RACHAL ZARA WILSON

NEUROSCIENCE for COUNSELLORS

Practical Applications for Counsellors,
Therapists and Mental Health Practitioners

Neuroscience for Counsellors

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Practical Applications for Counsellors, Therapists and Mental Health Practitioners

Rachal Zara Wilson

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For my children and my grandchildren,
who I will love forever,
no matter where or what.

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Introduction

About this book

While there is some sharing of knowledge and research between the fields of counselling, psychology and neuroscience, there is much learning that is relevant to practitioners of counselling that they are not aware of, as it is considered to be outside their domain, and therefore does not currently form part of their training. Some counsellors know that neuroscience learning exists that could be relevant to what they do, but haven't yet connected with it, and others have no idea that there is any connection between what is being learned about the brain in the scientific domain and what they do when they are working with clients. The focus of counselling is very much around talking and thinking, with not much emphasis placed on the fact that all of this originates within the brain.

Because of this lack of awareness in the counselling field in general, most counsellors do not have easy access to the exciting developments that have happened in neuroscience over the past few decades. Consequently, they have not been able to draw on this knowledge in their work with clients, or to integrate the ideas into their practice.

My aim is to place this knowledge within easy reach of counsellors, by compiling much of it in one place, and providing a basis of ideas arising from that knowledge that can be used by other counselling practitioners as well as myself. Some readers may come up with other ideas from the knowledge provided. I welcome that, because no one person can, or ever should, corner the market in ideas.

Because neuroscience is still a field in its infancy, the exciting thing is that as more discoveries are made, counselling and related disciplines such as psychology have the potential to be constantly enriched with

new knowledge that can be interpreted into the languages of our respective disciplines, and made relevant to what we do.

Joining knowledge from multiple fields of learning together, and interweaving what we know, will create a more holistic way of working for practitioners of all disciplines. I am always reminded of the story about the elephant and the blind people. Four blind people are in a room, and each feels a different part of the elephant. The first describes it as short and thin like a pencil, and wiry at the end. They are holding the tail. The next describes it as broad and flat, and triangular in shape. They are holding the ear. The third describes it as huge and hard and rounded, the fourth describes it as a long cylinder, and very flexible. They have the stomach and the trunk respectively. The point of the story is that they are all feeling the same elephant, but each is focusing on a different part, and none sees the big picture without further exploration.

My objective is to provide a bit more of that big picture and to help counsellors to explore a bit more of the 'elephant', which is one of my metaphors for the brain and its workings, and to help them understand how integral it is to what we do.

I have conducted a literature review comprising texts from 1985 to the date of completion of this book (late 2012). Some texts that have direct and seminal relevance to the topic, and that have been published before 1985, have also been included. Where possible, more recent literature takes precedence over older literature. This is a field where new discoveries often render older knowledge obsolete, so the most up to date readings have been given the most weight.

The review was focused on brain plasticity, as this is a process that makes change possible; change at structural and neural levels in the brain, and therefore change at behavioural, emotional and cognitive levels in the person. The capacity for change is bidirectional, in that change at environmental level and personal level can also lead to greater plasticity. Much of counselling is about facilitating change in life, whether that be through insight, or a change in behaviour or functioning. People come to counselling because they want support to behave differently, or to think or feel differently. The potential for change provides hope, and hope for change is a powerful force in counselling. It is what brings our clients to us.

Each section will include the main learnings from my literature review, and will be followed by some of the implications it is possible to draw from this data which have relevance for those working in the field of counselling, hopefully providing some new and challenging ideas for counsellors to integrate into their existing practice.

Personal factors

My reasons for being interested in neuroscience are personal. I've always been excited by the potential of the brain, in awe of the amazing things that it can do, and full of wonder at the mysteries of its function, especially the things for which we have no explanation yet. But I am also mindful of the mental pain which is felt by those whose brains are experiencing dysfunction.

I have three children, all of whom I love very much. Although they are much more complex than the following thought would suggest (and as they grow, they belong more and more to themselves and less and less to me), I often think of my youngest child as my social butterfly, my middle as my ambitious thinker, and my eldest as my special project. This is because, despite a high IQ, my eldest has suffered a number of mental and behavioural difficulties, which, as I began this work, were stopping him from fulfilling his full potential and from leading the life that he would choose to lead if he could.

His difficulties could be summarized as a lack of emotional resilience (and an inability to cope with even very small setbacks); extreme anxiety (which prevented him from leaving the house, going outside, going into shops, attending courses, appointments or holding down a job); difficulty making eye contact; a lack of impulse control, motivation, organization, planning skills, and emotional regulation (the last leading to seemingly uncontrollable behavioural meltdowns, especially when anxious); sensory sensitivity to noise and smell, and sometimes taste; and an apparent inability to tell the difference between truth and lies when he spoke them.

He was inflexible with his plans and could not change them without at least a day's prior notice. He could not multi-task or pay attention to more than one stimuli. He reported his brain as being 'not switched

on' in the mornings, and sometimes not for the whole day. He was intellectually vain, and could not be disagreed with. The meltdowns included much swearing and name calling, mostly irrational. He combined this with depressive tendencies, and before medication, also with some internal rituals of thought suggestive of Obsessive Compulsive Disorder (OCD), such as counting every roadside marker he passed while he was driving. Driving was a favourite pasttime and he was always wanting to go somewhere. He often fidgeted and paced up and down.

Until recently, he managed to exacerbate all of this with a major cannabis addiction. He felt it lowered his anxiety in the short term, and helped him to focus and see things clearly. He has always had a problem with attention and concentration. Unfortunately, what helped in the short term aggravated mental vulnerabilities in the long term. Combined with impulsivity, addiction led to stealing and lying and added to his existing obsession with money. Post addiction these three behaviours are still not entirely extinguished, although the stealing is significantly reduced, possibly due to greater availability of money as it is no longer spent on the addiction. He has a special interest in watching rugby as well as money.

Medication has improved some of these things and has probably led him to the point where he has been largely able to overcome the cannabis addiction. Other small improvements kept me hopeful. The journey through the mental health system has been a long one, and we have seen nine different psychiatrists, receiving variable diagnoses from each.

These included, in no particular order, Attention Deficit Disorder (ADD), Attention Deficit Hyperactive Disorder (ADHD), Aspergers, OCD, sensory sensitivities, dyspraxia, social anxiety and depression. We never saw a psychologist, so Oppositional Defiance Disorder (ODD) was not suggested, but it could easily have been if we had done. Some psychiatrists disagreed with some of the diagnoses, others added new ones, and each concentrated on the angle they believed to be most relevant. (That elephant again…) Medication was started, withdrawn and changed. Some medications promised so much and then had no effect past the honeymoon stage, which could be ascribed to a placebo effect, or to my son not responding to the medication after

his body adjusted to it. What he was taking as I began this project was helpful, but so much still remained to improve.

I am a counsellor, so I need at this point to list some of my son's strengths as well as his vulnerabilities. He is loving when not enraged, generous, very intelligent, good at maths and electronics, enjoys music and cooking (both of which he is talented at), and wants to be the best he can.

My choice of subject was very much inspired by a book about the plasticity of the brain, called *The Brain that Changes Itself* (Doidge, 2007). This book gave me hope for change and I found that very exciting. Whatever was happening in my son's brain has had a major impact, not just on him and his partner, but also on me and my other children, and the belief that things can be different was, and is, a wonderful thing to hold on to.

A field in its infancy

When I first started this journey with my son (in his late teenage years, because until then I had put his behaviour down to 'naughtiness' that he would grow out of, as so many parents do) I thought that all we would need to do to solve our problems was to see the psychiatrist and they would know all the answers. Sadly, this has proved to be incorrect. This does not mean that psychiatrists are not very clever people. Plainly, they are, and they have completed many years of study to get to become psychiatrists. What it really indicates is that the field of neuroscience, and the understanding of the brain, are still in their infancy (Doidge, 2007), much like the study of human anatomy, disease and dysfunction of the body was a few centuries ago when we used to let blood to try to improve a fever, and gave alcohol for shock. The latter practice was done until quite recently, because even in that field, new learning is happening all the time.

Nor does it indicate that there is no knowledge base in neuroscience. Anyone conducting a literature review such as I have done as research for this book will find that there has been a huge amount of study, with a huge amount of knowledge and data arising from that study. But because the brain is so fantastically complicated, what we do

know so far is but a drop in the bucket of knowledge still left to understand about its workings. Finding out more is always hampered by the technology that we have to study the brain with, and the ethical considerations of learning more about an internal organ that is at once incredibly sensitive and yet vital for our survival.

As I began my study, the complexity of the field became apparent to me. I had to pause to go through some text books on basic neuroscience, and my lack of a background in chemistry and complicated mathematical equations was a difficulty. I kept on, because I decided that detailed chemical and mathematical explanations were not going to be necessary for the readers of this book (hopefully many of them will be other counsellors) because what will make the knowledge that I intend to share with them accessible is to put it into the language of counselling. (A glossary of technical terms relating to the brain and some basic explanatory diagrams are available at the end of this book, as labelling of parts to make the information clear was in many cases unavoidable.)

Integration of complexity

One of the more helpful texts on basic neuroscience was an attempt to integrate neuroscience with itself, so that both the right and left hands know what the other is doing (Gordon, 2000). The brain has so many levels, both micro and macro, that once again each scientist or group of scientists has a different part of the elephant, and is focusing exclusively on the bit that they are examining. Much of the knowledge that is gathered is based on likely hypotheses, because even observable data can not necessarily be easily explained, and in many cases there could be multiple explanations for what is observed. Some of what I present as neuro-scientific discovery will be based on these hypotheses, which is where current scientific data are converging to indicate likely truths. To use another long-standing and well known metaphor, if we see only white swans for a hundred years, we begin to believe that all swans are white. But it only takes one black swan to disprove this hypothesis, and so it is for research into the brain.

It only takes one piece of new information to cause us to re-evaluate or widen a current hypothesis.

What I present will be gleaned from a multitude of methodologies, including a variety of types of imaging studies, direct electrode measurement of living brains, studies of both animal brains and the human brain, autopsy data, physiological tests (e.g. blood, urine), genetic sampling and experiments, and laboratory work. Brain tissue remains functional for some hours after being removed from its subject and recent advances have allowed animal brain tissue to continue to function outside the brain while still connected to the live animal (Colicos and Syed, 2006).

Where does counselling fit in?

The field of psychology has always been very active in researching the brain, albeit from the outside by studying the behaviour, thoughts and feelings which are expressed. It is a natural partner for neuroscience in that neuroscience complements psychology by studying the brain from the inside. Indeed, a 'new' field, a combination of psychology and neuroscience called cognitive neuroscience, has sprung up because the field of psychology – perhaps because of its focus on research and learning – has been quicker than the field of counselling to realize the potential that a greater understanding of the brain can bring to those clients we work with. (Often it is a matter of luck as to whether a client is referred, or self-refers, to a psychologist or a counsellor. Not many members of the general public could provide a helpful definition of the difference between the two if asked.) The Achilles heel of counselling has perhaps been a much slighter research base and a narrow view of the part of the elephant on which it is focusing, for example, how primarily talking therapies affect behaviour, emotion and cognition.

Counselling, however, is in a really good place to use the new information provided by the fields of neuroscience and cognitive neuroscience. Neuroscience, as well as being conducted for the sheer pleasure in the discovery of new knowledge, is mostly focused on providing answers to neuro-dysfunction by seeking solutions in the form of medication or structural alteration (surgery, electric shock

treatment, etc.). Psychology has its invaluable research contribution but seems primarily focused on 'fixing' people, from the position of an expert, when working with clients. Neuroscience and psychology are beautifully suited to learn more about 'how we work' in tandem with each other, but counselling is best suited to helping people to make use of this knowledge to build the lives they want to live. Counselling at its best involves clients being supported to make their own change. Change, when the goals are the client's own, is more likely to be long lasting and sustainable. As I will discuss later, forming new pathways in the brain is dependent in part on repetitive practice, and this will only happen if the goals for change are those of the client.

Among neuroscience, psychology, counselling and related practices, each discipline has strengths and vulnerabilities. Each is only a small part of the whole elephant. I believe that with the bidirectional sharing of the knowledge from the other disciplines, counselling will be strengthened and have more to offer those who are clients of its services.

Kandel (1998) says that in the near future, it will be possible to use imaging studies to see if and how talking therapies have made a positive difference to brain dysfunctions such as depression, and that structural change necessary to remission will be measurable. This is both exciting and scary: exciting to be able to measure the effectiveness of our work, and scary in that when counsellors are not making a difference, this will also be measurable. Many ethical questions will arise, and these are outside the scope of this work. Naturally, some problems will require a combination of medication and talking therapies, and some dysfunctions may be more amenable to medication alone. In some cases, the efficacy of talking therapies such as counselling may render medical intervention unnecessary. Already it has been shown that there seems to be equal efficacy in the treatment of OCD between current medications and talking therapies, as measured by functional magnetic resonance imaging (fMRI) scans (Kandel, 1998).

The talking and listening functions which allow a client to tell their story and be heard in counselling will of course never go out of fashion. These form the basis of many other functions in counselling: information gathering, validation and encouragement, reframing, and other therapeutic interventions that will be referred to over the course of this book.

Classification systems

New knowledge about the plasticity of the brain and its overarching effects into the function and dysfunction of the brain are leading some to call for an entirely new classification system for brain dysfunction (Peled, 2004). Many of the criteria and classification systems that we have now seem more and more arbitrary, and the significant co-morbidity between many conditions, as evidenced in my son, shows that it is unlikely that neat little boxes exist in the brain for each of the current diagnostic conditions. It appears that it is more likely that brain dysfunction, when it is not the result of physical damage to the brain, is a result of the malfunction of plasticity (more about the plasticity of the brain will be discussed soon) with significant overlap between the types of symptoms possible, dependant on which areas, or combination of areas, are primarily affected.

It is not that long ago that our system for classifying all living things was put to the side, and a new one developed, more able to incorporate and account for new learning about life on this planet (when Linnaeus's taxonomy replaced Tournefort's in the 1700s). When it becomes apparent that something has outlived the usefulness it once had, it is sensible to redesign it to create something more appropriate.

Making a difference

One of my hopes, going into this investigation, was that I would learn some things that would make a difference to my son. After all, this is my child's life, and nothing could be more important to me than my child having the best life that it is possible for him to have. My conclusion will discuss which parts of what I have learned have been helpful for him, or may prove to be so in the future.

Some of the knowledge here will assist counsellors to become better counsellors, which is not my role with my son, but what I have learned will have a significant impact on my own practice. Some of the knowledge will provide counsellors with information to share with clients regarding what is possibly happening neurally for them

(so that they can explore this with relevant practitioners in the case of brain dysfunction) and what they can practically do.

As well as the sections on how it all works (from a white swan perspective) and counselling techniques that can be helpful, I have also included a section towards the end on what we can recommend, as there are definitely ways for clients to take control of providing optimal conditions for their own brain plasticity. After all, people come to counselling for what is typically a short period in their lives. Anything we can do to assist them to live in optimal conditions and to maintain this throughout their lives is a good work by us. I am indebted to a counselling trainer of mine who once told me, 'anything that we do to put ourselves out of a job, because our clients no longer need counselling, is a job well done'.

The challenge of new knowledge

Jargon, or specialized language, is one of the things that gets in the way of the comprehension and/or the uptake of new knowledge. Because my aim is to make knowledge from one field (neuroscience) widely available to other fields of talking therapies (especially to the field of counselling) I have provided a key to this jargon where I was unable to avoid its use in the form of a glossary of terms, located at the end of this book. Reference to this list of definitions will hopefully facilitate fuller understanding, as it was not possible to redefine each word every time I used it if I wished to be concise, focused and reasonably brief in my explanations.

One of the texts that I reviewed spoke of how, every time it arose that practitioners (in that case teachers of education) were taught new knowledge, they would feel a sense of frustration that what they had been taught prior was now obsolete, only to be replaced by the next new thing, which in turn would be replaced in the same way (Wolfe, 1998). The text was about new knowledge about learning and how best to teach effectively, and the author showed how new knowledge builds upon the old, and how, just because we now have a black swan, it doesn't make a hundred years of white swans invalid. The black swan merely adds more richness and complexity. Sometimes the new

language around the new explanation makes it difficult to realize that the white swans are still the foundation on which the new explanation is built, but if you look carefully, there they are, forming the basis. The colour of the swan may be different, but the shape is still the same.

And so it is with the integration of what neuroscience and cognitive neuroscience will add to counselling. Some things will be but new explanations for old things, with the new knowledge confirming or adding to what we already do as counsellors. Occasionally, we may find ourselves challenged because an assumption that we have based practice on turns out to be false. Then we have a choice of continuing with what is comfortable and rejecting the new knowledge (and becoming obsolete and irrelevant as practitioners, or as a field in general) or taking that leap and doing things differently, taking it on board and increasing our efficacy.

Mostly we will find that what neuroscience does is to add to what we know and to show us different parts of the elephant, helping us build a more complete picture of it.

Those counsellors who are narrative focused will be interested in, among other things, the ideas around memory and re-storying discussed later in this book, and why they might work. Those who practise Neuro Lingual Processing (NLP) will gain further insight and ideas for their modality, which is primarily about connecting behaviour with the functioning of the brain anyway. Cognitive behavioural counsellors will do likewise, and some of the whys and wherefores of Cognitive Behavioural Theory (CBT) might become apparent and/or enriched by scientific validation. (It is sometimes tempting to conclude that even within our own discipline, we are often feeling different parts of the same beast.)

I am indebted to the many academic trainers who have worked with me over the years of my counselling journey, and the course presenters who have drawn my attention to new techniques and topics of interest. They are where I have drawn the basis of what I know about counselling from, so that I have been able to suggest new uses for it here, and adapt it to suit what I have learned from my research into neuroscience.

Some of the things that we do, which we know from our own experience work for clients, may still not have an explanation provided here. In those cases, we are still waiting for the black swan to appear

that will provide scientific context. And long after we get used to both black and white swans, there will be further swans of gold and silver and pure diamond, all waiting to be discovered, because the process of the accumulation and integration of knowledge is a never-ending one. The brain is a wonderful thing.

A cultural note

I was once part of a group learning about brain development in infancy and early childhood in New Zealand, where a Maori kuia (female elder) sat and listened and said at the end, 'so it's all about the brain now, is it?' Well, yes it is, but it always has been. Once again, the languaging of explanations can keep us from realizing that what is being spoken about is actually the same old thing, with new words around it to give us fresh understanding. When Durie (1994) developed his Te Whare Tapawha model (translated as the Four-Sided House), which is widely used among Maori in the helping professions in New Zealand, the hinengaro, or the mind, was one of the four cornerstones. The tinana (the body) is intimately connected to the brain. Wairua (spirituality) is perceived through the brain. Tinana and wairua are also key cornerstones of Te Whare Tapawha, with whenua (land) being the fourth representing the environment, which has an important impact on the brain's function.

Lagona is a Pasifika concept of a sixth sense, or feeling of knowing about others, used as a basis for interventions by Pasifika social service workers (Dalhousie, 2010). Neuroscience can provide a greater understanding of this, as discussed in the section about mirror neurons and how they help us to understand the feelings and intentions of others. And in India, Yogi and their followers have long practised the art of altering, or attempting to alter, their brainwaves so as to attain a different and more blissful state of consciousness.

The current neuro-scientific research pertains to everybody. There are some differences in the gene pools of different ethnic populations (Polednak, 1989), but brain function/dysfunction and brain structure are common to us all. The new learning being done now is by scientists from a wide variety of ethnic backgrounds and is being carried out

globally. What is being discovered is new even to first world scientists. The challenge is to be able to put the language around it to make it relevant to our own cultures. What we have will still be the same, but richer in explanation.

Interconnection

While writing this book, it was apparent to me that the workings of the brain are intimately connected with each other, and my difficulty was to create a linear document from a narrative that repeatedly circles back on itself and has connections in all directions, much like a complicated interconnected assembly of neurons (more on this shortly as well). Consequently, there will be times when sections overlap with each other. Rather than explain everything twice, three times or more, the reader will be directed to the relevant section if they immediately require more information on that particular topic, and they should feel free to skip back and forward between sections as they please.

(A reminder: an alphabetical glossary of terms is provided at the end of the book to promote clarity without constant re-explanation in the main body of the text.)

Chapter 2

Plasticity and How the Brain Works

Plasticity is one of the newer discoveries of neuroscience, and it changes our concept of the brain and how it works. It also changes our ideas about what is possible when the brain is working in a dysfunctional way.

Plasticity and BDNF

What do we know?

First, some very basic neuroscience, for which much more detailed and complicated explanation is available from other sources (Haines, 2006; Kandel, Schwartz and Jessell, 1995; Squire, 2008). Neurons are one of the two main classes of cells in the brain, the other class being glial cells. Neurons are the signalling units of the nervous system. There are different types for example, sensory neurons, motor neurons, and interneurons that perform a relay function. Neurons have tails, called axons, with dendrites extending from the axon like branches of a tree. Axons vary in length from very tiny to almost a metre long. They are typically sheathed in a substance called myelin, provided by the glia. Every neuron has thousands of connections with other neurons across small spaces called synapses. Communication across the synapses can be either chemical, through the secretion and reception of a variety of neurotransmitters, or electrical, via electrical charge from the head of the pre-synaptic neuron to the dendrites of the post-synaptic neuron

that polarizes or depolarizes the next neuronal cell. (These are called action potentials.)

Plasticity comes from the Greek word *plaistikos* (Johnston, 2003). In the term 'brain plasticity' it refers to the recently discovered ability of the brain to change itself and to adapt to its environment by weakening or strengthening different synaptic connections (Ben-Shachar and Laifenfeld, 2004). Hebb (1949) was the first to conceive of networks of 'cellular assemblies', or neuronal connections, which, when used repeatedly, formed preferential pathways in the brain. If key pathways are blocked or we chose not to use them the brain is able to recruit new neurons and form new pathways (Doidge, 2007). Plasticity is the brain's capacity to make change to these networks in order to learn, to remember, to forget, to recover and to restructure (Johnston, 2003). Plasticity is therefore the basis of adaptation and the key to human survival and evolution. A dysfunction of brain plasticity at any level causes an inability by the brain to change or adapt to environmental or internal circumstance.

The entire brain has plasticity (Doidge, 2007), even the spinal cord and parts related to physical function such as breathing. Plasticity is responsible for the uniqueness in each of our brains. The environment we exist in changes the structure of our brain as our brain adapts to that environment (Doidge, 2007; Kandel, 1998; Kolb, Gibb and Robinson, 2003; Lipton, 2005). Things such as diet, stress, disease, addictions, ageing, pollution, the amount of sleep we get, and the social and cultural practices we are exposed to all have an effect on our brains.

Doidge (2007) describes in his book how choosing to live differently according to culture has been documented as being able to subtly change the structure of the brain so that the way in which vision is processed can have a different outcome, producing new visual abilities, and sound is filtered in new ways in order to pay attention to a different set of aural stimuli. What we pay attention to as a result of repetitive cultural practice changes our brain structurally, so that the way in which we perceive the world is different.

