risks for individuals who go untreated are pervasive and can adversely affect numerous organ systems with potential long-term and irreversible consequences. Untreated, these disorders can become chronic conditions interfering with an individual's ability to effectively develop normal social, psychological, academic, and occupational goals. The mortality rate for patients who develop AN is nearly six times the rate in the general population (Arcelus, Mitchell, Wales, & Nielsen, 2011). Additionally, AN can have one of the highest rates of death among psychiatric disorders further putting those individuals who go untreated at risk (Harris & Barraclough, 1998). Inherent in the disorder is an inability or denial to understand the nature or seriousness of the disorder to the point that treatment refusal can threaten the total well-being of the individual. Civil commitment (compulsory treatment) must be considered and implemented when in the judgment of the treating clinician the individual no longer has the awareness to assist in their own care or treatment (American Psychiatric Association, 2006).

Individuals with SUD are also a population that show a high degree of reluctance for treatment and who do not display clear indications of an inability to assess the outcome of their actions. A study (Pinals & Mossman, 2011) found that less than 10 % of patients with SUD seek treatment. Most who seek treatment do so because of external, coercive influences (family, law enforcement) and that police pickup, emergency hospitalization, and civil commitment were the three forms of involuntary treatment most used by the states with legal statutes regarding involuntary commitment for SUD. Again, laws regarding involuntary commitment for SUD vary widely among states with only 38 states allowing some form of involuntary substance abuse treatment that is separate from any kind of criminal issues (Pinals & Mossman, 2011). The length of time in which a person can be involuntarily forced into treatment varies widely among states with the medium time being

3 days for emergency hospitalization and 3 months for civil commitment (Pinals & Mossman, 2011).

Internationally, civil commitment ranges from very strict legislation to none at all. Many western countries have similar language when it comes to civil commitment, but they tend to vary by region or state. Members of the European Union delineate that the compulsory admission of a mentally ill patient is legally permitted only when less restrictive alternatives might not be sufficient or available. Also, compulsory admission is seen as an intervention of last resort, or to be applied only in an acute crisis or state of emergency. Within the European Union, criteria for civil commitment can be categorized into three groups. A *serious threat of harm to the person himself and/or to others* ("dangerousness criterion") is an essential prerequisite for compulsory admission in Austria, Belgium, France, Germany, Luxembourg, and The Netherlands. Along with dangerousness, Italy, Spain, and Sweden use *the need for psychiatric treatment* as the crucial criterion qualifying a person for compulsory admission. Denmark, Finland, Greece, Ireland, Portugal, and the United Kingdom use the combination of *serious mental disorder and dangerousness* or *serious mental disorder and a need for treatment*. The French do not stipulate a specific legal framework for civil commitment but have established two broad procedures for civil commitment. The first, which is known as "Hospitalization d 'office," is executed by the police for persons suffering from mental health problems and considered an endangerment to public safety. The second, "Hospitalization à la demanded d'un tiers," entitles family members or other close persons to apply to have someone placed involuntarily who might be unable to ask for help or care by him- or herself (de Stefano & Ducci, 2008; Fennell & Goldstein, 2006; Jacobsen, 2012).

Germany, which has sixteen Federal States, independently organizes and regulates mental health care. Consequently, each Federal State provides a separate legal framework for regulating involuntary placement or treatment of the mentally ill. In Germany, their basic philosophy emphasizes human rights as well as the self-determination of mentally ill patients and demands appropriate mental healthcare delivery in the least restrictive setting possible. This has generated a variety of regulations or statutes across the Federal States, in an attempt to clarify or detail procedures for treating the mentally ill against their will. Nevertheless, despite all emphasis on need or right for treatment, the threat of harm to or by a mentally ill person marks clearly the crucial condition for civil commitment (de Stefano & Ducci, 2008).