And if behaviour or cognition changes, the brain changes too (Kolb *et al.*, 2003). Richer environments build denser brains with more connections (Fields, 2005; Kolb *et al.*, 2003). The converse is true for impoverished environments. Internal changes such as hormonal fluctuations also affect our brain (Kolb *et al.*, 2003). Input changes

the structure of our brain, and so does the output we generate. Even the way our genes are expressed can be altered by experience (Doidge, 2007; Kandel, 1998; Kolb *et al.*, 2003; Lipton, 2005) as will be discussed in further detail later in this book. (See also the section on gene transcription later in this chapter.) Every time our brains change in any way, it affects all other parts of our brain, so that the change is dynamic and ongoing.

BDNF is the common name for brain derived neurotropic factor. BDNF is a neuronal growth factor that plays a crucial role in plastic change in the brain by maintaining the synaptic connections that are important for providing the connections between the neural pathways (Cotman, Berchtold and Christie, 2007; Cotman and Berchtold, 2002; Doidge, 2007; Gomez-Pinilla, 2002; Trejo, Carro and Torres-Aleman, 2001). It is released when neurons fire together, to consolidate the synapses between the neurons so that they fire together reliably in the future. It also promotes the growth of the myelin coating of the axons, which speeds up the transmission of the electrical signals.

Low levels of BDNF have been implicated in many brain dysfunctions, such as depression, Bipolar Affective Disorder (BAD), eating disorders, epilepsy, schizophrenia, addiction, Alzheimer's, Parkinson's disease, and many more (Gonul *et al.*, 2005). Healthy levels of BDNF help to regulate the survival and differentiation (specialization) of neurons in both developmental stages and in adulthood (Gonul *et al.*, 2005).

There are thought to be two overlapping stages in synaptic plasticity. The first is related to early development and the unfolding of genetic template. The second stage is directly related to experiential learning and the manner in which our genes are accordingly transcribed (Kandel, 2000). It has also been discovered that new neurons continue to form in the dentate gyrus of adults, a region of the hippocampus, which is part of the prefrontal cortex (Eriksson *et al.*, 1998; van Praag *et al.*, 2002).

Synaptic plasticity represents lasting changes in synaptic connection. Neuroplasticity is slightly different in that it can refer not only to synaptic plasticity, but also to variations in neurotransmitter release, the shape, health and ability to function of neurons, and the generation of new neurons. These changes can be of short duration or long lasting (Schloesser *et al.*, 2008).

What does this mean for counselling?

It was not so long ago that it was believed that the functions of the brain were set and that each part performed a specific function, and that apart from specific periods of development in the younger years and decline in the very later years, the brain remained unchanging (Doidge, 2007).

Plasticity is hugely exciting for counsellors, and for anyone who wishes to do differently, think differently or feel differently. Provided that brain plasticity is optimal, the potential for people to make change in their own lives is huge. By changing their behaviour, and/or their environment, by learning new things or by choosing to think differently, they can change their own brains, which in turn affects everything else about them and their lives.

We all know that each moment, each year and each decade we are not exactly the same person as we were before. How we feel and who we are at 4 is not the same as how we feel or who we are at 21, which is not the same as how we feel and who we are at 40, and so on. Each moment, year and decade builds upon what precedes it, and we are richer for it (although maybe a little less innocent!).

Of course, even though brain plasticity is a new concept in terms of what we know, its function will have always been so. But the knowledge of this possibility will provide a gateway of hope through which people will realize they can walk. Without knowing that the gateway is there, many people will not have realized that the opportunity exists to pass through it and will not have taken the steps that they need to, to produce the desired change.

Some people will not have the benefit of the optimal brain plasticity required to make use of this. Depending on the cause, there are possibilities for some to take action to increase the plasticity of their own brains, so that the opportunity to make change is open to them too, and this will be discussed later (see Chapter 6).

Supporting clients to achieve their goals toward change is one of our key functions as counsellors. Knowledge of plasticity gives us hope to share with our clients, and knowledge of how to maintain or gain optimal plasticity is one of the ways in which we can support our client's journeys through life, so that they do have the capacity to learn, grow, recover, change, adapt and remember.

Neurons that fire together wire together

What do we know?

When Hebb conceived of his cellular (or neural) assemblies (Hebb, 1949) he also gave birth to the idea described by the adage, 'neurons that fire together wire together' (Shatz, 1992, p.64). By this he meant that when neurons form complex interconnected pathways, joined across synapses, and those pathways that are used repeatedly form a set of strengthened, preferential pathways when a neuron fires habitually at the same time as another neuron, those neurons also form their own connection. When the first neuron is fired in response to an action, a thought, an emotion or maybe an external trigger, it excites the companion neuron or neurons, which fire almost simultaneously. A closely related concept in psychology is 'conditioning', where it has been observed that a stimulus can produce a conditioned behaviour when it is paired with a reward (positive reinforcement) or punishment (negative reinforcement) (Kandel, 2000; Klopf, 1988; Pine, 2007). Neurons that are wired together and fire together do not necessarily require an external stimuli, for instance, the thought 'nobody likes me' could be paired with another thought of 'I'm a loser'. The thought 'I'm all alone' could be habitually paired with an emotional state experienced as sadness, or in a different person, with an emotional state experienced as joy. It is probable that conditioning reflects one aspect of this neural process.

What does this mean for counselling?

Clients (and counsellors!) often experience this phenomenon when they habitually engage in old behaviours or thoughts that have outlived their usefulness. Some examples might include eating all evening while watching TV, compulsively smoking while drinking alcohol, or feeling upset when we hear raised voices, even when those voices are nothing to do with us.

Now that we are aware of the plasticity of the brain we also know that even though the pathways between some neurons, or groups of neurons, have been preferentially strengthened through repetitive use, the opportunity is there to create new pathways. Doidge (2007) likens

the plasticity of the brain to plasticine, which has the ability to be remoulded. Another way of looking at it is to imagine a well worn pathway through the bush. It is easy to walk down because it is wide and clear and we know it well. If we want to go another way, we must push aside undergrowth, squeeze past obstacles and experience that unsureness about whether we are going in the correct direction. If we persist, and continue to take the new pathway over and over again, we become more confident and our constant treading clears the way, trampling undergrowth and obstacles. As it becomes the pathway of choice, the former pathway falls into disuse, although it will always be there, easily able to be cleared again if we choose to switch back to it, because some trace of it will always remain.

And so it is with brain connections. When we are in the habit of using certain neural pathways, using new ones can feel different and wrong and may take a lot of mental effort. It is easier to take the old pathway, even if it takes us somewhere that we don't really want to go. But with time and practice we can form new pathways that become just as easy to use as the old one was.

A lot of clients engage in old behaviours or thought patterns because it is easier, or it 'feels more normal', such as the woman who continually chooses abusive partners because it seems more normal that love between two adults be paired with abuse. When she meets a nice man, it 'just doesn't feel right', and the relationship ends or never begins. Abusive relationships that she forms continue despite her unhappiness, because she is following her well established neural pathways.

A first step in counselling, if the client expresses a wish to change a behaviour, a cognition or a feeling that they wish to be rid of, is to discuss the pathway metaphor with them, providing hope that change is possible but acknowledging that forming new pathways is hard, especially when we already have a strongly marked pathway in another direction. It will feel 'weird' and 'different' to be taking a new route. Being aware of this prepares the client for these feelings, and gives them a chance to choose their response according to how much priority they give to their goal for change. Change is possible even after previous failure. Narrative counsellors will know that sometimes clients have already unknowingly begun the process of creating new pathways and just need help to identify where this has happened. It is

easier to use a pathway that we have already successfully navigated even once before, despite the fact that it is still largely unfamiliar in comparison with our usual one.

The counsellor and the client could then discuss together which internal neuronal pathways have become strongly linked during the course of the client's life. Has going to the cupboard for food become associated with comfort? Has looking in the mirror become linked to feelings of self-hate?

In the section on learning and attention, I will discuss how new learning is stored in the brain through mass repetition and noticing/paying attention to that learning. The repetition of new ways of thinking, behaving or feeling will be familiar to CBT counsellors as client 'homework'. The noticing of the new way of being will be familiar to narrative counsellors as 'thickening the preferred story'.

Clients will need to practise their new behaviours, thoughts and ways of feeling in order to create the new, desired pathway, which could be planned together with the counsellor. What would they rather be doing, thinking or feeling (a question often asked of clients by solution focused counsellors in the form of the 'miracle question')? It is not enough to look wistfully at the possible new pathway. To make it a reality the pathway must be trodden as often as possible.

Because the client's current neural pathways are often strong, it is important to have a replacement neural connection to practise, for example, having a hot shower when arriving home instead of walking straight to the cupboard to get food. Rather than just blocking the old pathway, a new pathway is then available to tread. Changing entire neural patterns can help too, such as not watching TV while getting used to eating less in the evenings (and reading, gardening, sorting through photo albums instead) or not looking in the mirror at all while practising new thoughts or feelings about self-image.

It is probable that some of the co-wired neurons form part of our procedural memories, where automatic behaviour is stored in the form of neural shortcuts such as walking, changing the gears of the car while driving or brushing our teeth (Klein and Lax, 2010; Wolfe, 1998). (More about procedural memory can be found in the section on memory in Chapter 3.) In most circumstances, we don't have to think about how we do these things, we just do them. We can do them while our brain is engaged with other things, and the automatic

nature of the skills stored in our procedural memories frees our brains up to do those things. Pathways that form part of our procedural memories will be particularly hard to change, but we can and do learn to drive cars with different gear systems, or to walk in high heels. An initial burst of effort and repetitive practice is required, while thinking hard about the new skill. Until eventually it becomes the 'old' skill, and a new effortless pathway is formed in the brain thanks to the opportunities provided by plasticity.

And what of the woman who always chooses abusive partners? She can practise experiencing love with tenderness and support rather than abuse. This could relate to ending an abusive relationship, and practising providing these things to herself, seeking them from friends or family, and/or choosing only to continue in any new relationship that provides this. It is important to unpack which neural connections are contributing because there may be underlying connected neural wiring, such as between being alone and feelings of rejection, leading her to choose an abusive partner over no partner at all. If this was the case, practising new pathways for being alone would be the more relevant learning.

The modes of counselling that we already practise as counsellors are all suitable for supporting clients to develop new neural pathways and neural connections. The knowledge about how neurons that fire together wire together complements what we already do.

Plasticity/rigidity paradox

What do we know?

The flip side of brain plasticity is rigidity. There can be many reasons for this. Ageing brains typically show less plasticity (Doidge, 2007; Manji *et al.*, 2003). But because everything we input into our brain has an effect on our brain, we can keep our ageing brains in a more plastic state by continuing to learn new skills as this encourages the brain to stay supple, so to speak, and to continue to produce the trophic factors that it needs, such as BDNF, to support this continuing need for plasticity (Doidge, 2007). Keeping up complex skills already learned

does not promote plasticity, as plasticity is only required for change and adaptation. Someone who does a complex job and masters it does not require continuing plasticity, because the neural pathways required to do that set of skills are already set down in the brain (Doidge, 2007). Younger brains are more naturally plastic, because they could expect to be continually learning new skills.

There are dysfunctions of the brain that are likely to involve dysfunctions of plasticity. Schizophrenia, depression and bipolar affective depression, and autistic spectrum disorders (see Chapter 5) are among those that have been hypothesized to be related to a dysfunction of brain plasticity. These hypotheses have arisen as a result of recent neuro-scientific research. Often these pathologies are characterized by a rigidity of thinking, notably in conditions such as Aspergers, where those affected are unable to be flexible and are happiest when everything can be predicted and follows a set routine. Psychiatric disorders are correlated with localized changes in neural circuits (Kolb *et al.*, 2003).

There will be those who are not in a position to take advantage of the exciting potential of plasticity. Having said that, in his book, Doidge (2007) recounts the story of a woman considered to be retarded, who, with determination and practice, taught herself new mental skills until she overcame all her learning disabilities. And there are environmental changes that can be made to increase and/or facilitate brain plasticity, including increased exercise (Cotman *et al.*, 2007; Gomez-Pinilla *et al.*, 2002; Trejo *et al.*, 2001), good sleep patterns (Hobson and Pace-Schott, 2002), and taking dietary supplements such as Omega 3 (Kitajka *et al.*, 2004) (see the later sections on what we can recommend).

What does this mean for counselling?

It is important to be aware that some people will have more barriers to optimal brain plasticity than others. In terms of our pathway metaphor, some people will have a significant disability when it comes to creating new pathways, comparable perhaps to having no legs and needing to clear an actual pathway in the bush. This does not necessarily make a

new pathway unobtainable. The process may take longer and resources may need to be recruited to make it happen, such as prosthetic legs.

Because the barriers can be significant, it is up to each client to decide if the effort required is worth their while. Some clients may not have the ability to understand that they have the capacity to take a different pathway if their brain is significantly impaired. (In our pathway through the bush metaphor, they might be visually blind.)

For those that do have the ability to comprehend the possibilities of plasticity and new neural pathways, counsellors need to encourage: a) keeping the brain in a state where it continues to be constantly stimulated, to encourage it to remain in a state of plasticity (internal input); and b) taking the steps required to provide the brain with the optimal environment to achieve plasticity (external input).

Damage to the brain

What do we know?

Damage to the brain has provided neuroscience with the opportunity to find out more about the workings of the brain in general. Damage to a specific part is known as a 'lesion'. There have been many cases where people have suffered damage to the brain and managed to rewire neurons from other parts to perform the function of the damaged part (Doidge, 2007). Doidge gives the example of a woman who was born with only the right side of her brain but who manages to function within normal range. Plasticity is at its strongest in the young because they are still progressing through developmental stages and what are known as 'critical periods', where the brain typically develops specific skills, such as walking, talking or being able to perceive colour (Doidge, 2007).

Plasticity provides the brain with the opportunity to recover, by strengthening or weakening neural connections to change or restructuring existing pathways. Recovery in stroke patients (Doidge, 2007) shows that with determination, even adults can take advantage of plasticity and practise forming new pathways through repeating the lost skill over and over (sometimes by taking one small part of that

skill and practising it until becoming ready to go on to the next step) until the brain forms new neural connections for the old task.

Once again, where possible, providing optimal conditions for brain plasticity contributes to the brain's ability to recover and restructure itself. In some neuropathologies or dysfunctions of the brain, there is some evidence that the longer the dysfunction is present, the more structural damage this creates in the brain.

What does this mean for counselling?

Where effective medical treatment options exist for mental health issues, counsellors should discuss with clients their own options for referral to practitioners, such as psychiatrists who can give the client more information on what is available to them. Naturally, it is a client's right to make choices on their own behalf, but knowing that early treatment is likely to better protect the structure and plasticity of the brain is also a client's right.

For those working with children, it is also important to understand that the best hope of recovery from damage to the brain is as early as possible, to take advantage of greater plasticity in the early years, whether this is done through a referral to occupational therapy or through engaging in supporting the client to build new pathways by rerouting existing neurons.

For all clients, and this will be a theme throughout this book, making sure that the external environmental conditions are in place to promote optimal plasticity is key to any recovery that might take place.

We also need to acknowledge that sometimes recovery will be slow or non-existent, either because of the extent of the damage, and/or because of a failure of plasticity. As counsellors, it is likely that those clients who fall into this category will be working with practitioners from other professional groups as well as us.

Gene transciption

What do we know?

Long term plasticity in the brain requires changes to gene expression (McClung and Nestler, 2008). Kandel (1998) believes that the idea that our biological processes (including behaviour and cognition) are determined by fixed genetic inheritance, and that we are therefore powerless to change them, has led to a reluctance to incorporate brain science into social science. Practitioners of talking therapies, such as counselling must believe in the possibility of change or we would be redundant.

The view of genetics as heritable, fixed entities is only part of the story. There are two different functions of genes. One is the template function (Doidge, 2007; Kandel, 1998). This is the function that most people associate with genes, where genes are transmitted from one generation to the next, and are copied from a fixed template. The second function, known as gene expression, is regulated by environmental factors (Doidge, 2007; Kandel, 1998; Lipton, 2005). Gene expression does not affect the template function of genes (Kandel, 1998). Twin studies show the importance of both the template function and the expression of genes (Kandel, 1998).

Every single cell in the human body has a copy of every single gene coded onto our DNA, but not every gene is expressed in every cell. Gene expression is controlled by transcription factors, which are divided into promoters (the site of RNA polymerase binding to DNA chromosomes) and enhancers and silencers, which produce proteins that bind to promoters (acting as activators or repressors) enhancing or blocking transcription. RNA polymerase is an enzyme that transcribes a gene and determines how that gene is expressed. Put simply, gene expression is about whether a gene is switched on or off in any given cell (Kandel, 1998). When the gene is turned on, information on how to make the proteins is transcribed from the cell (Doidge, 2007).

Several studies have identified specific transcription factors which are important in the persistence of long term potentiation (LTP), long term depression (LTD) and other forms of neural and behavioural plasticity (McClung and Nestler, 2008). LTP concerns synaptic change and the plasticity that allows this, while LTD is the state of

long term stability of neural connection. (See Chapter 3 for further descriptions and explanations of LTP and LTD.)

Lipton (2005) uses the metaphor of the arm to describe gene transcription. If the arm is bare, it is expressed in a certain way. If it is clothed with a shirt it is expressed differently, and we would describe it differently. Clothed or unclothed, it is still an arm. Expressed or not expressed, each gene coded into our DNA is present in every cell of our brains and bodies. And every environmental influence that we encounter impacts the delicate biological process that regulates gene expression (Doidge, 2007; Kandel, 1998; Lipton, 2005; McClung and Nestler, 2008).

Environmental factors that can affect gene expression include, but are not limited to, the food we eat, the amount and quality of sleep that we get, whether we exercise or not (see Chapter 6 for more about these factors) hormonal factors (including pregnancy and post natal changes), environmental pollutants, drug and alcohol consumption and/or addiction (see also the section on addiction in Chapter 4) temperature, planetary dark-light cycles, viruses, inflammation and bacterial infection, and physical and emotional stress (Doidge, 2007; Kandel, 2000) (see also the section on stress in Chapter 4).

Genes do not directly control behaviour. A single gene encodes a single protein, and a protein is not a behaviour. Behaviour is generated by neural circuits, involving many cells, or neurons, each of which is expressing specific genes, which in turn direct the expression of specific proteins. Genes expressed in the brain encode proteins that are important in the development, maintenance and regulation of the neural circuits that underlie behaviour (Kandel, 1998). Alteration of gene expression involves changes in patterns of between neurons/nerve cells, and in some cases, either the growth or retraction of synaptic connections (Doidge, 2007; Kandel, 2000).

Behaviour itself can also modify gene expression, because it regulates the environmental factors listed above (Kandel, 1998). Gene expression and behaviour are thus linked in a symbiotic relationship where each constantly affects the other. The template function of genes is important, but it is the environment that determines how they are expressed, and whether they are switched on or off.

Lipton (2005) likens the relationship between genes and the environment to the behaviour of cells in a laboratory dish. If something

that nourishes the cells is placed in the dish the cells will flock to it and become healthier, plumper cells as a result. If a toxic substance is placed in the dish with the same cells they will shrink from it, and many, if not all, will die. Lipton (2005) draws a parallel between this cellular behaviour and our own interaction with the environment, given that our brains and bodies are merely incredibly complex collections of cells. He sees the environment as the most important factor in how we experience our lives, given that it regulates which template genes are switched on, and which are not. Healthy choices result in more optimal transcription of genes.

What we experience (from the external environment) and what we learn (how we process our experience internally) also has an effect on gene expression (Doidge, 2007). Early learning and development periods are particularly key in deciding how genes are transcribed (Kandel, 1998; Kandel, 1999; Pham *et al.*, 1999). Gene transcription is influenced by what we do and what we think (Doidge, 2007). It is also likely that psychiatric dysfunction is partially the result of alternate gene expression (Kandel, 1998).

What does this mean for counselling?

The fortunate thing is that most of us are in control of many aspects of our own environment. The template function of genes is indeed out of our control, and as the old saying goes, 'you can't choose your family'. What we inherit may be fixed, but whether that inheritance is expressed or not is something that we do have some control over.

Because early learning and development periods are very important in the expression or otherwise of genes, the physical, social and emotional environment that parents provide for their children is crucial. Many counsellors find themselves working at some time or other with clients who are parenting their children, and those children are often part of the client's story. Being able to share information about the importance of the world in which children grow up allows clients to make informed choices around issues, such as family violence and how to parent. This even includes basic decisions, such as what sort of food to give their children and when their bedtimes should be.

Obviously, the life that we choose for ourselves, and the decisions we make about how to live, the environment in which we place ourselves, how we control it, and what our response to it is, are hugely important for each and every one of us. Genes retain their potential for alternate expression throughout life, and can potentially be switched on and off by the changes we make.

Counsellors can work with clients to identify the presence of anything in their environment that may be adversely affecting them. For instance, this could include smoking cigarettes, living in damp conditions, eating a diet high in 'junk food', working night shifts and experiencing few daylight hours, or being the recipient of verbal abuse. I once had a client who found that the depression they were experiencing was alleviated when they moved to a warmer climate in Australia, with more hours of sunshine, and returned every time they came back to a New Zealand winter.

Counsellors can also support clients to make choices and set goals to optimize their environment, both physically (where they live, whether they exercise, how and what they eat, the amount and quality of sleep they get, any substances that they put into their bodies, etc.), and socially and emotionally (the presence or absence of relationships, who they are in relationships with, the quality of those relationships, the presence of support or the absence of abuse, etc.).

It is important for counsellors to remember that an optimal environment may vary from one person to another. For example, some clients may need more or less sleep than others, and some clients prefer more social contact than others. The goals for environmental change, if any, must as always be the goals of the client. If they are not, the client-counsellor relationship will be unsuccessful, and the goals will not be achieved in the long term, because any effort that the client makes will be in the service of pleasing the counsellor rather than because the client wants to make permanent change. Once the counsellor is absent, the client will revert to pleasing themselves.

Some clients are happy with what they have, and prioritize the rewards that it brings them over the discomfort that it causes them to experience, or the long term consequences. A client who smokes may prioritize the stress relief of smoking a cigarette over damage to their health, and a person on constant night shifts may prioritize the money that they earn to support their family over a healthy exposure

to daylight, and this is their right as autonomous beings. Once a counsellor has shared their knowledge of how environmental factors affect every cell in our brains and our bodies, and therefore who we are and how we think, learn and behave, it is then up to the client to decide what to do with that knowledge.

Counsellors need to remember also that not everyone's capacity for change is equal (see the section on plasticity and rigidity in this chapter). Some clients lack the right conditions for brain plasticity and do not have sufficient ability to create new neural pathways through rerouting synaptic connections. For others, some changes to their environment are just too hard. Poverty or low income can be a barrier to environmental change, as can duty or obligation to others (parents, children or partners), fear of the unknown or the new, fear of reprisal from others or addictive reward based pathways in the brain. Where barriers exist and clients are motivated to make change, counsellors can help clients make maps of social supports or help them to brainstorm solutions, including even small steps towards change to prepare for any openings that may allow it in the future. A person who doesn't wish to leave an abusive partner might begin a secret bank account in case they ever do leave. Someone else might investigate study options to better their financial position. Another client may choose to work on changing neural pathways for self-esteem, so that they have more ability to consider the thought of life without their current relationship.