Countries that belong to the Commonwealth of Nations have very similar civil commitment legislation. Very broadly, a person may be admitted to and detained in an approved mental health service as an involuntary patient when they appear to be mentally ill and when their mental illness requires immediate treatment and that treatment can be obtained by admission to and detention in an approved mental health service. Also, because of the person's mental illness, the person should be admitted and detained to protect his or her health or safety (whether to prevent a deterioration in the person's physical or mental condition or otherwise) or for the protection of the public. Additionally, civil commitment can be sought if the person

has refused or is unable to consent to the necessary treatment for the mental illness and the person cannot receive adequate treatment for the mental illness in a less restrictive manner. In Australia, mental health law is constitutionally under the state powers with each state applying different laws. Consequently, in some Australian states, the person must be a danger to society or themselves; while other states only require that the person be suffering from a mental illness that requires treatment. Like Australia, New Zealand law requires that the person must be a danger to themselves or others or be unable to care for themselves. Every province and territory in Canada has a legislation which permits individuals to be kept in a psychiatric hospital against their will. The conditions that must be satisfied are that the patient must be suffering from a mental disorder or mental illness and the patient must present a danger to self or others. If these two conditions are satisfied, the legislation authorizes the patient to be kept in a psychiatric facility for a short period (usually 72 h) for assessment purposes.

In the United Kingdom, legislation is based on different parts of the country with England and Wales following one set of statues with Northern Ireland and Scotland providing their own laws. For England and Wales, all cases of civil commitment must be justified on the basis that the individual has a serious mental disorder and that they pose a risk to harm themselves or others. Additionally, there must be access to appropriate treatment in the facility that one is being committed. In Scotland, a patient who has a mental disorder, and because of that mental disorder is not capable to make decisions about the provision of medical treatment, can be treated involuntarily. Civil commitment may occur when it is a matter of urgency to detain the patient in a hospital for the purpose of determining what medical treatment may be required. Also, the patient may be detained in a hospital if there would be a significant risk to the health, safety, or welfare of the patient or to the safety of any other person. Under Northern Ireland law, the criteria for civil commitment includes that the person must be suffering from a mental disorder and that failure to detain the individual would create a substantial likelihood of serious physical harm to the individual or others (Fennell & Goldstein, 2006).

30.3 Civil Commitment

Denial of illness and need for treatment and refusal to engage in treatment that prevents physical and psychological intervention are factors that can contribute to the consideration of civil commitment in a small group of people with ED and SUD. Clinicians who treat these individuals are often confronted with a decision of what is the best choice of care. Hospitalization can be considered when an individual becomes medically unstable with significant medical impairment and psychological and social abnormalities. Other reasons to consider hospitalization are a lack of response to a reasonable trial of outpatient treatment; significant comorbidity, especially suicidal thinking; a need for 24-h structured multidisciplinary care (psychological, behavioral, medical); and/or lack of family support or environmental toxicity (American Psychiatric Association, 2006).

As can be seen, an ED or SUD can be a debilitating, paralyzing, and life-threatening problem. Although some patients understand the risk of the disorder and that they are ill, these patients are reluctant to accept treatment even when they are severely impaired. This reluctance can lead to confrontations among the patient, family, and clinician. When the safety of the individual clashes with their desire for maintaining the status quo or the individual is incapacitated due to the consequences of an ED or SUD, a discussion of involuntary treatment must be considered. Whether the individual is driven by their sense of autonomy, a desire to maintain self-control, or an inability to see the nature of their disorder, the clinician must make decisions intended to prevent further deterioration of the individual's health and well-being. Failure of the individual to accept the intervention leads to civil commitment using existing legal standards to compel treatment.

The need for life-saving interventions creates numerous dilemmas for professionals who treat these disorders. The literature does not offer good data on outcome of civil commitment but there is some guidance when looking at AN. The limited empirical data, although contradictory in nature, suggest that involuntary treatment is beneficial (Andersen, 2007). It appears that a relatively small proportion of the ED population is severely ill enough to be detained for treatment. A study by Watson, Bowers, and Andersen (2000) indicated that 16.6 % ($N = 66$ of 397) of patients admitted to a hospital for treatment warranted civil commitment. One, 5-year follow-up of involuntary patients suggested that for severe cases, it appeared that treatment was of marginal benefit (Ben-Tovim et al., 2001). Patients with AN treated on an inpatient basis reported a stronger sense of coercion (Tan, Hope, & Stewart, 2003a, 2003b; Tan, Hope, Stewart, & Fitzpatrick, 2003). Ramsay, Ward, Treasure, and Russell (1999) confirmed that short-term treatment of involuntary and voluntary patients is comparably effective, but the long-term outcome is more problematic for the involuntary patients with mortality rates being higher at 5.5-year follow-up (Ramsay et al., 1999). Also, weight restoration was equivalent between voluntary and involuntary patients with slower progress for involuntary patients (Watson et al., 2000).