The exciting thing for counsellors is the knowledge that what we do, how we behave, and how we adjust our environment and adjust to it, bring huge potential for change at structural level in the brain, because the genes we inherit can each be switched off or on, and these changes in turn affect everything about our brains and how they function.

Myelination and white matter

What do we know?

Myelin is a substance that coats the axons of neurons. It is often referred to as the myelin sheath, and is composed of layers of lipids (fatty substances) and proteins. Myelin basic proteins are associated with strong immune responses (Kandel *et al.*, 1995). Inhibitory proteins in myelin bring critical developmental periods for learning to an end by suppressing both axon sprouting and the forming of new synapses after learning has been completed (Fields, 2008).

White matter is the region underlying the grey matter cortex, and is composed of neuronal fibres coated with myelin, providing electrical insulation. White matter comprises over half the human brain (Fields, 2008). Myelination of axons was previously thought only to occur in young developing brains, but is now known to continue for decades in the human brain. It can be modified by experience, and it affects information processing by regulating the speed and synchronicity of neuronal firing between distant regions of the brain. Myelination can be regulated by neuronal electrical activity. It is vulnerable to damage from metabolic disease and autoimmune disorders (Fields, 2008).

Development of white matter structure correlates with increased development of motor skills and cognitive functions. Conversely, incomplete white matter development correlates with poor cognitive ability and decision-making ability. Functional imaging studies also show strong correlations between IQ and the amount of white matter in the brain (Fields, 2005). Rats raised in an environment rich with stimuli have increased volume of white matter, showing that myelination can change according to environmental experience. It is interaction with the external environment that appears to increase myelination. Neglected children have 17 per cent less volume of white brain matter (Fields, 2005). Several mental disorders are associated with reduced white matter volume, including depression, BAD and other mood disorders, Alzheimer's, autism, attention deficit disorders, compulsive disorders, post traumatic stress, Tourette's and schizophrenia (Fields, 2005, 2008). Medications and drug abuse can have a negative affect on white matter (Fields, 2008). Myelin repair can be aided by exercise and physical therapy (Fields, 2008).

Practising complex skills increases the volume of white matter in the brain. The increase is in direct proportion to the time spent practising those skills. Fields (2008) believes that myelin plasticity complements synaptic plasticity. It is thought that myelin controls the speed of impulse conduction through axons, and from one neuron to another, so that information from different regions of the brain reaches the same place in a timely manner, allowing integration of that information (Fields, 2005). This is probably how the process of neurons wiring and firing together is synchronized. Timing deficits are associated with language processing disorders and dyslexia (difficulty in processing and using written symbols) (Fields, 2005). Myelin also directly controls synapse formation by inhibiting axonal sprouting after the formation of appropriate connections, and in this way is linked to synaptic plasticity (Fields, 2008). (Synaptic plasticity is activity dependent, and connections weaken or strengthen according to use.)

What does this mean for counselling?

The main learning to be gained regarding myelination is learning about our learning. Much like what we know about synaptic plasticity, myelin plasticity suggests that if we want to learn a new skill, be that cognitive or behavioural, we must practise it, and not only will we form new neuronal pathways but we will build the structure and resilience of our own brains. The volume of white matter can be increased by practising complex skills and exercise helps myelin repair, valuable knowledge for those affected by psychiatric disorders. (See also the section on exercise in Chapter 6 for other benefits to the brain through exercise.) What we know about myelination and white matter contributes to the message that counsellors can give to clients: change is possible with determination and practice.

Counsellors considering child protection agency notifications or other interventions in the case of child neglect would do well to consider the consequences of neglect to the white matter of the child's brain when making their decisions.

Left brain/right brain

What do we know?

Each of our brains is as unique as our faces or our fingerprints are, but there are basic similarities. The brain is divided into two hemispheres, the right and the left. The two hemispheres process things differently, but work in tandem to complement each other and increase our ability to perceive the world in different ways. The two halves are connected by a thick pathway called the corpus callosum, a neural structure made up of axons. Each hemisphere processes the same information, but filters and organizes it for different things before it is integrated across the corpus callosum (Bolte-Taylor, 2008). The right hemisphere controls the left side of the body in terms of motor control and vice versa (Kandel *et al.* , 1995). The left hemisphere is more concerned with detail, patterns and the analysis and organization of information. It processes information rationally and sequentially. The left brain makes decisions about right or wrong. The left brain tends to process things at greater speed. Our left brain perceives the shorter wavelengths of light, which helps it to see the sharper detail and to concentrate on lines and edges. The left hemisphere tunes into higher frequencies of sound, helping to detect, discriminate and interpret verbal language. The left hemisphere is good at perceiving time and keeping track of things.

The right hemisphere processes the 'whole picture', and is better at lateral thinking, or making novel connections. It is less likely to put judgement or moral value on to anything. The right brain processes information more slowly, possibly because it is processing in a less linear way. The right brain is sensitive to non-verbal communication. The right brain perceives the longer wavelengths of light, allowing it to concentrate better on visual input as a whole, because the data input gathered is softer and more blended. The right hemisphere tunes into lower sound frequencies, and is better tuned to the primal physiological sounds our bodies make. The right hemisphere is less concerned with the perception of time and its passing (Bolte-Taylor, 2008).

People have been known to function relatively normally with only one active hemisphere (Bolte-Taylor, 2008; Doidge, 2007). Some people seem to be more strongly connected to one hemisphere and are consequently more inclined to be logical and analytic and interested in detail if they are 'left brain' people, and inclined to be more creative,

lateral thinkers if they are 'right brain' people. Most people are a mixture of these things because the hemispheres integrate the input they receive (Bolte-Taylor, 2008).

The left hemisphere has a tendency to create 'stories' about possibilities that may or may not be true (Bolte-Taylor, 2008). The hippocampus located in the prefrontal cortex of the right hemisphere is concerned with filtering these to check their validity (Schacter, 2001). If the right prefrontal cortex is dysfunctional, people can be prone to confabulation, or making up stories which they believe to be true (Bolte-Taylor, 2008).

Each hemisphere is divided into a number of regions which perform different but parallel functions (Kandel *et al.*, 1995). Imaging technology makes it possible for differences in function between the two hemispheres to be observed and measured (Gordon, 2000). Many incoming stimuli and/or the brain's responses to those, including cognitions and behaviours, are primarily processed in one hemisphere or the other.

What does this mean for counselling?

There is incredible variation in the clients we see, and being aware of left brain/right brain differences as a contributing factor to personal style and the way that a client thinks or processes information is a tool that a counsellor can use to 'be on the same wavelength', so to speak, as the client. Many clients will be a mixture between left and right brain characteristics. Others will be very strongly focused on information and logical processing. Other clients will leap from place to place, and weave around the main topic of the conversation.

Some will need to process things one step at a time, with one thing leading to another. Others will make creative leaps. Some strongly left brain people will be focused on a significant detail or two; other strongly right brained people will be focused on the big picture. Allowing for differences in processing style is an important factor in being a good counsellor. Clients will vary in terms of the pacing that is comfortable for them in counselling, which will be partly correlated with IQ, but also according to which hemisphere of the brain is more dominant for them.

An important point to remember is that dysfunction of either hemisphere could lead to behaviours in clients that it is commonly assumed people should be able to take responsibility for. For instance, a dominant left prefrontal cortex, and an underdeveloped, dysfunctional or damaged right prefrontal cortex can lead to a combination of confabulation (or fantasizing), and a lack of ability to see the big picture or to make creative or flexible leaps in thinking. It can also lead to symptoms that mimic Attention Deficit Disorder/ Attention Deficit Hyperactive Disorder symptoms, such as a lack of ability to pay attention, poor memory, a lack of impulse control, and poor planning skills, given that the prefrontal cortex, most specifically the right orbitofrontal cortex, is the special region that mediates these skills. (See the sections in Chapter 3 on memory and learning and attention.)

Counsellors need to beware of judgements around whether someone should or should not be capable of personal responsibility for being truthful (keeping in mind that the truth is but an elephant in a room full of blind people anyway). It can be very irritating for a counsellor to base their whole session, or a series of sessions, around what a client expresses, only to find out later from another source that what was discussed only has some basis in the reality perceived by the majority. It is then important for the counsellor to evaluate in their own minds whether this could be due to a strong left hemisphere, weak right hemisphere combination.

If the counsellor considers this a possibility, they then have the option of 'bringing the issue into the room' in any subsequent sessions for exploration by both client and counsellor, and in doing so, establishing a more genuine basis for interaction by establishing and acknowledging that commonly held perceptions regarding any given events may not be what the client articulates. Some counsellors may be able to work with this while others may not. It is interesting to note that positive outcomes in counselling are still correlated with Rogers' core conditions of genuineness, warmth, communication and empathy on the counsellor's part (Rogers, 1957). Change is possible under widely variable conditions.

Chapter 3
Learning and Memory

Learning and attention

What do we know?

When the brain learns anything, it subtly changes the brain's structure. Permanent learning involves memory (Kandel, 2000), which will be discussed later in the chapter. Kandel (2000) regards memory as the outcome of learning, and as such, inseparable from it. Most forms of learning change the effectiveness of synaptic connections and therefore affect not only cognition and data storage but also emotion and behaviour (Kandel, 2000).

New learning occurs with 'massed practice', that is, practice that is repeated over and over again. This means learning is retained well in the short term memory. An interval before more massed practice helps cement learning further and it is retained as long term memory (Doidge, 2007; Kandel, 2000). Repeated practice strengthens the synaptic connections between the neurons that will form the new pathways allowing learning to be stored in the brain. Attention and focus on the learning task are also important, perhaps so that the learning can be laid down in memory (Doidge, 2007). Brain chemical reward, released in the synapses that connect the neurons (e.g. dopamine, acetylcholine), rewards and consolidates learning (Stephan, Baldeweg and Friston, 2006).

Mental training and/or enriched environments, which provide more opportunities for learning, create denser brains, as neurons develop more dendrites, make more connections and increase in size and blood flow (Fields, 2005; Kolb *et al.*, 2003). Post mortem data show that education increases the effect of the denser brain. Brain volume is increased as neurons push against each other, expanding

the space that they occupy (Doidge, 2007). Increased brain derived neurotrophic factor (BDNF) activates critical periods in the brain for learning and development and makes the brain more plastic and receptive to learning during these times (Doidge, 2007).

The speed of thought is influenced by plasticity. The more we travel a pathway, the faster and better we get at it, and it is the same with neural pathways (Doidge, 2007). As a skill is practised, the neurons become more precise because they are better at it. Therefore, less of them are needed to form the pathway, and those neurons are freed up again to do more learning of something new. A stronger, more specific neural pathway is created.

Incorrect or unwanted learning takes up space on our maps of neural connections, and leaves less room for learning that is correct or desirable. This is called competitive plasticity (Doidge, 2007). Despite neurogenesis (the birth of new neurons) continuing to occur even in adults, there is a finite amount of neural real estate available, and learning often builds upon previous learning because this provides not only integration of learning, but also the most efficient use of our neural capacity.

Because learning results in preferential neural connections and pathways in our brain, learning can result in us becoming primed, or prewired, to either anticipated trauma or pleasure when we notice certain cues (Corbetta and Shulman, 2002) (neurons that fire together wire together). When an incoming stimulus becomes linked to an automatic behaviour through experience and repetition, this is known as classical conditioning (Kandel, 2000). Triggers or cues related to past learning may bias the way the information is processed by directing that processing through prewired neural pathways. Recorded brain activity (as measured by functional magnetic resonance imaging, fMRI) significantly increases when the onset of a stimulus is anticipated. The pattern of brain activation indicates that the parietal cortex and frontal regions of the brain are involved in controlling attention (Corbetta and Shulman, 2002).

Attention is used to control the details of our awareness. It is not possible for anyone to take note of every detail of all incoming sensory input, and attention is what gives priority to what we are filtering from the outside environment (Postner and Rothbart, 1998). We screen out what isn't relevant to keep us sane and safe from overload (Wolfe,

1998). Our perception is unconsciously filtered through what we let through our sensory processing filters. This is probably the basis for the story of the elephant and the blind man. Technically, we are not feeling just one different part, because the piece of reality that we are experiencing is made up of an unknown number and combination of sensory inputs, which are in turn selected or given priority attention by our existing neural pathways (Corbetta and Shulman, 2002).

Incoming stimuli are processed by sensory cortices, which transmit their signals into the limbic system (the part of the brain associated with emotion, learning and memory) where they are integrated with each other so that they match each other in timing. This is known as 'reafference.' The limbic activity patterns of arousal are also sent into the motor systems of the brain stem and the spinal cord resulting in responsive action. The resulting integrated data are transmitted back to the primary sensory cortices, preparing them for the result of any motor action. After multi-sensory convergence, the limbic system updates the hippocampus, which is a part of the brain involved in what is called 'executive function', which relates to learning and memory and to planning and decision making, including which sensory data to attend to. Everything we know about the external world comes from a circular pattern of our action, reafference, perception, update, our action, etc. (Freeman, 1998).

The anterior cingulate cortex, close to the hippocampus, plays a key role in focusing our higher levels of attention. If it is a language task, parts of the frontal cortex are also involved, and also the right hemisphere of the cerebellum (Postner and Rothbart, 1998). Regions in the left posterior parietal cortex are recruited for switching attention from task to task (Corbetta and Shulman, 2002).

People ignore, forget or attempt to discredit information that doesn't fit in with the data set down in current neural patterns in their brains. It is difficult and distressing to perceive the world in new and unfamiliar ways (Doidge, 2007; Wolfe, 1998). We link what sensory input we allow through our sensory processing filters to prebuilt neuronal connections and networks (Wolfe, 1998, 2006). If there is a poor fit for our existing internal complexity we discard the piece of information that doesn't fit, much as we might throw away a piece of Lego that we were unable to fit into a complex structure we were building without breaking down what we had already built and

starting again. Not many of us are willing or able to cast aside elaborate structures that have served us well for many years. The interconnected nature of our neural assemblies and pathways mean that when one part is changed it necessarily affects many other parts, which in turn affect others in a sort of ripple effect. The use of metaphor is helpful for learning as it serves to link new knowledge into what we already know (Wolfe, 1998, 2006). The story about the piece of Lego just described provides an example of such a metaphor.

In contrast, when we habituate something we stop paying attention to that stimulus because we have come to take it for granted. Habituation leads to a decrease in the strength synaptic connection between excitatory interneurons and motor neurons. (Remember that the interneurons perform a relay function.) There is no change in the strength of synaptic connection between sensory neurons and interneurons (Kandel, 2000). In other words, we still receive the incoming sensory data, but we stop responding to it.

Sometimes when a directly harmful stimulus is repeatedly experienced it can result in sensitization, and we react more strongly against it. Repetitive practice (further consolidated if there is spacing between blocks of repetitive practice) which is necessary for plastic learning, produces longer periods of sensitization. Sensitization results in the growth of extra axonal dendrites, and habituation results in the pruning of dendrites (Kandel, 2000).

Brain washing can be produced by someone taking total control of someone else's environment, and providing conditions of massed practice and linking this with reward and punishment systems in the brain (conditioning) (Doidge, 2007).

Corbetta and Shulman (2002) believe that there are two different attention systems: the first, a bottom-up system controlled by sensory input, and the second a top-down system, mediated by existing neural connections and cognitive factors, such as knowledge, expectation and our current goals.

What we pay attention to can be affected by certain conditions (Corbetta and Shulman, 2002). For instance, we notice more of what we are looking for. If I am looking for someone in a crowd, and I know them to be wearing a certain colour, I will notice that colour everywhere, because I am looking for it, and fail to pay attention to other colours. In this way, bottom-up data (input from the colour I

am searching for) interacts with top-down data (my goal of finding the person I am looking for). We pay attention to what we have been primed to look for (either because it is our own goal, or because others have suggested that this is what we will notice) (Corbetta and Shulman, 2002).

What we pay attention to is easier to notice if it stands out in some way, for example, a red circle in a pattern where all the other circles are green. Attention also often goes to novel or new stimuli as we assess its potential for danger. Attention that is not based on priming or anticipation is processed in a different region of the brain; the right ventral frontal parietal network (Corbetta and Shulman, 2002).

The differences in types of attentional processing have implications for those with attention deficit disorders, such as ADHD or ADD, in which the A stands for attention, and the first D stands for deficit. Those with these conditions are very responsive to bottom-up sensory data but seem to give excessive priority to processing the new and novel stimuli (Corbetta and Shulman, 2002).

What does this mean for counselling?

Given that our reality is shaped by the attention or priority (or lack thereof) that we give to the sensory input available to us, one of the roles of the counsellor is to draw attention to other stories that could be constructed from the same sensory data, or to explore what sensory data may not have received attention. What else could fit into the story? What other stories can be constructed from the same data by giving different weighting to different information? What other data did other people present give attention to? What differed about their experience in contrast to the experience of the client as a result?

This is already a tried and true counselling technique, commonly known as reframing, and it allow clients to experience the 'elephant' from different angles.

Another role for the counsellor is to alert clients to the concept that what they are 'looking for' or giving priority attention to is what they will notice. If they are looking for the colour blue, then the colour blue is what they will notice, even though they are surrounded by a multitude of colours.

Most counsellors probably notice this phenomenon already, with some clients seemingly predisposed to notice negative events and experiences in their lives, and others focused on positive events and experiences. Even during discussion of the same event, one client may see the experience or outcome as distressing, while another processes the same thing as an opportunity or a learning experience.

Naturally, it takes more than noticing these patterns to make change for the client, if indeed they desire it. Some clients have well developed neural pathways for noticing the negative because it serves their goals, such as being able to obtain sympathy and help from others by relating the awful things that have happened to them. In this way, they are able to obtain the physical and emotional resources that they need.

For clients who would like to experience life as a more positive experience, it is back again to the conscious rewiring of neural pathways, and enduring that 'wrong' feeling while the new pathway is still fresh. The new pathway must be trodden over and over again (that is, repeatedly practised) to make it a strong one by strengthening the synaptic connections between the neurons and allowing the old ones (which give priority to negative experience from the processing of sensory data) to fall into disuse.

The use of metaphor has long been an effective strategy in counselling and now it has an explanation to link it to what we know of neuroscience. Metaphor, or the use of a familiar allegory, enables people to make linkages between what they already know and what is unfamiliar for them. Perhaps they are struggling to make the new learning fit with learning that they have already laid down existing neural pathways for in their brains.

An example of metaphor might be the use of a story about being on a diet (or more appropriately, a plan to eat more healthily) and not being able to resist opening the packet of chocolate biscuits at the back of the cupboard and eating one. There is then a choice to be made; stop at one or two biscuits or eat the entire pack? Many people are familiar with this situation. This can then be linked to practising another skill of restraint, such as avoiding the consumption of alcohol, or the thinking of an unwanted thought. Having slipped from the chosen path, the client has the choice of getting back on course or of giving up on their goal. When clients have one glass of alcohol, they

can then choose to stop or to get drunk and abandon their goal to stop drinking (providing they do not have existing neural pathways for alcohol addiction, which makes choice less relevant; see the later section on addiction). When clients think the unwanted thought, they can surrender to it and agree with its power, or they can correct it and concentrate their thinking on their pre-prepared preferred thought. Having the metaphor about the chocolate biscuits makes them aware that even if they cannot yet manage to be fully on the pathway to their goal, they can self-correct and return to that pathway even after a 'slip up'. And the more they practise restraint, the better they become at it, because of their ability to self-correct.

This metaphor allows a client to connect the new idea that achieving a goal is still possible, even after they have taken an action or had a thought that is incongruent with that goal, with the familiar situation of what to do after eating one chocolate biscuit when dieting. Different metaphors are of course suitable for different clients, and a person who has been underweight all their life may not relate to the example above. Part of the skill of the counsellor is to have gathered enough information about the client during the assessment phase (and in subsequent sessions) to know which metaphors a client will relate to.

Attention also needs to be given by the client regarding what cues or triggers from the past could prime them to process sensory data in a certain way, because in effect, these are the gates at the entrances to our neural pathways. It will require insight and conscious effort to prevent these gates from opening, but happily, the insight gained during counselling will be the foundation for new pathways, given that all learning changes the structure of the brain. Insight gives us new gateways to potential pathways. Attention and focus are required to keep us on them, and are a crucial part of consolidating new learning.

Habituation presents a challenge for many clients. Even sensory input that, when novel, would produce alarm signals requiring immediate action can be habituated, including abusive behaviour, such as family violence. Once habituated, the brain stops giving selective attention to the stimulus. To pay attention to habituated abuse, the counsellor would need to prime the client to attend to it and 'look for it', in order for the client to notice it as being out of the ordinary, and to formulate a response to it. Once upon a time, this may have

been labelled 'consciousness raising' and group programmes, such as protected person's programmes may also fulfil this function.

Being sensitized to certain cues or stimuli may also result in challenge for some clients. Being physically abused in the past may lead to a client ducking if someone raises their hand to wave to them. Having experienced verbal abuse in the past may lead clients to misinterpret what others are saying about them as negative comment when that is not what is intended. Once again, the role of the counsellor here is to provide new gateways to potential new neural pathways by facilitating insight.

Brainwashing is not necessarily the prerogative of kidnappers and terrorists. Repeatedly being exposed to certain messages, such as 'you're fat', 'you're stupid', or 'you're nothing without me', means that learning and absorption of those messages will occur if the person receiving them gives them their attention. (This applies to positive messages too, such as 'you're beautiful', 'you're intelligent', and 'you can do anything you put your mind to'.) Often it is the client's nearest and supposedly dearest who deliver these negative messages (parents, siblings, extended family, partners and close friends). Any harmful learning is made possible because of the client's repeated exposure to these messages and the chance for the learning to become consolidated.

Yet again, the role of the counsellor is in the reframing. Looking for exceptions to the learned pathway (as in narrative and solution focused counselling) provides the client with a neural pathway already in existence, either disused, or in its infancy, to begin to build a new preferred pathway with. Because the unwanted pathways have been formed over the long term (sometimes starting very early in our families of origin) the client must be warned to expect that it will take a lot of conscious practice (the basic condition for new learning) of the new pathway in order to make it strong enough to become the automatic route for neural processing.

Clients often need help with new learning that does not fit into existing neural pathways. An example of this might be to find out something about a parent or a partner that challenges everything that we have always believed about that person and our lives with them. Sometimes, we may have based our behaviour, or our opinion of ourself on those beliefs. Radical new learning may threaten to collapse

much of the foundation on which much of our assumption about life is constructed.