Is involuntary or coercive treatment justified in treating ED or SUD? When poor insight and the probable ego-syntonic nature of ED or SUD lead to treatment refusal and the potential for life-threatening outcomes, then the answer is yes! However, treatment to preserve health often violates the patients' desire for autonomy leading to an inherently paternalistic approach to care. The patients' response to involuntary weight restoration or forced cessation of the use of substances (paternalist care) can lead to a strong sense of coercion on the part of the patient. In fact, even in voluntary admissions to an inpatient unit, patients perceive coercive pressure from clinicians, family, friends, employers, or educators. Research suggests individuals with more frequent involuntary hospitalizations indicated that they were more resistant to treatment with 35 % of patients admitted to an ED specialty unit denying the need for admission (Guarda et al., 2007; Watson et al., 2000). Similarly these patients reported a significantly higher level of perceived coercion and pressure for admission by others than did patients who spontaneously requested admission. Researchers (Bindman et al., 2005; Rain et al., 2003; Swartz et al., 1999)

have also found that patients legally committed for involuntary treatment tend to hold more negative views of their hospitalization than patients admitted for voluntary treatment and report at discharge that little or no benefit has occurred. Ramsay et al. (1999) reported that involuntary commitment of patients with AN led to satisfactory short-term treatment results but increased long-term morbidity. The mortality at follow-up for the detained patients was 12.7 %, compared to 2.6 % for the voluntary patients. In contrast, other researchers (Guarda et al., 2007; Watson et al., 2000) have found that most involuntarily committed patients who initially objected to their commitment had positive views of their hospitalization and treatment at discharge and would want to be hospitalized in the future if they became dangerously ill again. Also, at the end of inpatient care, many involuntary patients report an understanding of and gave support to the need for treatment. These data reinforce involuntary treatment for some seriously ill patients (Watson et al., 2000). Additionally, the shift in initial resistance to treatment suggests that the patient's unrealistic beliefs are directly related to the disorder (Guarda et al., 2007; Watson et al., 2000). Nevertheless, for selected cases of individuals with ED or SUD that is life threatening and associated with denial of illness, a court-imposed use of involuntary treatment may be appropriate (Watson et al., 2000).

Compulsory treatment can be viewed as being in the best interest of the patient, family, and care provider. It is a serious decision that often creates a great deal of angst among the treatment providers and family. For the patient, civil commitment offers the opportunity to continue in treatment and work toward both physical and psychological wellness. The benefits for the healthcare professional include a means to address life-threatening medical and psychological problems when the patient does not agree with a professional's assessment. Equally important, civil commitment can provide the family or significant other with reduced emotional distress and reduce the stress of feeling scared or "trapped" regarding the health of their loved one.

On the other end of the continuum, civil commitment is viewed as violating the individual's rights to decide (autonomy). It has been suggested that civil commitment does not treat the disorder, as successful treatment requires patient cooperation. Some authors strongly suggest that civil commitment creates lasting damage to the therapeutic relationship, reducing the desire to remain in treatment. One criticism of civil commitment is that when improvement in the disorder does not occur past medical stabilization, there is no long-term positive outcome. Conflicting opinions exist regarding any involuntary treatment of a psychiatric disorder, especially AN. A review of the involuntary commitment literature (Hiday, 1996; Swartz et al., 1999) found that two hypotheses guide outcome studies of involuntary legal commitment. One hypothesis predicts that involuntary patients will be angry and negative about their hospitalization and treatment. Thus, they will be less likely to cooperate in the hospital and in the community, thus resulting in rehospitalization. The other hypothesis predicts that the initial anger and negativism of involuntary patients will subside and that they will become positive toward their hospitalization and treatment after they receive help.

30.4 Ethical Considerations

A problematic characteristic of ED or SUD is the reluctance of patients to accept treatment even when they are at significant physical and psychological risk. Although these patients typically deny intent to harm themselves, their actions can result in life-threatening medical complications. Severely ill patients may lack the capacity to make competent treatment decisions, and situations may arise when life-saving procedures are justified. There is uncertainty however around when and for whom involuntary treatment may be justified and ethical questions can arise during consideration of compulsory treatment.

In most cases, involving AN or severe SUD, by the time involuntary hospitalization and treatment is under consideration, the patient will have been deemed incompetent and lacking decisional capacity. The consequence of those behaviors directs the healthcare provider or family toward coercion. The perception of coercion in civil commitment is complex and not necessarily related to the degree of restriction of freedom. In thinking about and invoking compulsory treatment, we want to do what is right and good or at least what will be best for the greatest number. In considering civil commitment, there needs to be a balanced, ethically principled approach that respects the dignity or autonomy of the affected person while working to prevent deterioration of the person's health. Respect for personal dignity does not rely on autonomy (allowing patients to make their own decisions) alone, but is also supported by the principles of beneficence (providing care that will benefit the patient), non-maleficence (do no harm), and paternalism (interfering with a person's freedom for his or her own good). We recognize each of these principles as intrinsically ethical or moral. However, at some point, even the most profoundly caring provider may find themselves inclined to violate an individuals' autonomy when the extent of their illness makes it impossible to maintain health. Beneficence and paternalism can be seen as overruling autonomy (Kenney, 2012; Tansey, 2011). As noted earlier, people with AN appear to agree with the necessity of compulsory treatment in order to save life.

The decision to treat involuntarily should be subjected to the most rigorous standards. Primary questions that should be considered are the following (1) Is this an end of life situation? (2) How would the patient benefit from treatment? Will others benefit (i.e., family, providers, community)? (3) How realistic are our treatment goals? (4) What criteria will constitute success or failure? (5) What time frame may be agreed upon for imposing the treatment? (6) What financial concerns are relevant for the patient or family? (7) What is our standard of care? (8) What does the medical literature suggest about the efficacy of involuntary treatment? Each question must be considered by all involved parties (Kenney, 2012).

What ethical principles do we rely upon in this decision? What makes it morally permissible or morally repugnant? Is there a consensus for this decision? It may be morally permissible to force a person with a life-threatening mental disorder into treatment, but why? One idea that has been advanced is that the individual is no longer able to make competent decisions. Although, the individual may suggest

they are making a choice, poor decision making or incompetence will be influenced by impaired self-awareness or an inability to control the behaviors that are characteristic of the disorder or illness. Severe mental illness can lead to an unrealistic appraisal of the implications and consequences of the individual's actions. Respect for persons, coupled with concerns animated by beneficence, non-maleficence, and paternalism, provides grounds for the appointment of a surrogate decision maker that can request involuntary treatment. If they decide to compel another person into treatment, this course of action must proceed with personal humility, cultural humility, professional humility, and in awe of the dignity of the human person (Kenney, 2012; Tansey, 2011).

Of primary concern in civil commitment is the principle of autonomy (Silber, 2011). Related to the individuals control over his or her body, this principle has long been a central concept in medicine. Within the substance abuse literature, there is a view that mandatory treatment involves protecting third parties from extreme health risks or to prevent harm to those that are incapable of avoiding the person with substance abuse. However, when autonomy is compromised by a debilitating illness, there is a need to override autonomy with justified paternalism (Silber, 2011). Caplan (2006) has put forth the argument that respect for self-determination sometimes requires mandatory treatment as a way to create or enable autonomy. The premise is that people who are addicted do not have the full capacity to be self-determined or autonomous because they are caught up in their illness. If this is indeed true, then it may be possible to justify compulsory treatment in order to return autonomy to the affected individual. Caplan (2006) also advances the idea that addiction itself can be a form of coercion, as the person is driven by desires that go on to influence actions and behaviors. Physiological and psychological cravings and withdrawal symptoms are powerful motivators to continue use. If treatment can facilitate change, it will increase that individual's autonomy and his or her ability to maintain health. Maintaining health and the ability to make good choices about their actions in the long run would be the moral thing to do (Caplan, 2006).