Here, a counsellor has a holding role in providing some consistency in a changing world, and a blank page for the client to test new stories about their lives on. Some clients may find integrating the new knowledge too difficult and too distressing, and reject it rather than consolidate it as new learning, and this is their prerogative, for counsellors are not there to be arbitrators of truth or perception or to administer unpleasant knowledge and insist it be retained. The ability to filter sensory data helps to keep us sane and safe from sensory overload. Other clients may use more courage and begin the long process of rebuilding. Until they once again have an integrated view of reality, rather than conflicting assumptions about the world, they will remain in distress. But, to reuse the Lego metaphor, even though rebuilding causes frustration, confusion and sometimes grief at the loss of the model we were working on, the reconstruction of a new working model brings its own rewards, including faith in our newly constructed neural pathways for learning. The new working model also has the advantage of being better suited to its environment and of superior complexity.

A map of ourselves

What do we know?

Merzenich *et al.* (1983) found that our neural connections create neural 'maps' in our brains. Their experiments were pivotal to demonstrating the plasticity of the brain and the competitive nature of plasticity. Not only are there neural maps for the actions we perform and the skills we learn, but there are maps of neural connection representing every part of our body. These neural maps, made up of the connected pathways in the brain, can change, shift, extend or disappear, according to the principles of whether those pathways are currently in use or not, and if they are, which other neurons they are firing simultaneously with. For instance, Merzenich *et al.* (1983) did experiments that showed that separate maps for separate fingers can become a single map if the

fingers are joined or bound for several months so that they can only move together.

Neural maps of normal body parts, such as faces, can change every few weeks. The maps are grouped together according to the space and time between their common usages, for greatest efficiency. For instance, the parts of the hand that play an instrument together will all be represented next to each other in the brain. If the foot is used to tap along with the rhythm, it could well be represented next to the parts of the hand. Close attention must be paid to the skill or the manipulation of the body part in order for maps to be altered, or new maps to be formed, and as per the section above on learning and attention, new pathways to create new maps are formed by repeated practice (Doidge, 2007). The concept of a map can be related to the concept that Hebb (1949) put forward regarding neural or cellular assemblies: collections of interconnected neurons that fire together, either simultaneously, or one after the other, to complete specific actions.

We also have maps for where we are in space. The hippocampus contains a cognitive map of the spatial environment in which we move, and our location within it is encoded in the firing pattern of individual pyramid cells (a type of excitatory neuron). The same cells can be used for different spatial maps, and the maps can adapt and become stable within minutes, and self maintaining over time with regular use (Kandel, 2000).

A general failure of brain plasticity will mean a lack of ability to adapt to new environmental conditions, and a failure to be able to form new neural pathways representing new learning (Doidge, 2007).

What does this mean for counselling?

This section, which overlaps the sections covering plasticity and learning, has been included because it illustrates the way in which our images of ourself are mapped onto our brains. There are neural assemblies for fingers, noses, genitals and legs; every part of our body in fact. These maps are connected to our overall conceptions of our bodies and our faces, including how we perceive them, which we project as how others will perceive them, and therefore, us.

We live in a culture obsessed with self-image, which may or may not be different to how things have been in other places and times. The advent of the mirror and subsequently the photograph/digital image, have surely led to scrutiny of ourselves which was not previously possible. The condition of body dysmorphia is prevalent, where people have an internal image of their bodies as being other than how the majority of the world views them. There are people who believe themselves to be excessively fat, while they are in fact starving themselves to death; extremely overweight people who 'feel' as if they are of average weight; and people within the normal range of appearance, who consider themselves to be incredibly ugly and view the world from that perspective, dying inside when they interact with others because of what they believe those others are thinking about them.

These people are affected by neural maps created by thought patterns that they not only pay close attention to, but also practise repeatedly, the necessary conditions for learning and creating neural pathways and maintaining those pathways as strong, well used neural connections.

An underlying 'condition' in many clients who present for counselling is their self-image. It may never be brought forward in the counselling sessions, but their thoughts and beliefs may be based on their perceptions of themselves and their actions (or failure to act) based on what they therefore believe others are thinking about them. Poor self-image limits a person's confidence, and can lead to feelings of shame and a wish to avoid contact with others. It does the counsellor no good to argue a different perception, that they do not believe that the client is fat or ugly, because the client rejects this since it does not fit in with the beliefs represented by the strong, well connected neural pathways that they have already formed. (See also the section on learning and attention in this chapter.) The counsellor's opinion, so to speak, is like a jigsaw puzzle piece from an entirely different jigsaw, and is discarded by the client as a 'bad fit'.

As mentioned in the section on learning/attention, sometimes metaphor can be helpful here, because when the client doesn't have to directly apply the 'jigsaw puzzle piece' to their own 'jigsaw' it gives them longer to consider the piece before they decide whether to discard it or not. Similar techniques with the same outcome, such as asking a client what they would say to a specific friend or family member if

they disclosed the same feelings of self-hatred or self-dissatisfaction can be helpful, because they provide the basis for thinking about a similar situation with new neural pathways, and clients usually give their friend or family member the same advice as the counsellor would like them to internalize in relation to their own situation. This has the advantage of being constructed from the client's own neural workings, rather than those of the counsellor. When the counsellor draws what the client has said back to their own situation, the fit is better, and if not entirely a good fit, at least the client can see the possibility of working the piece into the puzzle at some stage in the future.

If no suitable person is available in the client's life to work in this way, the counsellor can ask the client's advice about what to say to a (fictional) person in a situation described by the counsellor which is similar to that of the client.

Another useful way of working is to have the client collect information from their own lives, which they have discarded as being a 'bad fit' for the internal neural representations of their current beliefs. For instance, times when they have received compliments, or times when other people have not acted in the way that they would have expected them to act towards them, i.e. by showing attraction, affection or admiration. The counsellor can then take the naive enquirer stance commonly used in narrative therapy, and wonder how these things can fit with the way the client has presented themselves and the client's beliefs about themselves. The client then takes the initiative to make the pieces fit, because they have retrieved those pieces from their own internal memory, which is part of the neural interconnection of the brain, so they assume that somehow those pieces must fit, even though on the surface it may appear to them that they don't.

It is my own experience that when clients are asked to retrieve the information they have discarded as a 'bad fit' they rarely do, because they do not believe that it will exist. The counsellor is more likely to be able to support the client by drawing attention to these things when they occur naturally in the client–counsellor conversation, bringing those unregarded pieces from the background to the foreground for closer scrutiny.

Clients may become addicted to their body dysmorphia, and not wish to let it go. Sometimes this is because of the brain chemical changes associated with addiction to natural biological rewards

(see the section on addiction in Chapter 4). It may also be because the beliefs it represents are so core to the world view and internal neural organization of the client, that to change them would be like living in a house and ripping the foundations out from underneath. Discarding the foundations means that the entire house will need to be rebuilt, and for many people, that will be simply too hard. Counsellors must always realize that goals for change must be the client's and not the counsellor's, because, quite apart from ethical considerations, only the client can do the necessary work to build and reopen new neural pathways and to allow old neural pathways to become disused.

If the client does have a goal towards changing their body image, counsellors should encourage clients to work towards the things that promote the brain plasticity required for new learning (see Chapter 6). These include adequate quality sleep, eating healthy food, abstinence from unhelpful addictions, Omega 3 supplements, and using mindfulness and breathing relaxation techniques.

General brain plasticity is also required for building new neural maps in spatial location, and someone who has a resistance to new environments (a new school, moving house/towns, going into hospital, etc.) should also be encouraged by their counsellor to do the things listed above before they practise feeling comfortable by allowing repeated brief exposures to that new environment, in order to build a strong new neural pathway.

Memory

What do we know?

Memory is used for forming a perceptual basis on which to decide how to respond to incoming stimuli (Freeman, 1991, 1998). A large body of evidence indicates that new neural connections created through the process of learning (see also the section on learning and attention in this chapter) become memory through alterations in glutamate dependant excitatory synaptic transmission. These modifications are actively stabilized by structural changes at postsynaptic sites on dendritic spines over hours or days (Lamprecht and La Doux, 2004).

Dendrites are the receptor sites of neurons (Haines, 2006; Kandel *et al*., 1995; Squire, 2008).

This probably requires long term potentiation (LTP). LTP can be artificially induced by high frequency stimulation in the laboratory, but the biological mechanism for LTP in the brain is proving elusive (Colicos and Syed, 2006). LTP is complemented by long term depression (LTD). LTP is the mechanism for change in synaptic connection, and LTD is the mechanism that stabilizes new synaptic connection, closing down the process of change so that the pathways can become long lasting, at least until LTP occurs again (Kandel, 2000; Lamprecht and La Doux, 2004). Although neuroscientists have not yet identified the precise mechanisms for LTP and LTD (Hobson and Pace-Schott, 2002), they can see its effects, and several candidate mechanisms have been proposed (Abbott and Nelson, 2000; Adamec, 1999; Johnston, 2003; Lamprecht and La Doux, 2004; Maffei and Turrigiano, 2008; Martin, Grimwood and Morris, 2000), although at the time of writing, they are not yet proven.

Synaptic plasticity is the term given to the physical process where specific patterns of neural activity lead to changes in synaptic efficiency and neural excitability, which outlast the events that trigger them. Remember, synapses are the spaces across which neurons transfer chemical or electrical messages, connecting one to another (Haines, 2006; Kandel *et al*., 1995; Squire, 2008). Synaptic plasticity is vital for memory, and processes such as LTP and LTD are likely to contribute to this (Martin *et al*., 2000).

Temporary neural changes are believed to represent what is accessible in short term memory, while more lasting changes are eventually transferred to long term memory. Long term memory is also thought to require changes in gene expression and the synthesis of new proteins to support this (Lamprecht and La Doux, 2004). LTP doesn't persist in animals injected with protein synthesis inhibitors (McClung and Nestler, 2008). LTP is believed to lead to changes in the number and shape of the dendritic spines of neurons, for example, the thickening of spines and the formation of new ones, as well as the shortening and widening of the neck of the neuron. The head of the neuron also increases in size, and glutamate sensitivity is at its highest with larger cellular heads (Lamprecht and La Doux, 2004).

Changes in synaptic transmission are initiated by raised levels of intracellular calcium, which is thought to trigger the neurons that fire together wire together effect. When the cellular neck widens, this may allow the influx of calcium into the dendrite. Dendritic spines provide closed compartments which allow rapid changes in the concentration of signalling molecules, such as calcium (Lamprecht and La Doux, 2004). Short and long term memory changes are also mediated by changes in the cytoskeletal molecules in parallel with changes which are produced as a result of protein synthesis (Lamprecht and La Doux, 2004). Cytoskeletal molecules are the parts of the cell which maintain its shape and facilitate the intracellular movement of the internal parts of the cell. Proteins that are linked with cytoskeletal molecules often have the ability to bind other things to the neuronal cell (Kandel *et al*., 1995).

The brain is not an equal opportunity recorder of all information received through the sensory processing system. Attentional focus (and the perceptual weight given to the received information) influences what is remembered (Paller and Wagner, 2002; Winkielman and Schwarz, 2001). There is a difference between the laying down of the neural assemblies that form our memories, and the retrieval of information from them. Contextual cues can prompt fuller memory retrieval because contextual memory detail appears to be stored separately from other memory detail (Paller and Wagner, 2002).

Forgetting is yet another process which interacts in the system that we call memory (Paller and Wagner, 2002). Long term inactivation between sensory and motor neural connections results in a one third reduction of dendritic terminals between the two (Kandel, 2000). It is probable that this is another instance of neural pathways falling into disuse and becoming dormant, which can be easily reactivated given the right opportunities. Just like real pathways, neural pathways seem to leave traces of their presence, albeit overgrown (to make use of metaphor) long after they have fallen out of active use. The more deeply worn the pathway over time, the longer the traces persist.

To summarize so far, the system of memory is a complex interaction between the laying down of retrievable memory, in forms of either short term or long term accessibility, and the retrieving of those memories for internal re-experiencing. In some cases, forgetting is part of the process. Retrieval of false memory, or the perceived memory of events

that did not actually occur (see the following section on false memory) is also a part of this memory system.

Short term memory is stored in the medial temporal lobe of the brain, possibly in the hippocampus, because its synapses are 'soft', or particularly capable of rapid plastic change (Alvarez and Squire, 1994). The medial temporal lobe comprises the hippocampus together with the adjacent entorhinal, perirhinal and parahippocampal cortices. Some hypotheses postulate that rather than being stored as a representation in its entirety, short term memories are stored in differing sites in the brain, and that the hippocampus is the mechanism used to retrieve them. Fragments of the same memory may be stored at different regions, for example, the emotional content in one area and the visual content in another. According to the neurons that fire together wire together principle, a cue that triggers memory retrieval of one fragment of the memory will trigger memory retrieval of the remaining fragments. Whether memories are stored as representations or in separate fragments, the medial temporal lobe can locate them holistically upon retrieval. Damage to the medial temporal lobe can produce short term amnesia (Alvarez and Squire, 1994).

Long term memory is transferred elsewhere, away from the site of short term memory, probably to varying locations of specialized cortices in the neo-cortex according to the type of memory, and is no longer vulnerable to disruption. Synapses in the neo-cortex are considered to be 'hard' or less vulnerable to synaptic change, and are therefore more suitable for lasting neural storage. Because hippocampal synapses are capable of rapid change, short term learning can occur from just one experience. Long term neo-cortical memory is more likely to be consolidated slowly, over time. This is necessary for constancy of memory (Alvarez and Squire, 1994).

Some neuroscientists believe that once sufficient neural pathway has been reinforced, the hippocampus is no longer necessary as a mediator, and this change facilitates the change from short to long term memory. The hippocampus is then freed up for more learning and mediation of new short term memory (Alvarez and Squire, 1994). But without the relevant neural pathways being activated from time to time, they, and therefore the memory, will be further and further from the centre of our busy assembly of neural connections, and harder to access (Schacter, 2001). It is the effect of constant attentional focus, or

repeated practice, described in the section on learning and attention, that places things in our short term memories (Kandel, 2000; Wolfe, 1998). Short term memory allows us to make temporary use of neural connections without forming permanent neural pathways that will be of no use to us in the long term. Neural space is freed up again if the memory is not transferred to the more lasting long term memory (Alvarez and Squire, 1994). Once in our short term memory, consolidation to long term memory occurs when we attempt to retrieve the knowledge we placed in our short term memory after a spaced interval (Kandel, 2000; Wolfe, 1998).

The complementary processes connected with the plastic changes to synaptic connections when memory is being transferred from short to long term memory are changes in gene expression and new protein synthesis. Chemical changes between the synapses activate the changes in gene expression. This dictates the formation of new synapses (Kandel, 2000). The gene transcription factor CREB (cyclic AMP response element binding) protein has a role in memory storage. CREB overactivation produces an immediate long term memory, even of experience that normally wouldn't be stored. When the CREB repressor is overactivated, this blocks storage of long term memory (Kandel, 2000).

Long term memory is known to have at least two forms: procedural memory and declarative memory, sometimes known as episodic or explicit memory (Kandel, 2000; Klein and Lax, 2010; Wolfe, 1998). Procedural memory is concerned with actions that we take automatically, such as walking or driving a car, once those skills have been mastered. If we had to pause to remember how to place one foot in front of the other every time we wanted to walk somewhere, it would take us a long time, and procedural memory is an example of our neural connections becoming extremely practised and efficient, firing faster, and requiring fewer neurons to do the same job that once took many. Procedural memory gives us an evolutionary advantage, and frees us up to learn other tasks without having to give any attentional focus to the basic ones we have already learned (Klein and Lax, 2010; Wolfe, 1998).

Declarative memory is our memory for events that we have experienced, and once retrieved, tends to be placed into narrative form. It is therefore sometimes referred to as episodic memory.

Declarative/episodic memory is often able to be recalled as located at a particular and unique point of space and time (Klein and Lax, 2010). Declarative memory also stores factual information, such as places and names, as well as events (Klein and Lax, 2010; Wolfe, 1998). Klein and Lax (2010) call this type of factual declarative memory 'semantic memory'. In contrast to procedural memory, declarative memory requires conscious recall (Kandel, 2000).

Schacter (2001), has detailed what he considers to be the seven sins of memory, or the imperfections of memory, which give us insight into its working. He lists transience (memories are harder to access over time), absent mindedness (forgetting to do things, or forgetting what we have already done), blocking (temporarily inaccessible memories), misattribution (remembering something, but linking it to the wrong context), suggestibility (false memories implanted by suggestion or leading questions), bias (memory skewed to fit our current neural wiring for knowledge and beliefs about the world), and persistence (unwanted memory of traumatic experience, which does not lessen over time, but remains sharp and clear, affecting our thoughts, feelings and behaviour in the present).

fMRI studies show that the left medial temporal lobe and the left prefrontal regions of the brain are not engaged in instances of transience where memories are harder to access over time, and that right prefrontal regions play an important role in the memory retrieval process, active when subjects need to closely monitor small differences in item familiarity (Schacter, 2001).

Sometimes 'blocking' occurs; memory which seems retrievable, but which remains inaccessible (Marll, Wagner and Schacter, 2001; Schacter, 2001). A simple example of this is the saying, 'it's on the tip of my tongue', where the speaker knows that they know the required information, but cannot bring it into consciousness at that point in time. Trying to retrieve blocked data, or reactivate blocked neural connections, seems to involve the prefrontal cortex and the anterior cingulate cortex (Marll *et al.*, 2001). These two sections of the brain also seem to work together when the brain needs to decide between two competing memories, such as whether the composer of the music being listened to is Verdi or Vivaldi.

Anderson and Green (2001) believe that unwanted memory can be suppressed through 'executive control' by using the prefrontal

cortex and the orbitofrontal region of the brain. Their studies show that those people who purposefully suppress memory when memory would normally be retriggered by external cues find it harder to access those memories at a later date. Executive control requires the dorsolateral prefrontal cortex, the anterior cingulate cortex and the ventral prefrontal cortex to be sufficiently developed (Anderson and Green, 2001).

Anderson and Green (2001) suggest that two alternative hypotheses may be that neural pathways are created which divert external cues that would trigger the suppressed or blocked memory away from that connection, or degradation of synaptic neural connections between the pathways processing the external cue and the neural pathways representing the memory. Lack of 'practice' or internal repetition of the memory is very likely to lead to weak or disused neural pathways, as per the knowledge we have on how learning occurs and is stored as neural pathways connected by synapses (Wolfe, 1998). (See also the preceding section on learning and attention.) Active suppression of memory in the presence of triggering cues seems to lead to the formation of neural pathways to avoid it, which eventually become habituated and automatic – well worn neural circuitry. People who actively suppress memory when triggered by external cues find it more difficult to access the memory at a later date than those who have not experienced any external cues of this type (Anderson and Green, 2001).

Some people are not able to suppress unwanted memory, and find it overwhelming. It may constantly be re-experienced unbidden. This is known as Post Traumatic Stress Disorder (PTSD). (See also the section on PTSD in Chapter 5.) Risk factors for PTSD are low hippocampal volume (Bremner *et al.*, 2007; Hull, 2002; McNally, 2003), a traumatic or abusive childhood (Bremner *et al.*, 2007; McNally, 2003), a history of mood or anxiety dysfunction (McNally, 2003), a history of previous stress (Bremner *et al.*, 2007; McNally, 2003), and a negativistic outlook on life (McNally, 2003). Indications are that PTSD occurs when traumatic memory is wrongly stored in the non-verbal, automatic procedural memory, instead of in declarative memory (Brewin, 2001; Hull, 2002). Processing the memory correctly by verbalizing it can help extinguish the visual flashbacks that accompany PTSD (Brewin, 2001).

Data may only be partially stored through neural connections, for example, a first letter of a word or a sound. It is probable that the brain works as Roediger *et al.* (2001) suggest; to fill the gap in the narrative creatively by combining memory with other functions of the brain. Klein and Lax (2010) note that in those with Attention Deficit Hyperactive Disorder (ADHD) episodic memory is poor, but semantic memory is generally intact. They also speculate that those people with Autistic Spectrum Disorder (ASD) may store things differently in semantic memory; separately and discretely, and not as a form of meta-representation, leading to an excessively literal interpretation of language.

What does this mean for counselling?

It is possible that unused connections stored in long term memory are subject to competitive plasticity, available to be overwritten with memory that is more constantly retrieved, and certain that neural connections stored in short term memory in the medial temporal lobe, accessed by the hippocampus, are affected by competitive plasticity. This is an example of the maxim, 'use it or lose it'. Transience and misattribution are probably at least partially caused by competitive plasticity, where the neurons forming the pathways in the brain are underused and subverted to other purposes.

The more we 'practise' our memories through revisiting them, and in doing so strengthen the neural pathways representing those memories, the stronger they get. It is possible for us to shape our remembered pasts by paying more attention to some memories than other memories, or to some components of a memory than others. The regularly trodden pathways are most easily accessible, and often it is automatic that we will choose those pathways, so familiar are we with them.

Because of the power we have to mould and shape our memories, it is little wonder that we all remember things differently, even if we experienced what we remember together. When you add in misattribution, suggestibility and bias, it is not surprising that people can remember the same experience so differently, and therefore perceive it differently. We create our pasts as surely as we create our futures.

However, most people believe their remembered pasts to be solid, unchangeable fact which they have no power over. Part of the 'magic' of counselling is to be able to support people to rehearse their memories in different ways, creating new pathways from the same experience, which leads to a different perception of their past and often to different feelings and thoughts about it in the present. This in turn can generate different ways of behaving.

Because our memories are not always accurate due to the fact that we fill in any gaps in order to make a coherent narrative, it can be helpful for counsellors to discuss with clients how details of events that seem to be solid memory may not be so, even though we have formed strong neural connections for them by revisiting the parts of them created by our imagination when we have revisited the fragments of the memory which we truly remember. Knowing that our memory is not solid makes it easier for clients to work with memory in a fluid way, where reframing and re-contextualizing is possible, and where clients can experiment with different attentional focuses. Naturally, it is unwise for a counsellor not to value the learning and expertise that the client brings, including in the form of memory.

Procedural memory is obviously in action very early in life, as children learn to smile, frown, grasp, walk, talk and climb. Declarative memory seems to come later, possibly because of the huge amount of activity that must be taking place in the procedural memory. Some people claim to have traces of declarative memory from before the age of two, others not before the age of four. Whichever applies, long term declarative memory is the foundation upon which our view of the world is built. Everything we learn through experience affects everything else that comes after, meaning that each of us has a unique and complex set of neural connections through which we filter the world. There is an apt saying which goes, 'Life is lived inside your head. Everything is a perception, not a truth' (source unknown).

Simple 'tip of the tongue' type blocking of memory tends to involve proper nouns (names of people and places) and may reflect the fact that our memory usually uses imagination to fill the gaps in our narratives, as supplied by memory. It is more difficult to use imagination for the purpose of creating names, because these are verifiable facts. Semantic memory requires conscious recall. A counsellor working with a client who is struggling to remember things could suggest that they focus

on the place and time in which the memory occurred. Any contextual cues that they can recall or obtain from others who were also present when the remembered event took place are likely to promote a more comprehensive memory (keeping in mind that the imagination is prone to filling in gaps in our memories…).

Blocking of parts or the whole of distressing events in the absence of damage to the brain probably reflects that the pain caused by these events means that the client chose not to think about them, and therefore rehearse them through thought, and the neural pathways have become weak through disuse. In order to create neural pathways repetition, in this case of rehearsed thought, is needed. Facilitation of the transfer of information from short term to long term memory requires the revisiting of the neural pathways (i.e. in this case, remembering) after an initial spaced interval following the repetition.