A similar idea has been suggested when treating AN (Hope, Tan, Stewart, & McMillian, 2013). These authors propose that the ED subtly compromises autonomy inhibiting a clear understanding of actions and decisions about shape and weight that affect overall health. Looking at the experiences of individuals with AN, it was found that treatment refusal was based on emotional experiences and false beliefs that contributed to an inability to make knowledgeable decisions. As a result, competence and autonomy were significantly compromised. The ED inhibits an individual's awareness of the outcome of their actions which in turn leads to treatment refusal. The refusal of treatment may seem autonomous but in reality is influenced by the disorder. Compulsory treatment is seen as a method to reduce ambivalence about treatment and assist in changing false beliefs about weight, shape, and health while restoring the person's autonomy to make appropriate healthcare decisions.

30.5 Psychiatric Advanced Directives

An alternative to civil commitment or a follow-up after the commitment has ended is Psychiatric Advanced Directives (PAD). This approach can help minimize many of the ambiguities commonly associated with mental health crises (Sarin, 2012; Srebnik et al., 2005). PAD are legal documents that allow competent individuals, through advance instructions, and or designation of a healthcare agent, to declare preferences for future mental health treatment. With a more long-term or chronic disorder, a PAD can be potentially helpful for the following reasons (1) they can empower consumers to assume control over treatment decisions; (2) they can enhance communications about treatment preferences between consumers, families, and treatment providers; (3) they may facilitate appropriate and timely interventions before situations deteriorate to emergency status; and (4) they may lead to reductions in adversarial court proceedings over involuntary psychiatric treatment (Sarin, 2012; Srebnik et al., 2005; Swanson, Van McCrary, Swartz, Elbogen, & Van Dorn, 2006).

Most state statutes presume that people are competent at the time they draft advance directives. These laws generally require the directive to be signed by two adult witnesses who attest to the person's capacity at the time the instrument is drafted. More difficult questions arise over determining capacity at the time advance directives are used for healthcare decisions. In some states, a judge must make capacity determinations. Another controversial issue concerns the potential use of PAD to refuse all treatment. All states that authorize PAD, except Maine, specify that individuals or patients may use these directives to consent or refuse psychiatric treatment (Sarin, 2012; Srebnik et al., 2005)

Laws specifically authorizing PAD have been enacted in twelve states (Minnesota, Alaska, Hawaii, Idaho, Illinois, Maine, North Carolina, Oklahoma, Oregon, South Dakota, Texas, and Utah). These laws establish the right of persons with mental illnesses to write directives, when competent, indicating their wishes concerning acceptance or refusal of psychiatric treatment. An unresolved question concerns revocation of PAD. Clearly, an individual may revoke a PAD when competent. While PAD are quite promising for empowering consumers to more actively participate in their treatment, there are legitimate concerns about these instruments and the possibility of abuse.

One of the thorniest issues concerns determinations of legal capacity or competence. Questions about capacity arise at two different points in the process. First, individuals or patients must be competent at the time they draft a PAD for these documents to be valid. Second, a PAD may be used for psychiatric decisions only when the individual or patient is not competent to make these decisions himself/herself. A more difficult question arises when an individual tries to revoke his/her PAD while actively symptomatic and in need of treatment. The insertion of a so-called Ulysses clause in an advance directive could effectively avoid this dilemma (Sarin, 2012).

This name originated from the Greek hero Ulysses who knew that the song of the beautiful Sirens was so powerful that he would be compelled to sail his ship toward

the rocks thereby destroying it. He ordered his men to bind him to the mast of the ship and to keep sailing straight, no matter how strongly he argued to the contrary. A Ulysses clause instructs treatment providers about specific treatment preferences and explains that any statements made refusing treatment during periods of incapacity should be ignored (Sarin, 2012; Srebnik et al., 2005).

The use of PAD is in its infancy. There are more unresolved questions than answers about these instruments. Ongoing research projects and pending court decisions will provide more comprehensive information in the future. A parallel study with Huntington's disease has been suggested as template for ED (Bisson, Hampton, Rosser, & Holm, 2009). Having easy-to-follow instructions, consistent verbal and written information helped completers of PAD feel empowered by the process (Henderson et al., 2004). PAD may help patients with ED face the seriousness of their condition without diminishing their self-esteem (Sheehan, 2009; Thiels & Curtice, 2009). Until then, PAD must be strongly considered as a way to empower consumers to take an active role in their own treatment and as a way to avoid damaging, divisive conflicts over treatment and medication issues.

Conclusion

Research on the outcome of involuntary treatment for ED or SUD is limited, and long-term follow-up after mandated or "coerced" treatment is needed (Andersen, 2007). Importantly, however, case-control studies of involuntary versus voluntary inpatients treated in specialty programs suggests that discharge outcomes are equivalent for both groups although weight restoration may be slower in involuntary cases (Watson et al., 2000). Furthermore, involuntary patients retroactively judged being detained as justified and helpful (Watson et al., 2000). Although patients with AN treated on an inpatient basis report a stronger sense of coercion (Tan et al. 2003a, 2003b), one inpatient study found that a majority of patients who reported high perceived coercion around the admissions process and denied a need for admission, converted within two weeks of hospitalization to agreeing that they did needed inpatient treatment (Guarda et al., 2007). The data would cautiously suggest value but more research is needed.

Civil commitment is a legitimate tool in emergent situations when an ED or SUD becomes life threatening. Legal interventions regarding court-imposed treatment are reliant on the governing bodies of the state in which the patient resides. Mental health laws outline the steps to be followed and what standards must be met before someone can be ordered into treatment. The need for compulsory treatment standards typically includes qualification for care based on at least one of the following standards (1) the person's inability to provide for needed psychiatric care, (2) the person's inability to make an informed medical decision, and (3) the person's need for intervention to prevent further psychiatric or emotional deterioration. Most clinicians will use involuntary commitment as a "last resort" when patients decline voluntary hospitalization and their physical safety is at risk.

It is suggested that civil commitment be limited to situations in which therapeutic gain is likely from hospitalization (Guarda et al., 2007; Watson et al., 2000). To some degree, the length of civil commitment will depend on how a state's law defines danger to self or others. With the greatest risk of relapse occurring during the first year following discharge from initial treatment, as well as weight maintenance over the next 12 months predicting better treatment response, civil commitment may assure greater adherence to therapy and afford better long-term outcomes (Herzog et al., 1999; Isager, Brinch, Kreiner, & Tolstrup, 1985; Kaplan et al., 2009; Pike, 1998). Given this data, it could be argued that civil commitment should be maintained at least 1 month after the end of inpatient weight restoration. Given the legalities and complexities of involuntary hospitalizations in patients with ED, consultation with a psychiatrist is recommended to help with the assessment of patient capabilities (Gans & Gunn, 2003; Tan, Stewart, Fitzpatrick, & Hope, 2010; Testa & West, 2010). Unfortunately, there are no clear guidelines to determine duration of time when one is committed under substance abuse laws.

Guarda et al. (2007) suggest the high conversion rate in ED inpatients who initially did not believe they needed hospitalization raises questions regarding the ethics of coercive pressure for treatment. However, since ED and SUD are characterized by impaired judgment and treatment resistance and are associated with high morbidity and mortality (Engstrom, Adamsson, Allebeck, & Rydberg, 1991; Herzog et al., 1999), admission may be justified under the medical principle of beneficence. Although controversial there is a role for civil commitment in the treatment of ED and SUD. *Efforts must be made to maintain a positive therapeutic stance at all times. Coercive pressure is justified only by the likelihood that treatment will be beneficial. This likelihood should be assessed on a case-by-case basis*; *it may be higher for a chronically ill patient who has constantly evaded treatment than for the "professional patient" with whom numerous admissions and treatment modalities have failed* (Guarda et al., 2007, p. 112).

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Index

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