Trauma often creates strong memories, and repeated trauma more so, because the subject usually spends a lot of neural effort seeking to make sense of and come to terms with what has happened, but for those who have sufficient control to suppress thoughts relating to the traumatic event and turn their thoughts elsewhere, it is possible to block the formation of memory and the strong neural connections which would represent it, or to allow the neural connections which it represents to fall into disuse and to become inaccessible without effort. In these cases, blocking appears to represent a choice that has protective value for the client, and should the client wish to continue this protective stance, this should be respected by the counsellor.

If clients wish to block out memories that are causing them distress, and have been so far unsuccessful in doing so, one method which could be supported by the counsellor is the mental hook/pause button/eraser technique. Known by a variety of names, this is a single technique linked to different metaphors. The client first decides on a preferred 'fantasy', which could be the replaying of a pleasant memory as equally as a pleasurable vision of the future. When thoughts connected with the unwanted memory are experienced by the client, the unwanted memory is hung on an internal hook/paused with an imaginary remote control/rubbed out with an imaginary eraser. The client then immediately switches to thoughts of the preferred fantasy. Because unwanted thoughts are sneaky, they often re-emerge as part of the preferred fantasy, and the client must use the technique again and

replace it with a version of the preferred fantasy that does not contain thoughts of the unwanted memory as many times as necessary. In this way the brain can be neurally retrained not to revisit and strengthen the neural pathways for the unwanted and unpleasant memory. (Note that this technique can also be used with clients who have specific, distressing worries about the future.)

It is important to share this technique only when clients express distress about not being able to cope with unwanted memories, because in general it is healthy for the brain to process and come to terms with the unpleasant memories we have, and to build neural connections in response which support and incorporate our learnings from that experience. Counsellors should also keep in mind that clients with more fully functioning and developed brains will more easily be able to have the executive brain control needed to suppress traumatic memory at will.

Other clients may come to counselling ready to create safe memories of an unpleasant, distressing or traumatic event that they have formerly repressed, or which is still present and affecting their lives in unwanted ways. Counsellors who help clients recover or rebuild a memory of a traumatic event should remember that there are many perceptions of truth depending on the angle from which it is viewed and the data that are emphasized, and support clients to build memories where they are able to understand their own reactions and responses to trauma in context, and which concentrate on the positive elements of these while simultaneously acknowledging that there will have been external things which were out of the client's control. Strengths based approaches, where client and counsellor notice positives in the client's reaction and response to trauma, are helpful here.

Rewiring of neural connections for remembering the past can be beneficial because past learning is what our responses to the present and our predictions for the future are built on. Skills such as walking and driving a car are stored in procedural memory, where a neural shortcut is used to prevent us from having to think through each of those things every time we practise that skill. Many behaviours and responses are likely to be neurally wired to cues and triggers which evoke stored memories. If these are stored in procedural memory, we are acting and responding automatically without new cognitive evaluation.

Some examples might be eating when we're stressed, avoiding specific situations, or feeling rejected when we experience things that remind us of the times when we've experienced rejection in the past. These things will be part of our automatic procedural memories if they are strong neural connections which have been practised, repeated or rehearsed many times for a prolonged period. Often, we don't fully (or even partly!) understand why we do what we do. Counselling is an opportunity to examine our present behaviours and responses to stimuli and our predictions for the future by looking at what created these neural pathway shortcuts. It allows us to evaluate whether these shortcuts are helpful or harmful to us, and if we decide that they no longer benefit us, to practise stopping when we notice the triggers so that we can consciously choose new behaviours and create new pathways. Naturally, this needs to be practised, repeated or rehearsed for a prolonged period as well if we wish to make the pathways permanent. A good counsellor will support a client in these endeavours, and explain the process for how this can happen.

False memory

What do we know?

Contrary to popular belief, memory is not stored in playback form (Gonsalves and Paller, 2002). It is not a tape, a photo, a recording or a book that stays true to itself. Sometimes, memory is constructed from incomplete data (Gonsalves and Paller, 2002). Either we didn't have full access to all the data in the first place, we have not stored all the data in our long term memories, or we have lost our ability to retrieve some of it (in either the short or long term). Human beings like to construct whole narratives to make sense of the world, either in explaining to ourselves, or someone else (Roediger *et al*., 2001). This predisposition may open the door for Schacter's (2001) memory 'sins' (see the previous section on memory) of misattribution (linking real memories with other stored memories in the wrong context), suggestibility (allowing unverified or known false information to be remembered as real), and bias (where memory is skewed to fit in with

our beliefs and the way we view the world). It is probable that when presented with gaps, we simply 'make it up' to create a whole memory (Roediger *et al.*, 2001).

fMRI, positron emission tomography (PET) and event-related potential (ERP) scans all show that similar regions of the brain construct true, verifiable memories and false memories that the subject believes to be true (Gonsalves and Paller, 2002; Schacter and Slotnick, 2004). Scans show greater activity in the brain when perceptual, sensory and contextual data were required to be recovered when the memory is a true one, presumably because these data have actually been encoded through sensory neural pathways (Schacter and Slotnick, 2004).

Regions within the medial temporal lobe, including the hippocampus, are involved with storing and retrieving associated information as well as the storing and retrieval of actual memories, and this can lead to neural confusion, and the formation of fake memory (Schacter, 2001). If a false statement is made, and the listener is told that it is false, probability still increases that in the future the listener will believe the statement to be true. Confusion seems to exist between familiarity and truth (Schacter, 2001; Schacter and Slotnick, 2004). Being warned about the possibility of incorrectly recognizing fake suggestions as truth does not prevent subjects from continuing to do this (Roediger *et al.*, 2001).

False memories are most susceptible to being created when an implanted suggestion overlaps with an actually occurring experience. It can be difficult to distinguish similar events, and sometimes similar experiences can become merged into one memory in an efficient use of neural storage space (Gonsalves and Paller, 2002).

Memory is stored learning, and we most easily store what fits into the assembly of neural connections we already have (Doidge, 2007; Wolfe, 1998). (See also the section on learning and attention in Chapter 3.)

When people are given lists of similar types of descriptive words, such as a group of words all associated with taste, and are then asked to recall them later, they are likely to (incorrectly) recall a taste associated word such as 'sweet', which wasn't in fact in the original list (candy, sour, sugar, bitter, good, taste, tooth) (Marll *et al.*, 2001; Schacter,

2001). This shows that we sometimes store our knowledge as global generalizations; in this case that the list was comprised of 'taste' words.

Greater prefrontal cortex and medial temporal lobe engagement is correlated with effort to filter out false truths. Illusory truth incidence (believing something false to be a true memory) is reduced when people recollect specific details about statements they encode, rather than just remembering it generally. Higher brain activation leads to clearer memory, caused by deeper, more specific neural pathway encoding, and this type of brain activity is more likely to retrieve factual rather than illusory memories (Schacter, 2001; Schacter and Slotnick, 2004).

We remember our imaginings, just as we would remember actual events (Gonsalves and Paller, 2002). Remembered imaginings are more likely to involve the higher sensory processing regions rather than the primary sensory processing regions that deal with external sensory input (Schacter and Slotnick, 2004). The more strongly and vividly a false event is visualized or imagined, the more likely it is to be later mistaken for actual memory. And the more often a false event is visualized, the more likely it is later likely to be recalled as true (Paller and Wagner, 2002). This is unsurprising, given that repeated practice strengthens neural connections.

Some studies show that in general, the richer in perceptual detail a recovered memory is, the more likely it is to be true (Gonsalves and Paller, 2002). Imaging technology shows greater activity in the auditory cortex during the retrieval of true memories, and this could be because subjects are retrieving stored language or sound related to that memory (Gonsalves and Paller, 2002). Schacter and Slotnick (2004) have found similar activity in the visual cortex when a subject is retrieving true memories. Events that actually took place involve the gathering of sensory data from the external world (Roediger *et al.*, 2001). It is probable that as imaging techniques continue to improve that it will become possible to assess whether any given memory is comprised primarily of illusory truth or of valid truth (Gonsalves and Paller, 2002).

McNally (2003), after examining studies for and against 'recovered' memories of sexual abuse, warns that highly traumatic memories are more likely to be experienced as PTSD (see also that section in Chapter 5) and that the majority of evidence shows that 'recovered'

memories of sexual abuse remembered during talking therapies are likely to be false memories of illusory truth.

Despite the right hemisphere being the hemisphere of the brain most associated with creativity, the left frontal cortex has a role in the creation of possible narratives, and the hippocampus in the right prefrontal cortex balances this out by filtering for validity indicating actual memory according to other existing neural connections. Anyone who has a damaged or underdeveloped right prefrontal cortex will be more prone to false memory (Bolte-Taylor, 2008; Schacter and Slotnick, 2004). (See also the section on left brain/right brain in Chapter 2.) This seems also to be due to an inability to focus on detail, and instead to concentrate more globally on themes, or 'gist' to recover memory, rather than to recall accurate specifics (Schacter and Slotnick, 2004). The more closely related false information is to information that the subject has actually been given, the more likely they are to recall it as true (Roediger *et al.*, 2001). This may be illustrative of neural connections wired for global theme rather than specific detail, and is borne out by the experiments with word association lists (Marll *et al.*, 2001; Schacter, 2001), described previously.

Damage to the left orbitofrontal region of the brain means subjects have difficulty locating the 'source' of memories, one of the ways our brain filters for false memories, and these people are often prone to confabulation (Schacter and Slotnick, 2004). The left hemisphere is also normally concerned with detail (Bolte-Taylor, 2008). In normally functioning brains, the left and right hemisphere work in tandem, joined by the corpus callosum (Bolte-Taylor, 2008), and where memory is concerned, this may be a useful adaptation to allow the imagination to complement actual memory to create a full narrative to help us understand and make sense of the world, and therefore create a basis for making decisions about how to respond to incoming stimuli.

What does this mean for counselling?

If what we become familiar with is easily confused with truth, what we say to others about themselves, and what they say to us about ourselves is of high importance. People who constantly hear that they are stupid, dumb, lazy or ugly will confuse the apparent opinion of the

person who makes these statements with the truth, and code this as such in their neural wiring. Repetition will reinforce it. Counsellors often work with people who have poor self-esteem and/or self-image because of negative verbal putdowns over a prolonged period of time. The sources of these putdowns can be parents, siblings, teachers or relationship partners, former or current.

A counsellor has two roles here, first to support the client if their goal is to remove themselves from the source of the putdowns, and second to help the client examine their lives for evidence which would contradict the putdowns. For instance, someone who has been repeatedly told that they are lazy might be encouraged to remember occasions when they were energetic, or when they put effort into something. Someone who has been told that they are ugly might be encouraged to remember and focus on occasions where they received compliments about their physical appearance from others, or when they noticed something attractive about themselves. (The narrative and solution focused techniques of looking for exceptions to the story are useful here.) In this way, new neural connections and pathways begin to form, although it must be stressed that like all learning, they will require repeated practice, something which can be built into the client's action plan towards achieving their goals. This can take the form of CBT 'homework', which is really just practice done by the client between sessions to facilitate new learning and the forming of new neural pathways.

Because we are vulnerable to false memory, counsellors need to be careful not to unintentionally 'lead' their clients into false memory through suggestion. (In recent decades, this has become of particular contention in the area of 'recovered', or suddenly remembered, memories of sexual abuse.) This is also relevant when working with children, who may disclose abuse against them that we need to report in order to protect them. Counsellors need to be fully aware that even things that we know not to be true when we first hear them can seem to be true when we later recall them. A client who has specific detail around a memory is more likely to be remembering a true event, but not necessarily, because vivid imagining and re-imagining of detail can produce that detail as a specific and complex memory at a later time.

People with poor prefrontal cortex development (left and/or right) or damage to this area, are likely to have more experience of false memory.

This can take the form of lack of detail in memory, misattribution (or failure to recall the memory in its correct context), or confabulation (mistaking ideas, thoughts and incorrect information for true fact). Not every client a counsellor deals with will have the capacity to be 'truthful', although because memory is such a subjective matter of perception, truthfulness is already a loose concept. Confabulation is easily mistaken for intentional lying, which is more usually a survival skill to avoid unpleasant consequences. Confabulators are generally the ones who seem to be 'making things up for the sake of it', and the ones who have increased suggestibility. Because consistency of perception is important for counselling to be effective in taking advantage of the brain's plasticity, it can be highly frustrating for counsellors to deal with clients whose brains seem disorganized and where the pathways are a random jumble of confused connections.

The counsellor's approach should differ in relation to clients who lie (who can plan to make new neural pathways to not doing so when it is safe not to, if this is their goal) and people who confabulate, who need support to optimize the function of their brains by increasing the neurotrophic factors which promote brain plasticity. (See also Chapter 6.) Counsellors should also make allowances for the decreased brain capacity of confabulators to recognize fact from fiction and true memory from illusory memory.

Purposefully implanting false memory in a client's brain is something that would be ethically inappropriate if the client was not a partner in the goal to do so. But there may be occasions where a client is troubled by a specific memory and wishes to overwrite the traumatic nature of that memory with one that is more neurally helpful. For instance, a client who is unable to trust relationship partners because their parent constantly rejected them may wish to work with their counsellor to take advantage of a technique sometimes known as 're-storying'. Transparency is key here for the counsellor, and while the counsellor may make the technique known to the client, the client must be the one to choose to use it or not within the counselling session.

The re-storying technique involves the client remembering the initial memory and the counsellor helping them to re-imagine the traumatic component by guiding them through visualizing the memory while replacing key parts of that memory with different visualizations. This is best used not in cases where there has been

a definitive and unchangeable outcome, such as a death, but where there is the perception that if people could have behaved differently, the client would have experienced a different emotional outcome. For instance, a client could re-imagine the memory with a support person present in the memory, or re-imagine another person in the memory behaving differently: a parent hugging them instead of turning them away, or a teacher praising their effort at public speaking instead of humiliating them.

The client should be encouraged to add as much perceptual detail as possible, for example, what they and other people are wearing, what the fabric feels like, and what colour it is, what the light is like and any smells or sounds that might be present. The more sensory detail imagined, the more likely it is to form neural connections and be stored and acted on as a real memory. The client's role in creating this memory is to repeatedly practise it and to carve out new neural pathways for it, which will become strong in comparison to the formerly remembered 'true' memory, which was subject to perception anyway.

In this way, counselling, if practised in a client centred way, can assist clients to not only create their futures, but to recreate their pasts in ways that help them to function better in the present.

Memory and self

What do we know?

Information that is neurally encoded in relation to our own selves gets better memory storage. Self-referent information activates the medial prefrontal cortex, which is a different region to that used for other normal memory encoding (Klein and Lax, 2010; Schacter, 2001). Trait self-knowledge (i.e. knowledge of the sort of person we perceive ourselves to be) can remain when neural or cognitive damage means that other types of memory do not. For instance, in cases of amnesia, which affects declarative memory (primarily episodic memory, but also semantic memory) most patients are left with their trait self-knowledge intact. Even people with neural damage who cannot

recognize themselves visually can still have a knowledge of their own personality traits (Klein and Lax, 2010).

Research suggests that trait self-knowledge is stored in the semantic memory – a subcategory of declarative memory – as a set of facts that we know about ourselves. We don't need to recall events to infer these things. But trait self-knowledge is more resilient than other semantic knowledge, such as knowledge of our age, or our partner's name (Klein and Lax, 2010). Amnesiac patients can update their trait self-knowledge even while unable to access any episodic memory. Amnesiacs who perform poorly on a task, while rating their trait self-knowledge highly in that area of competence, exhibit distress, but do not exhibit distress when the reverse is true, demonstrating the resilience of trait self-knowledge (Klein and Lax, 2010).

In contrast, patients with brain degeneration, such as Alzheimer's, are unable to update their trait self-knowledge and rate their trait self-knowledge according to their pre-Alzheimers' personalities. Similarly, they cannot update their knowledge of the trait characteristics of their loved ones (Klein and Lax, 2010). And in the wider population, trait knowledge of others does not show the same resilience as our own (Klein and Lax, 2010).

Trait knowledge seems to be neurally created by a set of interrelated but functionally independent systems. It is unknown how these systems interact with each other to create a sense of unity of self-knowledge (Klein and Lax, 2010). As well as personality traits, Klein and Lax (2010) list other systems that contribute to a sense of self. They include: episodic memory of life events (including those in the public domain that relate collectively to society), semantic memory for the facts of one's own life, e.g. birthplace, the experience of continuity through time (probably related to episodic memory), and the expectation of continuing into the future, a belief in the ability to have the agency to affect one's own future, the ability to self-reflect, and the ability to recognize and neurally represent our own body.

When retrieved, events from our episodic memories are re-experienced with a conscious awareness that they happened to ourself. Consistency of trait self-knowledge contributes to a unified sense of ourselves over time. Both episodic and semantic memory, each part of the declarative memory system, are likely to contribute in different ways to trait self-knowledge (Klein and Lax, 2010). There are no

recorded cases of a person losing all trait self-knowledge and retaining other components of a unified sense of self, which suggests that trait self-knowledge is the most fundamental component of that sense of self (Klein and Lax, 2010).

fMRI studies show that both remembering the past and imagining the future use similar processes in the brain (Addis, Wong and Schacter, 2006). There is a specific memory system for remembering and imagining self-relevant events, as evidenced by the fact that damage to the brain which impairs these abilities leaves intact memory and predictive skills for political, national and public events (Addis *et al.*, 2006). There is no evidence of a region of the brain that reconstructs past events that does not also process predictions or imaginings of the possible future. In many parts of the processes of remembering and predicting self-relevant events, brain activity is identical. When not identical, the process is still taking part in the same region of the brain. The neural overlap is most extensive during the elaboration stage of past recall and future imagining (Addis *et al.*, 2006). Imagining the future creates more brain activity than remembering the past (Addis *et al.*, 2006).

Suicidally depressed patients who show memory impairment show a corresponding inability to visualize their future (Addis *et al.*, 2006). A person's ability to construct detailed and specific predictions of the future is correlated in time with an ability to clearly recall the past. For instance, if someone can clearly recollect detailed events up to two years ago, but not earlier than that, that is how far ahead they will be able to construct detailed possibilities for their future. Someone who can detail something that happened three decades ago will be able to make detailed future self predictions for three decades into the future (Addis *et al.*, 2006).

Lack of detailed recall of childhood memory can lead people to believe that their experience must have been negative, and sometimes to believe that they are suppressing unpleasant memory, although there is no evidence to show this is likely to be the case (Winkielman and Schwarz, 2001). Addis *et al.* (2006) postulate that the function of memory is to provide experience on which to build response and reaction to future events, and to provide learning which helps to plan for these future events. In this scenario, memory exists to provide an adaptive advantage. The ability to use memory to simulate future

events means that we can modulate our present behaviour to allow for future needs to be met, and for our goals for the future to be achieved. They also postulate that some distortion of memory may be accounted for by what we wish to use it for in creating and imagining our futures.

What does this mean for counselling?

It is futile for counsellors to attempt to change a client's self-knowledge without the seeds for change coming from within a client. Self knowledge about personality traits can only be updated internally. The most a counsellor can do is to point out evidence in the client's own stories which might challenge those beliefs, and encourage the client themselves to re-examine that particular belief. The counsellor must keep in mind that changes in beliefs about the self need practice to become permanent neural pathways, and this will only happen if the client is motivated to change those beliefs and replace them with new ones. A client's knowledge about themselves will not change just because a counsellor believes that it should, or because the counsellor works to change it. (This is good to remember when parents send their children to us so that we can wave a wand and present them with better self-esteem!)

A counsellor's role is to support clients to look for exceptions and new evidence when the client is open to updating their trait self-knowledge in a positive way. Because trait self-knowledge is a fundamental part of our memory, it is clearly a fundamental part of our learning, and therefore how we view the world and react to it. When counsellors and clients find themselves going in circles in terms of behaviour, thought and feelings, and no progress is being made towards the client's goals, it could be helpful to review the client's trait self-knowledge and see how this is contributing to this situation.

For suicidally depressed clients with significant memory impairment relating to past events in their own lives, or even difficulty remembering detail from a few days ago, counsellors can assume that there will be a corresponding inability to imagine or predict a future that is different from the internal emotional suffering they are experiencing in the present. How far forward they can visualize will probably be correlated with how far backwards clear, detailed memory

extends. Any action that a depressed patient can be supported to take that increases the neurotrophic factors, such as BDNF, which support brain plasticity and an optimally functioning brain (e.g. exercise, healthy eating and doing things that will produce quality sleep) will improve attention, concentration and learning, and also memory. (See relevant separate sections elsewhere in the book.) This will correlate to an increased function for visualizing the future, which is necessary if a client is to visualize a future without the present depression in it.

This is more complex than it sounds, because depressed clients often lose the will and motivation to take these actions. For some people, a referral to a mental health professional for pharmaceutical intervention may provide the bridge they need to be able to take these actions for improving brain function. For others, support from family, friends and other significant people in their lives may provide the necessary motivation and/or accompaniment to get them started. Counsellors can play a role in both referral to mental health services, and in helping the client to identify their natural support networks.

A client who lacks the ability to visualize the future may have blocks to remembering the past, possibly due to trauma experienced. The role of the counsellor in these cases could be to practise future visualization with the client, by helping them to visualize one of the millions of possible futures that could be theirs, preferably a pleasant one! Starting the visualization with something non-threatening, such as their dream house or preferred location, helps many people get around the blocks to visualizing a future with themselves in it. Counsellors should ask for specific detail, such as the colour of the house, because when visualization is difficult, it helps to have a smaller decision to make. Asking for too much detail at once can be overwhelming for a client who finds future visualization difficult. Once one detail is ascertained, the counsellor can ask for another, and another, until a larger picture is built up. If the client is able, within that session, they may be able to place themselves in that future visualization, but for other clients, this may take several sessions. Neural connections for new skills strengthen with practice.

Clients and counsellors can experiment with how far into the future they wish to create a potential future, for example, five or thirty years time. It is my experience that creating a future sometime ahead is

easier for clients, because then they don't feel constrained by current barriers, such as finances or family obligations.

Counsellors should remember that as ability for visualizing the future improves, some clients may be troubled by increased experience of past traumatic memory, and be prepared to assist clients with processing this, should it arise. Other clients who practise visualizing the future could find it improves their overall memory recall. Likewise, when counsellors assist a client to recall positive childhood memories, this has the power to change the client's perception of their lives. When negative stories are dwelt on to the exclusion of all else, the client misses out on being able to build a perception of a safer, happier world. Not being able to access this opens the door to anxiety and depression (see also Chapter 5).

Memory distortion in general is a normal part of life, and counsellors should not worry over 'the truth'. The exciting thing for the field of counselling is that perception of the past has direct bearing on our present and our futures, and forming new perceptions of the past is within our own capabilities, whether we are client or counsellor. We literally have the opportunity to create ourselves and counselling can be part of this process.

Chapter 4
Other Workings of the Brain

Mirror neurons

What do we know?

Mirror neurons were first discovered in the brains of Macaque monkeys, and have since been located in human brains. We all have mirror neurons. They are a special class of neuron found in the pre-motor cortex of primates (Grezes *et al.*, 2003; Iacoboni *et al.*, 2005). Ordinary canonical neurons will fire at the presentation of an object, while mirror neurons fire when merely observing an action involving that object (Rizzolatti and Craighero, 2004). Mirror neurons literally mirror and replicate the neural pathways that activate when we perform an action (such as holding, grasping or tearing) or when we speak or feel (Gallese *et al.*, 1996; Grezes *et al.*, 2003; Iacoboni *et al.*, 2005; Pines, 2003; Rizzolatti *et al.*, 1996; Umilta *et al.*, 2001). For instance, if I watch you kick a ball and my brain was scanned in that instant of watching you, the same neural pathways would light up in my brain as would if I had actually kicked it myself. Your neural pathways for ball kicking are activated, but so are mine; although I have only observed this action.

Some mirror neurons are 'strictly congruent' (about one-third of all mirror neurons) and fire the same pattern, whether I am observing or performing a specific action. Other mirror neurons are 'broadly congruent' (representing the other two-thirds of the neurons in the mirror neuron system) and fire the same pattern in response to similar actions observed or performed. For instance, broadly congruent mirror

neurons will fire for different types of object grasping (Gallese *et al.*, 1996; Iacoboni and Dapretto, 2006; Rizzolatti and Craighero, 2004).

Pines (2003) links the mirror neuron system with the neural foundations of procedural memory, where well used neural pathways for commonly performed actions fire automatically. This is because observation is often the initial trigger for our own motivation to learn, perform and practise a skill, and when we are competent at that skill, it becomes part of our procedural memory base. It is probable that the pathways laid down by the mirror neurons during the process of observation help to build our set of neural pathways representing social skills and responses.

So far, in humans, mirror neurons have been identified in two cortical areas, the posterior part of the inferior cortex, and the anterior part of the inferior parietal lobe. These areas are closely connected and form an integrated fronto-parietal mirror neuron system (Iacoboni and Dapretto, 2006; Rizzolatti and Craighero, 2004; Rizzolatti and Luppino, 2001). Mirror neurons in the premotor and parietal regions of the brain provide plausible neural mechanisms for a wide variety of important social behaviours, from imitation to empathy (Iacoboni and Dapretto, 2006).

Human neurons are more evolved than the mirror neurons of other primates and can code each part of an action and not just an action as a whole (Rizzolatti and Craighero, 2004). The spinal cord system shows a depression in activity when mirror neurons fire, suppressing automatic, uncontrollable, reflex imitation of observed actions (Rizzolatti and Craighero, 2004). The failure of this process is echopraxia, the compulsive imitation of other people's gestures, irrespective of the appropriateness or safety or doing so. Echopraxia is seen in some patients with severe psychiatric disorders (Rizzolatti *et al.*, 1999).

It is most likely that mirror neurons have enabled humans to develop the tool of language, so that we could copy what we heard from others. Some mirror neurons are specific to mouth action, such as eating and drinking. Others are specific to language. Those mirror neurons involved with communication are still involved with ingestion processes, suggesting that they evolved from the basic eating and drinking neurons. It makes sense that humans were eating and drinking long before they could communicate vocally, and that this

process of evolution has occurred (Ferrari, Rozzi and Fogassi, 2005; Rizzolatti and Craighero, 2004).

There are aural mirror neurons related to hearing. These mirror neurons mean that if I hear something happening, rather than directly witnessing it, my mirror neurons will light up as if I have observed the action, rather than just heard the noise(s) arising from it (Iacoboni *et al.*, 2005; Kohler *et al.*, 2002; Rizzolatti and Craighero, 2004). Mirror neurons do not fire if the observed action is only mimed (Gallese *et al.*, 1996). Mirror neurons can fire when part of the action is hidden from view, and the observer only sees part of the action, but infers the completion of that action (Umilta *et al.*, 2001).

Functional magnetic resonance imaging (fMRI) studies show greater mirror neuron activity in the inferior frontal cortex when the observed action has a context, such as grasping a cup, either to drink from it, to refill it or to put it away, and noticing whether the cup is empty or full. This suggests that mirror neurons also code the intention of the observed subject, as well as the action. It enables subsequent actions to be predicted (Gallese and Goldman, 1998; Iacoboni *et al.*, 2005). Mirror neurons provide a basis for empathy, or understanding others, by being able to experience what they are experiencing in their brains at the same time as they do. Iacoboni *et al.* (2005) showed that mirror neurons trigger the activation of another subset of neurons in the inferior frontal cortex when they fire, facilitating this coding for intention. When the mirror neuron system codes for social intention, it allows us to neurally experience the motivations of others (Dapretto *et al.*, 2005; Iacoboni and Dapretto, 2006; Rizzolatti and Craighero, 2004; Rizzolatti *et al.*, 1999).

The special mirror neurons for the mouth, linked with eating, drinking and language, are also linked to facial expression, suggesting that mirror neurons have a role in the understanding of the emotional states of others, perhaps also providing clues as to intentions arising from those emotional states (Iacoboni and Dapretto, 2006). Mirror neurons are activated by the imitation and observation of facial expression in normally developing ten-year-olds, and the extent to which this is happening in each child's brain is correlated with empathetic concern and interpersonal competence (Pfeifer *et al.*, 2008).

Yawning, smiling and laughing tend to be behaviourally mirrored by those who observe them and are known as 'resonance behaviours'

(Rizzolatti *et al.*, 1999). Perceiving action in others through the mirror neuron system can induce motor resonance – a disposition (but not a compulsion) to copy the observed action (Dapretto *et al.*, 2005). Performing an action may also prime our mirror neurons to be sensitive to perceiving the actions of others who are acting in a similar way to us, and therefore play an important role in social connection and in founding successful social interactions and relationships, with mutual empathy and shared motivation forming the basis for unified action (Dapretto *et al.*, 2005).

Mirror neurons are also implicated in the retriggering of addictions. Drug related cues excite brain activity associated with craving in current or former addicts (Kauer and Malenka, 2007; Kelley, 2004). When people observe other people acting in a way that has brought them neural reward in the past, such as the observation of others handling or using drug paraphernalia, those neural pathways are especially vulnerable to being reactivated, possibly through the mirror system.

Mirror neuron systems may provide the basis for contagious behaviour. In flocking shore birds, this takes the form of the entire flock taking flight because one or two birds start wing flapping. The others around them imitate this behaviour until they are all doing it, even though the majority of the flock doesn't know where or why the behaviour originated. This type of behaviour has probably developed to confer evolutionary advantage (Rizzolatti *et al.*, 1999) through greater safety from predators.

The mirror neuron system and the limbic system (which is linked to feeling and emotion) are anatomically connected by the insular cortex (Augustine, 1996), making the mirror neuron system an ideal location for neural processes involving empathy (Dapretto *et al.*, 2005; Iacoboni and Dapretto, 2006). People who are more likely to be skilful in imitating others, and to do so in social situations, are in general more empathetic towards others (Chartrand and Bargh, 1999). This includes accent, language structuring, body language and facial expression. When monkeys were exposed to the sight of a researcher using tools to eat, initially none of the monkeys' mirror neurons fired, but this changed after repeated exposure to the researcher's actions. The mirror neuron patterns fired in the monkeys' brains were for neural pathways which did not include the use of the tool, but which coded the intention of the researcher, in this case to

eat (Ferrari *et al.*, 2005). This suggests that the mirror neuron system can be shaped by experience (Iacoboni and Dapretto, 2006).

Pines (2003) uses exposure to people with good social skills and high empathy to help those lacking in these things to develop the necessary neural pathways, and therefore skills, as a form of therapy, and describes changes in emotional capacity in some subjects. Other therapists also use empathy training for both children and adults, which involves observation of the actions of another and then practising exploring/inferring the emotional state and/or needs of the subject. The Canadian Roots of Empathy project teaches these skills at school and preschool level (Gordon, 2005).

Activity in the mirror neuron system increases when the subject is observing two people interacting (Iacoboni *et al.*, 2005). This could be because there are more complexities to observe, but it is more likely to be because the mirror neurons are busier coding the social interactions (Iacoboni and Dapretto, 2006). In adults, mirror neuron systems are most active when observing someone with skills or actions most like our own, for example, a ballet dancer watching another ballet dancer, as opposed to a dancer of another genre watching a ballet dancer, or a non-dancer watching a ballet dancer (Calvo-Merino *et al.*, 2005; Dapretto *et al.*, 2005).

People have greater mirror neuron activity in the premotor and inferior parietal regions of their brains when observing videos of their own actions than they do when observing videos of others' actions. Static images on screen can cause mirror neurons to fire too (Johnson-Frey *et al.*, 2003; Rizzolatti and Craighero, 2004), but this effect is not as strong as watching actions in person (Iacoboni and Dapretto, 2006). There are links between an infant's ability to self-recognize, for example, to understand that a reflection in a mirror is their own, and the extent of their imitative behaviour (Asendorph and Baudonniere, 1993), suggesting that self-recognition also involves the mirror neuron system (Iacoboni and Dapretto, 2006).

Rizzolatti *et al.* (1999) suggest a difference in ability between humans and other primates to store and build upon the firing of pathways in the mirror neuron system. A complicated concept is 'reciprocal mirroring'. This takes the form of mirroring back to others their own behaviour by copying it, as we do with young children. This allows a child to build a sense of self by having their mirror neurons

activated by our actions, perceiving themselves through perceiving us, and forming self-identity through the understanding of their own actions. This behaviour is thought to take place between adults on a more subtle level (Dapretto *et al.*, 2005).

The intensity of mirror neuron activation is not affected by the nearness or distance of the observed action (Rizzolatti and Craighero, 2004). Pines (2003) believes when people who have had a stroke deny their own one-sided paralysis, as is common, and also often deny noticing any movement or any actions on the affected side, that among other damage to the brain, the mirror neuron system that controls that side of the body has also been damaged, leaving the person unable to neurally experience or take note of any actions or behaviour on that side of the body.

There are structural abnormalities in the mirror neuron regions of the brains of those affected by autism (Hadjikhani *et al.*, 2006). Studies have shown temporal (time) delays in neural connections between the regions of the brain involved with the mirror neuron system in those who have Autistic Spectrum Disorder (ASD) (Just *et al.*, 2007; Nishitani, Avikainen and Hari, 2004; Villalobos *et al.*, 2005). Mirror neuron activity is associated with Mu rhythm suppression in the brain (Hari *et al.*, 1998), and this suppression was lesser in the brains of those diagnosed with ASD, which includes those with both autism and Aspergers, than in the brains of a control sample without ASD (Oberman *et al.*, 2005). A recent fMRI study showed reduced mirror neuron activity in those with ASD during the imitation of finger movements (Williams, 2006).

There is strong evidence to support mirror neuron system dysfunction as a core deficit in autism, and that levels of neural activity in the areas of the brain governed by the mirror neuron system could be an effective bio-marker for the impairment of patients with ASD (Iacoboni and Dapretto, 2006).

There is a direct relationship between lack of activity in the mirror neuron system of ASD children and symptom severity. Conversely, the greater the activity within the mirror neuron system, the more highly functioning a child is. Brian activity in the insular and the limbic structures was also correlated with increased social function (Dapretto *et al.*, 2005). Studies have shown that imitating children with ASD can help those children to learn to imitate others in turn

(Dapretto *et al.*, 2005). Damage to the mirror neuron system can affect the ability to predict action or intention of others, through body language or facial expression (Dapretto *et al.*, 2005). fMRI scans show that ASD children pay attention to facial expression, and can imitate facial expression, but a lack of mirror neuron activation means that the processes for understanding others' motivations, and for having empathy for others as well as an ability to understand the nuances of social interaction, are affected. There is evidence that we can only perceive and understand in others what we are capable of perceiving and understanding in ourselves, and a damaged or non-performing mirror neuron system will directly affect this (Dapretto *et al.*, 2005).

What does this mean for counselling?

The presence of mirror neurons in human brains suggests that the basis of much of our learning – from physical skills, to language, to social ability and empathy – begins with observation. To be able to do these things well, we must have people around us who can successfully model these actions and behaviours. And not all of our clients do, particularly in terms of social skills.

A failure to be competent at any of these things could be due to a number of dysfunctions of the brain, including a dysfunction of the mirror neuron system itself. When manifesting as a failure to uptake and practise social skills, practitioners could consider whether the client could be somewhere on the ASD scale. Interestingly, this condition can also be accompanied by dyspraxia (Baranek, 2002; Williams, Whiten and Singh, 2004), which is a clumsiness that affects physical competence, sometimes in big, gross muscle skills, and/or in fine motor finger/hand muscle skills. Symptoms can include banging into things, difficulty tying shoe laces or holding a pen to write for any length of time. Dyspraxia could also be linked to a mirror neuron dysfunction; an inability to properly copy and master simple physical skills.

For other clients, a lack of opportunity to have appropriate social skills modelled for imitation may be the case. Dapretto *et al.* (2005) found that our mirror neuron function causes us to seek out those with similar behaviours and social skill sets to our own, which has the potential to compound this problem.

Pines (2003) has had success in lifting the abilities of people with low empathy and poor social skills by ensuring that they have regular and repeated time in close proximity to people who have good empathy and good social skills. Programmes such as the Canadian developed 'Roots of Empathy' programme (Gordon, 2005) are implemented at senior primary school level, and have been designed to teach young children empathy, making use of the mirror neurons system process as they practise observing a baby and drawing conclusions as to what the baby's actions and behaviours might mean it is feeling. These programmes also provide the opportunity to use the mirror neuron system to build new pathways through the children observing and imitating the adults (the parent(s) of the baby, and/or the early childhood educators) providing care and nurture to the baby.

Counsellors who encounter clients who lack empathy for others, or who present with issues that suggest poor social skills, may want to support those clients to seek opportunities to regularly observe and imitate those who have more competencies in these areas. Research into mirror neurons supports the value of regular social mentoring programmes. Like all successful learning, this observation and imitation would need to be practised often and regularly, and when the programme (or other form of opportunity) comes to an end, it would require a follow up series of sessions/opportunities at a later date in order for the learning around social skills and empathy to be retained through the building of permanent new neural pathways (Wolfe, 1998).

When counsellors find themselves working with clients who are unable to pick up on or imitate social skills, or who display low levels of empathy, they could consider a referral to mental health services for assessment for ASD, if these issues are causing difficulty in the life of the client. (There seems little point in putting clients who dislike or are anxious about social interaction through a system where they will be required to have further person to person interaction with new people, if they are happy as they are.) This needs to be weighed against the fact that those who have a brain dysfunction involving a faulty mirror neuron system can find it discouraging and disempowering to continually fail to learn these skills if they are unaware of the extra barriers that they face. Having said this, progress is sometimes still possible, as brain plasticity leaves the door open to the rewiring of new neural pathways.

People who know that they are affected by ASD, and who realize their barriers, can be supported by counsellors to understand that they will need to put extra effort into acquiring what comes naturally for others, and that they may need to use different strategies, such as intellectual analysis followed by imitation, rather than mirror neuron processes followed by imitation to achieve social skills.

A dysfunctional mirror neuron system, where the observer is unable to predict the intentions of the actor based on the actions they are performing, will affect a client's ability to feel safe in the world. This will affect both clients with a diagnosis of ASD and those who could potentially be diagnosed as such; some of them down the very mild end of the scale, and some not. Many socially awkward or anxious people, and also many eccentric people who do not live their lives conforming to social norms, could fit somewhere on the ASD scale, but will never be diagnosed, because of a lack of awareness about poor mirror neuron function, and because their behaviour, while in some cases creating personal challenges for themselves, is not sufficient to cause a problem for society.

When a counsellor encounters clients like this it is helpful to screen for mirror neuron dysfunction – do they exhibit varied facial expressions, are they able to predict the behaviour of others based on body language and/or facial expression or social context, do they understand potential reasons for the actions of others, do they demonstrate basic empathy and understanding of other's emotional states? Some, but not all people at the more extreme end of the ASD scale also exhibit a flat, toneless voice. They have learned the words that make up spoken language, but not its social subtleties.

If mirror neuron functioning appears low, counsellors can work with clients to find other strategies to develop intellectual frameworks for understanding the behaviour, intentions and emotional state of others, rather than relying on social understanding laid down in procedural memory. For instance, they can be shown to look for creases between the eyebrows that indicate another person's displeasure or disapproval, or to look for the direction of the other person's body to indicate whether they have interest in the current conversation. A higher tone of voice means a question rather than a statement. A loud spoken voice either indicates displeasure, or that the speaker has difficulty hearing.

This gives the client more understanding of social interaction without reference to the mirror neuron process, and with insight comes a greater ability to manage this social interaction, to check for social safety, and to feel safe. Counsellors can also work with clients to find safe social opportunities to practise these skills, which have been taught by the counsellor, rather than learned through spontaneous social imitation. It appears to be the part of the mirror neuron system that relates to social skills which is most likely to be affected by dysfunction (though, as previously mentioned, sometimes basic physical skills can also be clumsy). Brain plasticity suggests that when these skills are practised repetitively, and neural pathways developed for them, those on the ASD scale can learn these skills by a different route, even if mirror neurons fail to fire at initial observation of other's social behaviours.

One of the immediately relevant things for counsellors, associated with the mirror neuron system, is what this means in terms of our own empathy as counsellors. Many people attracted to the profession of counselling have naturally high empathy, fulfilling the ethical requirement to be non-judgemental of others, because the counsellor is able to place themselves in the client's shoes, and imagine how life is for them. People with high empathy are also more likely to notice and feel compassion for the emotional suffering of others, and to be attracted to a role in life where they can offer some relief to people who are struggling at any point in time (Breggin, Breggin and Bemak, 2002). The mirror neuron system of effective counsellors functions exceptionally well.

Many counsellors will be familiar with the occurrence of mirroring the feelings of the client. Sometimes the tears may come for the counsellor before they come for the client, or a feeling of anger or elation that does not fit the counsellor's own personal circumstances. There may be a knot of anxiety between the lower ribs of the chest, or a sense of hopeless greyness that can leave one feeling drained at the end of a session with a client suffering from severe depression. Counsellors need all the relaxation techniques that they share with clients (breathing, self-talk, muscle relaxation) and it is not for nothing that boxes of tissues are prominent in most counselling rooms, able to be reached by both client and counsellor! Being able to explain how mirror neurons work on a very simple level is a helpful tool for

self-recognition of our own emotional states in counselling and for explaining to clients why we too are crying.

For counsellors who are unaware of the likelihood of picking up on the emotional states of clients and who do not practise self-care, including reflective practice, debriefing through supervision, good food and sleep and regular time away from counselling/work to do other things and interact with a different set of people, burn out lies ahead (Maslach, 2003; Sprang, Clark and Whitt-Woosley, 2007).

On a more basic level, mirroring the posture and body language of clients has long been a recognized counselling technique. People who are taught it during training often find that they are doing it already, particularly when the counselling process is going well. (Mirroring of body language can easily be observed in other positive social interactions outside of counselling as well.) We tilt our bodies in the same direction as our client or companion, we cross our legs when they do, we lean forward towards them when they lean forward towards us, and we lean back when they do. These things send unspoken messages between people, such as 'I'm listening closely to you', 'I understand where you're coming from and I accept it', and 'I'm on your wavelength'. The messages that we send as counsellors enable our clients to feel more comfortable about sharing things with us that they don't share with others, and to see us as people who will work alongside them, seeing the world from their perspective.

Knowledge of the mirror neuron systems and how contagious behaviour, such as bullying, peer pressure, conformity, mob behaviour, mass/crowd hysteria, and so on, has its roots in these functions of the brain and is useful for working with any of these things in counselling. We may work with a bully, or the victim of a bully or group of bullies. We may work with people who have committed violent crimes alongside others, who are not sure what forces motivated them at the time, or are not aware of actually making a conscious choice to behave in that way. We may work with gang members, or people who are employed by corporates or organizations with particular cultures, who are behaving in ways that are counter to their own values or beliefs. An understanding of the mirror neuron system could contain the seeds of possible solutions to the behaviours of our clients, and/or to managing/responding to the actions and behaviours of others in their lives. It is likely to be appropriate to share some basic knowledge of

the workings of the mirror neuron system with many of our clients, so that they too can use this knowledge to make changes in their lives.

For instance, counsellors can use CBT techniques to teach people to think past their reflex actions and to scan for whether these are a match for their own morals and values, which could be helpful for those who find themselves surrounded by a behavioural culture that encourages them to behave otherwise, or who are in a situation where they are mimicking the behaviour of others to do something which is 'out of character' for themselves. Narrative or solution focused counselling could help clients look for exceptions to these behaviours in other areas of their lives. Part of the role of counselling may be exploring whether the client should remain in this situation, if the situation is a long term one, and if they choose not to, helping them to identify what they will replace it with.

Mirror neuron systems encourage us to seek out others who behave similarly to ourselves, and who have similar skills. Is this congruent with what the client wants for their life in the future, and if not, what actions will they need to take to do things differently? Counselling can help to identify social situations that clients can place themselves in which will take advantage of the function of the mirror neuron system to further their goals towards being the people that they want to be. (This is not necessarily connected to perceived social status, but rather to prosocial skills, world view, and/or to career opportunities or recreational opportunities for socializing, relaxation, fitness, etc.)

Mirror neuron systems are also relevant to addiction counsellors who work with clients trying to overcome addictions or avoid former addictions. For those who are in remission from addiction, knowing that watching others performing actions that have brought us pleasure in the past, including the handling of implements related to addictions (e.g. a beer glass, a bottle, a P pipe, a needle, a cigarette or lighter) makes our own neural pathways light up as well, gives us the insight that we are opening gateways to neural pathways that we may hope are becoming dormant through disuse, making us vulnerable to relapse.

The fact that mirror neurons can code for intention when an observer only sees part of an action, and that mirror neurons still fire when an action is heard but not seen, has implications for situations involving family violence. Parents and caregivers often assume that just because a child has not actually witnessed the violence, they will suffer no long

term effects or not be affected at all. What research shows about the function of mirror neuron systems in the brain tells us that hearing violence (and/or its symptoms and after effects) whether it be the sounds of arguing, screaming, shouting, banging, crashing, shattering or crying, will cause mirror neurons in a child's brain to fire.

This not only means that children will experience emotionally what is happening but are more likely to imitate the behaviours modelled, whether those of perpetrator or victim, in their own lives as children and adults. The role of a counsellor might be in working with parents or caregivers, and helping them to understand this, so that they can make more informed choices. On occasion, it might mean making a child protection agency notification to keep children safe from emotional harm, where previously a notification might not have been considered if the child(ren) did not directly witness family violence. Counsellors may also be working with affected children, and a knowledge of what is happening for them when they hear family violence or only witness the before or after effects, could help counsellors to provide more relevant therapy and support to these children and young people.

Encouraging parents to spend as much time as possible role modelling prosocial skills to their children should form the basis for any good parenting programme or therapy. Counselling may also help parents and caregivers unpack the parenting they experienced themselves as children, and how this affected their own social learning through their mirror neuron system bringing them to where they are today, and how they can access the potential for further learning through the mirror neuron system to learn new skills and empathy, which they can apply to their own parenting or caregiving.

Important learning for parents and caregivers is how vital it is to provide face to face personal contact for babies and young children, who are progressing through critical development periods for picking up social skills (including facial expression, interaction with others and emotional regulation) and language, because if they don't have the opportunity to observe and imitate these actions repeatedly, they will not use their mirror neurons to build neural pathways for these competencies. Children who are spoken to often in the home build wider and more comprehensive vocabularies, and this is predictive of both academic and social success later in life (Dickinson and Neuman, 2006).

When a key caregiver is depressed, possibly as a result of post natal depression/distress, they may be unable to motivate to provide this important interaction with their young baby or child, and as well as supporting the caregiver to receive appropriate care for themselves, counsellors should also talk with clients and/or their families about the need to have someone else who belongs in the child's life provide this function.

Because mirror neurons fire more weakly when actions are observed on a screen rather than in person, this function should not be left to TV, DVDs and/or videos by any parents or caregivers. These things may have their place as entertainment, but they do not replace the ability to learn physical, social, emotional and language skills directly from another person. Older children should be encouraged to spend time with real friends more often than they spend time with virtual friends on Facebook and similar social networking sites. Today, these sites are a part of life and need not be detrimental, as long as real time person to person contact is also practised. They may also strengthen existing connections with real friends. It is not up to counsellors to set these rules for families other than their own, and their role is to make this information available to parents so that they can make informed choices around their own parenting.

Emotion

What do we know?

Emotion is a very difficult thing to observe in the brain, and consequently, while there are many theories, actual data on how emotion is triggered and processed and what its role is, is sketchy. Some emotional processes are better understood than others. There is controversy over how many emotions there are, whether some are more basic than others, whether different emotional states have different neural patterns, how emotion might influence cognition and vice versa, and the importance of conscious versus unconscious cognition in emotion (LeDoux, 1996).

It is believed that the limbic system is a mediator of emotional process. The amygdala is the part of the limbic system most specifically concerned with emotion (Kandel *et al.*, 1995). In humans, electrical stimulation of the amygdala results in feelings of fear and apprehension. Fear, or anxiety, is probably the best understood of all human emotions at this point in time. Because, for survival purposes, fear requires the most rapid response (known as the 'fight or flight' response, and sometimes the 'fight, flight or freeze' response), fear is processed most rapidly of all the emotional states (Kandel *et al.*, 1995; LeDoux, 1996).

The amygdala is composed of many nuclei that are reciprocally connected to the hypothalamus, the hippocampal region, the neocortex and the thalamus. The basolateral nucleus in the amygdala assigns emotion to incoming stimuli (Shekhar *et al.*, 2005). Stimulation of the hypothalamus produces physical reactions that are usually the product of strong emotion, such as alterations in heart rate and breathing, bladder contraction and the fine hairs on the skin standing on end (Kandel *et al.*, 1995).

Pleasant or unpleasant stimuli have dual effects. First, they cause the amygdala to trigger autonomic, (unconscious self-regulating) and endocrine (endocrine glandular system, which releases and regulates hormones) responses. These responses are integrated by the hypothalamus, altering the internal state and preparing the physical body for attack, flight and retreat, sexual experience or other adaptive behaviours. When we interact with the environment, a second set of mechanisms is activated in the orbitofrontal cortex. This allows the perception of the emotion and modulates the behavioural response to that emotion according to cognition, which takes into account prior learning and current context (Kandel *et al.*, 1995).

In addition, the brain stem has collections of neurochemically specialized neurons with widely branching axons throughout the forebrain. The chemicals that they release diffuse through neuro tissue, and bathe the neural populations in the orbitofrontal cortex and the striatum. These chemicals modify synaptic efficacies of transmission and are known as neuro modulators. The nucleus of each neuron makes its own neuro modulator, which interacts in complex ways with other neuro modulators. Neuro modulators include: acetylcholine (associated with memory), dopamine (associated with pleasure), endorphins

(associated with relief of pain), histamine (associated with arousal), melatonin (associated with sleepiness or wakefulness), norepinephrine (associated with fear and apprehension), serotonin (associated with relaxation and calm feelings), oxytocin (associated with sexual orgasm and imprinting/bonding) and vasopressin (associated with aggression) (Freeman, 1995).

Neuro modulators influence awareness, motivation, mood and affect and disposition. The strength of conviction with which people hold a belief is more closely related to the concentrations of neuro modulators by which that conviction is mediated than to the degree of truth involved (Freeman, 1995).

When emotions are habitually paired with certain stimuli (neurons that fire together wire together) a conditioning process takes place, and specific neural pathways are formed to connect the two (Kandel *et al.*, 1995). In the absence of a conditioned response, the hippocampus and the prefrontal cortex provide a self-regulating mechanism for emotion, based on context and appropriate response (LeDoux, 1996; Schore, 2000).

What does this mean for counselling?

The perception and processing of external stimuli gives rise to emotional state in preparation for our behavioural response. Most people are able to regulate this emotional response with the feedback from the hippocampus formation in the orbitofrontal cortex. When this feedback is missing because of either damage, dysfunction or underdevelopment of the cortex, emotion is much more difficult to consciously self-regulate. This is natural in children, particularly in the early stages of development, but it also persists in some adults.

As well as a lack of ability to control emotion in general, some clients are unable to integrate prior learning (from previous experience) or context to help regulate emotional response. Fear and anger are the most common emotions that clients struggle to control at the moment of impulse, and this may be because of the swiftness of their processing, given their role in the fight or flight mode survival paradigm.

The section on mindfulness in this book (included alongside deep breathing and meditation) describes a strategy where clients are

encouraged to focus on their emotional state without judging it, and to notice how it manifests itself in both brain and body. Being able to notice the autonomic physical changes (i.e. hot cheeks, clenched jaw, sweaty palms, rapid breath) may give clients momentary advance warning of emotions that they find difficult to self-regulate. Focusing inwardly on the emotion, rather than moving forward to the behavioural response, can provide a circuit breaker in the established neural pathway of stimulus, emotion and automatic unwanted response, such as emotional meltdown, inappropriate aggression or flight from situations regarded as safe and manageable by the majority.

Clients who demonstrate other symptoms of prefrontal cortex damage, dysfunction or underdevelopment – difficulty with attention and memory, lack of impulse control, lack of planning capacity, difficulty with problem solving – may not have the same ability to regulate their emotions as other adults and find the task frustrating and bewildering, especially as they observe others as not experiencing the same difficulty in this task.

The limbic system has synaptic plasticity if the conditions for this are optimal, and as such, established neural pathways for response to emotion are able to be synaptically modified. This is demonstrated by the knowledge that we have that emotional responses can be paired with stimuli, and responses conditioned. As is described in the sections on brain plasticity and neurons that fire together wire together in Chapter 2, this means that we all have the potential to change our neural wiring.

Many emotional response pathways and the pathways for stimuli that they are connectively wired to will have been set down early in childhood, and could be expected to be very well trodden pathways by the time a client enters counselling, and as such, resistant to change. It is unlikely that such pathways will ever be completely extinguished, but as always, determined and repeated practice can form new ones, which get stronger with concentrated usage.

If brain plasticity is functional, then even the clients with barriers to an optimally functioning prefrontal cortex and executive function have the chance to strengthen these weak neuronal pathways. Unfortunately, lack of executive function can become a vicious cycle, as the skill of attention (in order to complete both the focus and practice tasks needed for new learning) is key to building the new pathways. The counsellor needs to consider the capabilities of the client before

suggesting that they attempt this task, because clients do not come away feeling good when they attempt change and fail.

For those who have become conditioned to pairing emotional states with certain stimuli, or who have wired neural pathways between a particular emotion and a particular response (or who have even connected a fixed stimuli-emotion-response pathway or pathways), the counsellor's first role is in helping the client to gain insight. When did these pathways first become connected? What other emotions or responses might be possible? Have they observed these alternate pathways being displayed by others in response to the same stimuli or emotion? Who in their early life might have modelled emotions or responses to their emotions similar to their own? How are those emotions and responses serving them? Would they prefer to experience that stimuli in an emotionally different way, or respond to that emotion with a different behaviour or set of cognitions?

New learning through insight subtly changes the brain, as does all learning (see the section on learning and attention in Chapter 3). New neural pathways are already beginning to form. Repeating and mentally going over insight arising from the counselling session is the beginning step to practising the new neural pathway. Then the counsellor and client need to work together to practise the client's desired goals for new neural pathways, if any. CBT would seem to have a natural role here, with its focus on the thinking, feeling and behavioural triangle, and the potential for changing one (i.e. emotional state) by changing another (i.e. cognition or behaviour) and getting the client to practise this during sessions. CBT could be summarized as the practice of forming new neural pathways, and is particularly suitable for making changes to emotional state, and the cognitive or behavioural response to emotional state.

Attachment

What do we know?

The study of attachment was introduced by Bowlby (Bowlby, 1969; Bowlby, 1973a), and has been popular with both counsellors

and psychologists ever since. But while the behaviour is externally observable, the neuroscience of how the process might work internally in the brain is still largely a mystery. Bowlby (1969), posited the theory that attachment mapped onto our internal maps of the self (see the section on 'A map of ourselves' in Chapter 3).

Most neurological studies of attachment to date have been conducted on animals rather than humans, so caution needs to be exercised in transposing those results. These studies do however, offer probabilities for models of human attachment (Insel and Young, 2001).

Three key phases of attachment have been identified. The first is an increased arousal towards or avoidance of the subject of potential attachment. The second is the organization of long term memory to consolidate the attachment bond. The third stage is when the attachee begins to avoid new potential subjects for attachment, while consistently approaching and/or following the imprinted subject of attachment (Insel and Young, 2001).

The study of attachment in infant mammals is yet to identify a specific neural circuit or neurochemical system for this process, but it is believed that the neuropeptide oxytocin is involved (Insel and Young, 2001). (Peptides cause excitement or inhibition when applied to target neurons. Some peptides, such as oxytocin, have hormonal function.) Oxytocin is implicated in an imprinting process in rat pups involving the olfactory system (sense of smell). Inhibition of oxytocin inhibits social bonding in the rat pups (Insel and Young, 2001). While no neural network for bonding and attachment has been identified in juvenile rats, specific neural pathways for attachment have been located in chicks, involving early and persistent release of amino acids, and changes in post-synaptic structure within specific cortical regions. There are, however, lesion studies that show that when there is damage to the medial preoptic area or to the bed nucleus of the stria terminalis, social bonding in rats is inhibited, which suggests that these parts of the brain may be part on a neural attachment network (Insel and Young, 2001).

Oxytocin is important in establishing maternal behaviour, but is not required once maternal behaviour is established in mammals. Maternal behaviour requires the suppression of the instinct to avoid the odours of newborns, which would normally repel us. Oxytocin release decreases olfactory processing in the brain, facilitating approach

behaviour (Insel and Young, 2001). Vaginocervical stimulation, which occurs during the process of giving birth, is a potent stimulant for oxytocin. Recordings of the olfactory bulb in ewes show that there is a 60 per cent increase in the number of cells that respond preferentially to lamb odours in the first weeks after the ewe has given birth, and 30 per cent of those cells respond only to the smell of their own lamb. Within twenty-four hours of birth, the smell of a ewe's own lamb results in a significant increase of extracellular concentrates of the neurotransmitters glutamate and GABA (y-aminobutyric acid). This process has also been observed in mice (Insel and Young, 2001).

Oxytocin is released during forgetting or 'unlearning' (Freeman, 1995). Because more oxytocin is released when sheep have their second lamb, it may be that oxytocin helps to clear a blank slate for bonding (Insel and Young, 2001).

Sexual activity releases oxytocin in prairie voles, and it is thought to be important in the process of pair bonding, or relationship attachment, in adults as well and infant/caregiver attachment (Insel and Young, 2001). In humans, sexual ejaculation releases oxytocin, and the level of release is higher in women. The partner who leaves straight after sex and does not spend waking time with their sexual partner afterwards will not bond/imprint to the other person in the same way (Freeman, 1995). Another neuro peptide, vasopressin, when combined with oxytocin, seems sufficient condition for pair bonding in prairie voles, mice and primates. It is not known whether oxytocin and/or vasopressin increase preference for a partner, or facilitate recall of the subject as the preferred partner (Insel and Young, 2001). Vasopressin is linked to aggression against potential sexual rivals (Freeman, 1995).

In human brains, oxytocin receptors are concentrated in dopamine rich regions, which are connected to the neural pathways for reward. The process of attachment probably involves the same neural pathways required for other forms of motivational behaviour. Dopamine pathways in the forebrain, especially in the nucleus accumbens and in the ventral pallidum, are implicated in partner preference formation. For attachment to occur, oxytocin and vasopressin must link social stimuli to dopamine pathways associated with reinforcement (Insel and Young, 2001).

fMRI studies of humans show that adults looking at pictures of their partners activated similar parts of their brains to the parts of the brain activated when mothers hear their newborn crying (the anterior cingulate, the medial insula, the caudate and the putamen). The pattern was distinct from other brain activity patterns, such as facial recognition and sexual arousal.

In humans, neuro peptides, such as oxytocin and vasopressin, which are modulators of fast neurotransmitters, have discrete central neuro system distributions and are regulated by highly plastic receptors, seem especially likely candidates for mediating attachment.

A more structural model of how attachment builds the brain is put forward by Schore (2000). This model is supported by electroencephalography (EEG) and other neuroimaging data suggesting that the positive emotional exchange in a parent/child bond involves the right hemispheric cortical and subcortical systems that participate in emotional modulation. The right hemisphere interprets non-verbal social cues, such as facial expressions, body language, and gestures, as well as vocal tone. The orbitofrontal cortex has been shown to mediate the highest level of behavioural control, especially in regard to emotion and impulse (Schore, 2000). Early experiences, including attachment experiences, shape the developing brain and influence the formation of neural pathways (Freeman, 1995). These pathways may be stored in implicit/procedural memory systems in the right cortex.

Representations of expectation regarding our own social capacities and the response of others to our social behaviour are processed by the orbitofrontal cortex, which is known to be activated by 'breaches of expectation'. If the system is working optimally, information received from the external environment can be integrated with internal data, such as emotional and physiological state. Sensory data are relayed into the limbic system triggering adjustments in emotional and motivational states, and in this manner the orbitofrontal cortex integrates the two (Schore, 2000).

The right hemisphere mediates empathetic cognition and the ability to perceive the emotional states of others. The orbitofrontal cortex is essential to empathy and Schore (2000) believes that this may be built on neural pathways laid down for attachment. Because the orbitofrontal cortex is also essential in the self-regulation of emotion, Schore (2000) also theorizes that healthy attachment may

be a necessary condition for this self-regulation to take place. Schore (2000) links social interaction experienced during early attachment experience to the ability to self-regulate emotion and arousal, as does Kandel (1999), who believes that early stressful memories lead to brain connections and pathways that are stored in basic procedural memory.

Right orbitofrontal cortex damage affects the capacity for attachment, empathy, emotional self-regulation and appropriate social response to stress. The major coping systems for external stress, the hypothalamus–pituitary–adrenal cortical axis (HPA axis) and the sympathetic adreno medullary axis are under the main control of the right cerebral cortex (Schore, 2000). An indicator of secure attachment is resilience in the face of stress. (See also the sections on stress in Chapter 4 and personality disorders and Post Traumatic Stress Disorder (PTSD) in Chapter 5.) Early insecure attachments may inhibit the growth of this control system in the right hemisphere. Schore (2000) suggests that this initial insecure attachment (where the attachee cannot depend on the subject of their attachment to meet their physical and emotional needs) can be linked to a diverse group of psychiatric disorders and the neuro physiological changes that accompany them. He believes that we may soon be able to map relationships between differences in early attachment experiences and changes in neurochemistry and brain organization.

What does this mean for counselling?

I have concentrated on the neuroscience of attachment here because of the amount of attention it receives from practitioners of counselling and other verbally interactive therapies. In my experience, clients have often had it suggested to them that somewhere in their story, or that of their children, there is 'an attachment disorder'. It pays for the counsellor who wishes to retain credibility to be able to discuss this intelligently with the client!

There is not much that human beings can do to modulate their own hormones, other than the avoidance of sex (at least with specific partners). If the neuropeptide oxytocin bonds us to a partner, and is released during sex, it follows that if our goal is to end a relationship,

particularly if it is an unhealthy relationship, we should avoid having sex with that person.

Because oxytocin is involved with unlearning and is strongly released (especially in women) during sexual activity, which generally is at a high level at the commencement of a new relationship, this evolutionary strategy for imprinting is probably also responsible for the plasticity that allows us to mould our old selves to fit better together with our new partner. We may start liking a sport we didn't bother with before we met them, or staying home on Saturday nights because they do. Unfortunately, this highly plastic state also makes us vulnerable. Because all those touch stones of self that were so firm prior to the start of the relationship are now malleable, including standards and moral values, when a new partner is abusive, many people adjust to being the recipient of that behaviour in a way that they wouldn't have if a stranger had perpetuated it. (And neural pathways, linking sex with the new partner and rewarding feelings, are also bedding in, giving strength to the newly formed attachment bond.) Knowing this produces interesting insight for clients when they are blaming themselves for allowing the abuse in the first place. Often people have an incredulous sense of 'How did that happen to me? I used to be so strong!'. Understanding the underlying biological and neural processes helps to shift the focus away from a sense of failure.

The role of smell in relationship building and in parent–child attachment is interesting. Step-parents struggling to treat stepchildren with the same flexibility and tolerance as they treat their own children sometimes report that their partner's children 'smell different'. When someone loses a loved one, either because they have died or because of relationship break up, they often carry something that retains the smell of that person, such as an unwashed favourite shirt. Counsellors can discuss with clients the potentials regarding the retention or otherwise of these items, depending on the situation and how the client views it. For instance, a mother may wish to carry a missing son's shirt with her, whereas a partner who has ended a relationship with a cheating spouse may (or may not depending on whether they hope to resume the relationship or not, and who left who!) wish to buy a new set of sheets and other bedclothes.

The issue with attachment to stepchildren is a little more complex. It could be useful if a step-parent is committed to trying to form a

bond with a stepchild for them to spend regular time in that child's company, so that their smell becomes familiar, or for them to regularly sniff an item imprinted with that child's smell, which sounds a bit creepy but need not be if the sniffer's intention is to facilitate bonding. Sometimes, it may just be a matter of the step-parent coming to terms in counselling with the fact that the instinctual bond is not there with a child who is not their own, and helping them to plan and practise new neural pathways based on cognitive choices for fair and competent parenting of that child.

Because the brain is plastic an insecure, or non existent, pattern of early attachment need not be the end of the world. If a person can go on to practise healthy attachments with new caregivers (or it becomes possible to recreate a healthy attachment with the original caregiver) or even in adult relationships, they can begin to build whichever neural pathways are required for attachment, and eventually these will become strong healthy pathways.

It is important to remember that the best and most effortless time for attachment to be laid down is during the critical period for attachment learning, when these areas of the brain are at their most plastic. But this does not preclude the brain's ability to build and strengthen new neural pathways at any time if the conditions for synaptic plasticity, such as the availability of brain derived neurotrophic factor (BDNF) in the brain, are met. A counsellor's role would be to encourage the client to focus on the attachment relationship goal, to practise skills of attachment, and to maintain this practice (see also the section on learning and attention in Chapter 3) and to help the client monitor their efforts to see what outcomes are being achieved.

For some people who experience inappropriate response to stress, a lack of emotional regulation and low empathy to the emotional states and feelings of others, practising secure attachment and developing neural pathways for this could be helpful in improving these conditions, although it is important to note that poor function of the right prefrontal cortex (which would produce these symptoms among others) could be due to factors other than insecure attachment, or the absence of attachment.

Addiction

What do we know?

The process of addiction shares striking similarities with the neural plasticity associated with learning and memory (Kauer and Malenka, 2007; Kelley, 2004; Nestler, 2005; Robinson and Kolb, 2004), and it is thought that these systems overlap with neurochemical systems of motivation and reward (Hyman, Malenka and Nestler, 2006; Kalivas and Volkow, 2005; Kauer and Malenka, 2007; Kelley, 2004; Nestler, 2005). Drugs of abuse have different mechanisms of action and are highly diverse chemical structures, but they converge on the brain to produce similar effects of reward, even though they may produce different behaviours and physiological effects (Nestler, 2005).

The exact process of addiction has been difficult to identify, particularly the downstream chain of effects (Hyman *et al.*, 2006; Russo *et al.*, 2008), but it is known that two neurotransmitter systems play a large role: the dopaminergic and glutamatergic systems. These neurotransmitter systems are widely distributed throughout the frontal cortex, the limbic stem and basal ganglia regions, and are known to be implicated in learning, memory and motivation. They are involved in reward related learning, the outcome of which can be addiction (Everitt and Robbins, 2005; Kalivas and O'Brien, 2008; Kalivas and Volkow, 2005; Kelley, 2004; Nestler, 2005). Dopaminergic and glutamatergic systems are critical for adaptive changes in gene expression and synaptic plasticity, which means the reconfiguring of neural networks, and therefore, behaviour. The main function of glutamate is to encode sensory, motor and memory information, and dopamine provides the sensation of reward. In conjunction with each other, they play an essential role in shaping synaptic configuration (Hyman *et al.*, 2006; Kelley, 2004).

A key circuit involves dopamine cells in the ventral tegmental area of the midbrain, which release dopamine into the limbic forebrain: the prefrontal cortex, the amygdala and especially the nucleus accumbens (Hyman *et al.*, 2006; Kalivas and O'Brien, 2008; Nestler, 2005; Russo *et al.*, 2008). Every drug that carries emotional reward converges on the ventral tegmental area/nucleus accumbens pathway (Nestler, 2005). Glutamate plays a role first in integrating information from the declarative memory regarding pleasurable experience associated

with the drug, and then, as the addictive habit forms, it integrates information from the procedural memory where automatic learning is stored. In this process, glutamate projects from the prefrontal cortex to the nucleus accumbens, which is thought to be important for the prioritization of motivational importance and deciding goal directed behaviour (Kalivas and O'Brien, 2008; Kalivas and Volkow, 2005). During the process of addiction, the brain produces more glutamate than usual because of reduced inhibitory presynaptic regulation, and greater transferability of glutamate between neurons. Protein signalling in the prefrontal cortex also increases the excitability of the neurons projecting to the nucleus accumbens (Kalivas and Volkow, 2005). Dopamine and glutamate participate not only in the addiction process for stimulants, but in also that for nicotine and alcohol (Kelley, 2004).

The process of addiction comes in three stages (Kalivas and O'Brien, 2008), the first being social use or the development of addiction, the second being regulated relapse where a person has repeatedly used the drug and consciously chooses to use it over other biological rewards at socially appropriate times (such as at a party when the children are with a babysitter), and the third stage being compulsive relapse, where natural motivation towards biological reward (such as for eating, or avoidance of danger) or social reward (such as caring for children) is completely subsumed by desire for the drug, which replaces natural reward with artificial chemical reward from the neurotransmitters (Hyman *et al.*, 2006; Kalivas and O'Brien, 2008; Kauer and Malenka, 2007; Kelley, 2004; Russo *et al.*, 2008). Other neurons become less sensitive to those neurons which are activated by natural rewards (Nestler, 2005). Regulated relapse is associated with declarative memory, based on recall of other drug experiences in conjunction with factual knowledge in order to make the best decision. Compulsive relapse and long term addiction is associated with procedural memory and automatic behaviour (Kalivas and O'Brien, 2008). This probably applies to all types of addiction, and not just to addictions involving drugs of abuse.

Natural reward systems are not always active and are only triggered by biological need, such as hunger or danger. When hunger is sated it brings contentment and sometimes pleasure. When danger is avoided, it brings relief. Addictive drugs allow the person taking them to receive

a reward sensation without a biological trigger, and to experience the pleasurable sensation of reward at will (Kelley, 2004).

Neuroimaging supports a link between the prefrontal cortex and drug seeking. The intensity of change in metabolic activity in the orbitofrontal cortex and in the anterior cingulate cortex correlates with the intensity of self-reported craving (Kalivas and Volkow, 2005). The dysregulation in these two cortices contributes to the overwhelming desire to seek and ingest drugs. The amygdala, hippocampus, hypothalamus, and several regions of the frontal cortex interact with the ventral tegmental area and the nucleus accumbens, and are part of the system affected by the chronic changes associated with addiction. This is known as cortical hypofrontality, and brain imaging studies show reduced base line activity in the prefrontal cortex, the anterior cingulate cortex and the orbitofrontal cortex. It is thought that this has implications for emotional memory, which could perpetrate drug use, and also implications for memory loss, attention deficit, impulsivity, reduced decision making, and other prefrontal cortex dysfunction (Hyman *et al.*, 2006; Kalivas and Volkow, 2005; Nestler, 2005). The imaging studies also demonstrate complex changes in the glutamatergic outputs of these regions, which are implicated in impulsivity and compulsiveness, both features of addiction (Nestler, 2005).

Long term exposure to some drugs of abuse, such as cocaine, nicotine, amphetamine and morphine, alters the form and structure of the dendrites and their spines (Kalivas and Volkow, 2005; Robinson and Kolb, 2004; Russo *et al.*, 2008). Dendrites are the receptor mechanisms of neurons. The two major sites for synaptic input are the spine and the shaft of the dendrite (Kandel *et al.*, 1995). The initial studies of Robinson and Kolb (2004) suggest that long term drug use permanently limits the ability of the brain to reorganize neural pathways, but the authors say that it is also possible that other factors could mitigate this effect, and it requires further study. The change to the spine density and form is thought to be a result of alterations in post synaptic proteins (Kalivas and Volkow, 2005).

Several drugs of abuse, after long term use, reduce neurogenesis or the birth of new neurons in the dentate gyrus of the adult hippocampus, including cocaine, opiates, alcohol, nicotine and cannibinoids. This could also affect memory function (Nestler, 2005). Another change in

the brains of addicts is the lower base line activity of cortical pyramid neurons (Nestler, 2005).

Dopamine transmission usually signals novel events as part of its role in the process of learning (Kalivas and Volkow, 2005), and this may explain why drug seekers find it hard to recapture that first initial high. All addictive drugs produce similar effects of negative emotion upon withdrawal (Nestler, 2005). After chronic drug taking, natural dopamine levels in the brain drop in an effort to readjust to normal levels in the presence of artificially induced dopamine (Kalivas and Volkow, 2005; Nestler, 2005). The abnormally low levels of dopamine then reduce pleasure in life, and there is far less reward for biological achievement, such as eating. The lower levels of dopamine also make it more difficult for the drug to create the same level of pleasure as it did with initial use, and a tolerance to the drug is created, so that higher and higher doses become less and less effective for the user (Nestler, 2005).

Chronic drug use also sensitizes the dopamine system to the drug of choice. This sensitivity lasts long after drug taking ceases and probably relates to drug craving and relapse (Everitt and Robbins, 2005; Hyman *et al.*, 2006; Nestler, 2005). Sensitization can last for weeks, months or years, and increases with increased dosage and length of use (Kauer and Malenka, 2007). Some drugs of abuse have been shown to induce cross-sensitization to each other (Nestler, 2005).

Chronic drug taking is also associated with changes to the central corticotropin releasing factor systems (CRF systems). Cold turkey withdrawal leads to activation of CRF containing neurons in the amygdala, reflecting underlying adaptations in these neurons, most likely induced by the cAMP response element binding (CREB) protein, which is induced in the amygdala by stimulants. The CRF gene is regulated by CREB. These neurons are linked to fear and other unpleasant emotional states, and are partly responsible for the negative emotions and unpleasant physical symptoms that occur during drug withdrawal. The urge to avoid them and return to the process of dopaminergic reward is a major factor in the craving/relapse process (Nestler, 2005). CREB is also required for long term behavioural memory, which plays a role in the habitual nature of addictions (Hyman *et al.*, 2006).

Stress (see also the section on stress in Chapter 4) is known to make neural pathways for drug craving vulnerable to reactivation

(Kauer and Malenka, 2007). Exposure to drug cues (viewing others taking drugs, drug paraphernalia, being in situations or environments where drugs have formerly been available) reactivates drug craving mechanisms in the brain (Hyman *et al.*, 2006; Kauer and Malenka, 2007; Kelley, 2004). Cue-primed drug seeking and stress-induced drug seeking engage different regions of the amygdala (Kalivas and Volkow, 2005). Prefrontal accumbens drive (as described above) and neuronal pathway adaptation are thought to give motivational salience to the availability of the drug cues (Kalivas and Volkow, 2005).

Addiction can also be developed around stimuli which would normally provide biological reward in response to need and/or natural activity that promotes biological drives, such as food, exercise or achievement. The ventral tegmental area/nucleus accumbens pathway also mediates positive emotions for natural rewards, such as food, sex and positive social feedback, and similar abnormalities are found in the brain scans of those with drug addictions and those with addictions to overeating, sex, gambling, exercise and so on (Nestler, 2005).

Some studies point towards a role for BDNF in addiction (Kalivas and O'Brien, 2008; Kauer and Malenka, 2007; Russo *et al.*, 2008). Growth factors like BDNF may mediate linkage between rapid changes in synapses caused by drug exposure with longer lasting modifications of circuit activity. BDNF injected directly into the ventral tegmental area enhances drug-seeking behaviour in animals that have addictions, even after several weeks of withdrawal period. BDNF levels increase in the ventral tegmental area during prolonged drug withdrawal (10–15 days) (Kauer and Malenka, 2007; Russo *et al.*, 2008). BDNF levels in regions of the brain associated with reward can be regulated by opiates (Russo *et al.*, 2008).

It is also probable that long term potentiation (LTP) and long term depression (LTD) which are causal to synaptic change and synaptic permanence respectively, play a role in the process of addiction by altering gene and protein expression through their ability to alter synaptic connections (Hyman *et al.*, 2006; Kauer and Malenka, 2007). It is thought that drug exposure triggers LTP, which is then followed by LTD once new connections are formed (Kauer and Malenka, 2007). Repeated drug use reduces the ability of the brain to respond plastically to other life experiences (Robinson and Kolb, 2004).

Because the neural pathways created during addiction and the physiological changes addiction produces in the brain seem very persistent, Hyman *et al.* (2006) consider that no treatment episode, even when effective, can be considered curative.

What does this mean for counselling?

A distinction needs to be drawn between full blown drug addiction and drug dependence. Full blown addiction is used to signify the compulsive relapse state, where the addict craves and compulsively relapses into drug taking. Drug dependence, where the client uses drugs to block reality (or even purely for pleasurable sensation) but is not yet compulsively taking them at the expense of natural biological rewards, such as eating or caring for children, is more likely to indicate the regulated relapse stage of addiction. There is no firm clear line between the two states, just as there is no firm clear line between social use and regulated relapse. Clients may report that they take drugs to avoid unpleasant realities in their day to day lives, but could also have moved into a state where they are now also motivated to use the drug to satisfy the chemical needs of the reward pathways of the brain for dopamine on demand.

Where clients are in the stages of social use, or regulated relapse, and the use of the drug is still a conscious choice, counsellors should continue to help clients unpack the historical and current reasons for the drug taking, and what the clients can change in their lives if they wish to make changes to their drug use. It is important for counsellors to make information available to these clients regarding how their ability to choose will recede if they move into the compulsive relapse stage of addiction.

Ironically, it may be those clients with more brain plasticity than others who are most easily addicted. In general, brain plasticity would be an advantage, leaving the brain open to new learning and quick adaptation to the environment. But those with greater brain plasticity may adapt more easily and quickly to accommodate the drug, making new neural changes at a faster speed. In a further irony, once drug use has reached the compulsive relapse stage, the changes the chronic drug use bring to the brain reduces the brain's ability to utilize

plasticity and make new neural connections, leaving the client stuck in the state of brain rigidity, unable to further learn or adapt because of the biological changes within the brain.

Counsellors need to be aware that clients who have used drugs of abuse heavily, or for an extended period, are likely to suffer symptoms similar to those usually associated with ADHD (see also that section in Chapter 5) where the executive function of the brain is greatly reduced, leading to poor or non existent attention and concentration skills, poor impulse control, lack of ability to plan or make decisions based on how those decisions will affect what happens in the future, poor emotional regulation, and memory loss.

There will also be a lack of motivation towards prioritizing social reward, and a client in the stage of compulsive relapse will be able to do little more than give lip service to the goals of making their parents proud, caring for their children, not disappointing their families, and not causing further pain to those who they love. Counselling is about supporting people to make positive change, and for these clients a long period of supported withdrawal in a residential centre is their best chance at getting past the negative effects of ceasing to use the drug and into a stage where they can begin to build new brain pathways to replace the persistent pathways created by the drug. Patience is needed in supporting these clients, as they will have suffered lasting brain changes that make new learning more difficult, and which also make it more difficult to achieve their goals.

Counsellors must recognize full blown addiction as an invisible disability, and that those that suffer from this disability do not have the same use of an optimal rational brain as someone who is not affected. The neural pathways subverted by the glutamate/dopamine learning and reward system, now encoded in their memories, will have skewed their motivational prioritization and make it difficult for them to resist cravings and to begin or continue the withdrawal process. Personal responsibility for our own choices is important, but it is also important for counsellors to take a client's capacity to make rational choices on their own behalf into account, given the powerful chemical cycle taking place in their brains (which are seeking to replace lost dopamine) and the reduced plasticity for relearning to do things differently.

Several things will provide opportunities to sabotage an addict's attempt to become clean of drugs. Connecting with others who are associated with the drug, being in environments where drugs have formerly been available or seeing drug paraphernalia can all retrigger the neural pathways for obtaining and using the drug. Part of the role of counsellors who are supporting drug users is to help them plan to move their lives in a direction where these occurrences will at the very least be minimized.

To give new neural pathways a headstart, counsellors and clients can use role play to open, or reopen, more appropriate brain connections. Clients can plan, discuss or practise what they will do or say in difficult situations, such as when they are offered drugs, or when an old friend who has used drugs with them in the past comes to visit. They can plan how to interrupt familiar neural circuits, perhaps by ringing a friend, or a counsellor on duty/phone counselling service, or by going for a walk or having a shower. What works best for any given client will be a matter of individual preference.

For some specific addictions there may be medication interventions that can lessen the desire for the drug, and counsellors can refer affected clients to those who can prescribe these interventions. Much research is being done to find medications that can break the neurochemical cycle of addiction (Kalivas and O'Brien, 2008; Nestler, 2005).

Because of the strength of the neural pathways created by addiction, it is probably true that once addicted, having gone through the period of withdrawal, it only takes one instance of using the drug to reopen the pathways related to that addiction. Programmes such as the Alcoholics Anonymous Twelve Step Programme (Wilson and Smith, 1939) are already based on this understanding. Counselling models such as harm minimization are more appropriate for those in the earlier stages of social drug use or the regulated relapse/drug dependant stage where usage is still a choice, rather than for those who have become full blown addicts through chronic use.

In the main, counsellors are already on the right track in working with clients who are troubled by drug use, and the new learning for some will come with the confirmation that the brains of addicts work differently and are processing differently to the brains of non addicts, and that techniques such as motivational interviewing may not work for clients dealing with chronic drug usage.

Counsellors may also work with clients who are addicted to an over saturation of natural rewards, such as compulsive eaters, fitness fanatics, etc. These clients should also be acknowledged as having brains where the normal reward pathways have been subverted, and be supported as having an invisible disability where the brain needs to be retrained into new neural pathways if wellness is to be obtained. Clients, whether drug users or addicts of natural biological reward, are not weak when they give into cravings. Instead, they are incredibly brave when they make efforts to overcome their addictions. For this reason, it may take several attempts before they are able to do so successfully, and this should be acknowledged at the outset by both counsellor and client. Counsellors should continue to work with clients for as long as they wish to make these attempts, and realize that the process of building new neural pathways to replace those of addiction will take as long as it takes.

It is probable that only addicts and their families and those others who love them truly understand how gut wrenching addiction can be. Thankfully, brain plasticity, although reduced with chronic addiction, still gives hope that change is possible, and that the brain can relearn and make positive changes.

Stress

What do we know?

The hippocampus (the seat of learning and memory) is vulnerable to degenerative changes caused by chronic stress. Chronic stress also affects neurons in the amygdala, probably leading to behaviours of enhanced emotional reaction. Stress induced structural plasticity in the neurons of the amygdala is likely to relate to affective mood disorders triggered by chronic stress (Vyas *et al.*, 2002). In the hippocampus, chronic stress is characterized by a reversible shortening and de-branching of neuronal dendrites (Smith *et al.*, 1994; Vyas *et al.*, 2002).

The hippocampus gives negative feedback regulation of the stress response via the HPA axis. This axis links the hypothalamus and the pituitary glands in the brain with the adrenal glands in the body,

which are located above the kidneys. Stronger hippocampal input suppresses the HPA axis, but enhanced input from the amygdala has the opposite effect. Chronic stress causes dendritic atrophy in the hippocampal pyramid neurons, and causes extra growth in some types of the basilateral amygdala neurons, creating a state where the HPA axis is working overtime (Smith *et al.*, 1994; Vyas *et al.*, 2002).

Predictable chronic stress produces a greater amount of neuronal atrophy in the hippocampus than chronic unpredictable stress. The type of neurons in the amygdala which are increased in size during predictable and constant stress are unaffected when the chronic stress is unpredictable. The other type of amygdaloid neurons is affected in exactly the opposite way, being more responsive to unpredictable stress (Vyas *et al.*, 2002). Stress also hastens the loss of hippocampal neurons that occurs as part of the ageing process (Smith *et al.*, 1994).

Recent studies of amygdaloid long term potentiation (LTP, see the section on memory in Chapter 3) show key differences in mechanisms of synaptic plasticity between the hippocampus and the amygdala. Differences in synaptic plasticity can alter the cellular response to the same stressful stimulus. Chronic stress could lead to an imbalance in the HPA axis through a loss of hippocampal inhibitory control, as well as a gain in the excitatory control exerted by the amygdala. Stress disorders are often characterized by a lack of cognitive function (an underperforming hippocampus) and an abnormally high fear response (an overactive amygdala). Stress may impair memory through a lack of hippocampal function (Smith *et al.*, 1994; Vyas *et al.*, 2002).

Chronic stress reduces BDNF in the dentate gyrus, which is the part of the adult human brain where new brain cells are formed. Conversely, small amounts of non-acute stress increased levels of the neurotrophic factor known as Neurotrophin-3 (NT-3). BDNF and NT-3 express stress responsive genes, and a lack of BDNF may be responsible for producing the physical degeneration in the brain caused by stress. NT-3 might also be important in providing an adaptive role in coping with stress (Smith *et al.*, 1994).

Corticosteroids are adrenal steroids whose secretion is triggered by stress (Magarinos *et al.*, 1996). Glucocorticoids have a role in structural change during chronic stress. Glucocorticoids are not toxic in themselves, but they inhibit glucose transport in hippocampal neurons, making the hippocampus more vulnerable to insult (Smith

et al., 1994). (The normal role for glucocorticoids is to maintain the granule neurons, which are the site for neurogenesis, or the birth of new neurons, in the dentate gyrus of the hippocampus. NT-3 is also involved in maintaining the granule neurons.)

In animals, stress increases dopamine release in several regions of the brain, showing that stress produces neurochemical changes in the brain other than corticosteroids (Abercrombie *et al.*, 1989).

Subordinate male tree shrews lost weight and had elevated levels of cortisol in their urine soon after the onset of conflict with dominant male tree shrews. The decreased body weight was related to a lower intake of food and water. The increased urinary cortisol levels indicated a prolonged activation of the HPA axis. The subordinate shrews withdrew from the field of vision of the dominant shrews, changed their sleeping patterns and had increased adrenal hormone levels and decreased gonadal activity. Dendritic atrophy occurred in the hippocampus and contributed to cognitive impairment, which was found to be reversible (Magarinos *et al.*, 1996).

Cacioppo (1994) found that social stress also has a negative impact on the immune system, because it down-regulated (switched off) several genes important to multiple aspects of cellular immune function. It is thought that the genetic down-regulation is mediated by corticosteroids released when chronic stress is experienced. Brief stress does not activate corticosteroids. It has been suggested that non acute stress activates the sympathetic adrenomedullary system, but not the HPA axis (Cacioppo, 1994).

What does this mean for counselling?

While it seems that experiencing some stress isn't harmful (and is probably stimulating) it is obvious that experiencing chronic acute stress is damaging to the brain. Effects such as memory loss, cognitive dysfunction (such as an inability to think clearly) and low immunity to disease and infection can be expected to be observed in clients who have a high level of long term stress in their lives. Many clients may not associate these symptoms with the stress that they are experiencing, and put their constant bouts of the 'flu down to a 'bad year' or their constant forgetfulness down to 'stupid mistakes'.

Clients under stress may be more emotional, prone to crying, or being dramatic about things that would not normally be such a big deal, commonly known as 'making mountains out of molehills'. Clients under stress may also have symptoms of anxiety. Because the adrenal glands, which are part of the HPA axis, are located in the abdomen above the kidneys, this may be responsible for the sick, nauseous feeling reported by many people under stress, due to an over supply of adrenalin.

A counsellor with insight into the effects of stress on the function of the brain can bring these to the attention of the client. They might enquire about what life was like before the onset of stress, whether the client observes the effects of stress that they are experiencing in people they know who are not under stress, and how they think their own life might be different if stress wasn't present. How would they be coping with life without the stressful stimulus? What would they be doing? Is the client currently behaving like the subordinate tree shrew and changing their behaviour to accommodate relationships that cause them stress? Is stress causing changes to eating or sleeping patterns? Is it affecting their demeanour, their body language, the volume of their voice, whether they speak or not in certain situations, their self-esteem, or where they go and where they don't go?

The encouraging thing is that while stress negatively affects the structure of the brain and neurochemically transcribes genes in a way that exacerbates this process, when the stressful stimuli are no longer present, these effects are reversible.

If clients, either coming in to the session, or in discussion with the counsellor, identify that they are being affected by chronic stress, the next stage is to also identify the goal relating to that stress. Does the client wish to do nothing, because the benefits of enduring the stress outweigh the physical effects? (For instance, a client may insist that they wish to continue to work for an employer who bullies them because the money is so good.) Do they wish to explore their options further, or do they wish to work toward being stress free? And are there any compromises needed between these choices, such as looking for solutions to reduce stress while remaining in the stressful situation?

If the client's goal is to reduce or eliminate stress, then the counsellor can support them in deciding how this will be achieved. Techniques such as those used in solution focused counselling are ideal for this.

As discussed above, a good place to start is to identify what life would be like without the stressful stimuli and to enquire how the client would be behaving differently. Would they have stopped working for their employer, ended their relationship, talked to the bank, or asked someone they know for help (among other possibilities)? The picture of how life will be when it is stress free again can lead to the solutions, or at least the small steps, that the client can implement to get to their goal of managing or eliminating stress.

Narrative techniques are also useful in externalizing stress as something that the client can strategize against, rather than something internal which is an integral part of their life. Narrative strategies can also help clients to look for exceptions to stress, and show clients that there are probably already times that stress is not present, pointing the way to identifying what conditions are needed to be free of stress.

Both narrative and solution focused counselling modalities also use scaling, where clients place themselves on scales from one to ten, and in this case, they could chart their progress as to how they are improving in the fight against stress and to what level they are still being affected. This will allow improvement to be charted and celebrated, because at least some of the stress in our lives is related to other people and therefore may not be able to be eliminated completely. For instance, a parent of a terminally ill child may not be able to eliminate stress, but they can still work to manage that stress, by allowing other people to care for them (the client) and to help them to get through the time of sadness and distress.

When counsellors support clients to develop a plan to manage or eliminate chronic stress, they are helping the client to get to a position where they are more emotionally resilient, less constrained by fear, more physically healthy, and where they can think, learn and remember more clearly. This puts them in a better place to resolve other issues in their lives, or to make positive change.

Chapter 5
Specific Dysfunctions

Some clients find themselves with diagnosis for specific dysfunctions and others will describe symptoms that may correspond with one or more of the broad categories currently accepted by the psychiatric profession and listed in the DSM-IV (American Psychiatric Association, 2000). The neuroscience literature, and the arising implications for counselling which relate to these dysfunctions, are described below.

Post Traumatic Stress Disorder

What do we know?

Post Traumatic Stress Disorder (PTSD) is generally defined as memories of trauma which intrude into daily (or nightly!) thinking unbidden, and which are unable to be suppressed. Sometimes these take the form of visual flashbacks, perceived as being experienced in the present time, which the sufferer is not able to 'switch off' (Brewin, 2001). Flashbacks can be triggered by external or internal cues (Brewin, Dalgleish and Joseph, 1996; Brewin, 2001). PTSD is often accompanied by disorganized thinking and an incomplete memory of the traumatic memory, a sense of unreality, and a sense of time distortion (Brewin, 2001). Symptoms that can occur before PTSD is present, and which can predict its onset, are: time distortion (where time slows down or speeds up), a feeling of bodily distortion, emotional numbing and an elevated heart rate after trauma (McNally, 2003). Bremner *et al.* (2007) also list dissociation (time distortion, a feeling of unreality or dream like state, watching the self as if in the

third person, an outsider disconnected from the body, and seeing as if through literal tunnel vision or a wide angle lens) as a common predictor for PTSD. Glutamate, released during stress, is thought to play a role in dissociation (McNally, 2003). (See also the next section about dissociation.) Excessively high levels of glutamate are toxic to the brain (Bremner *et al.*, 2007).

Childhood physical or sexual abuse, a pre-existing mood or anxiety disorder, or a family history of mood or anxiety disorders predisposes people to PTSD after a traumatic event (McNally, 2003). Other risk factors for PTSD include a prior history of stress, a low level of education, a young age, and a lack of social support. The most prevalent risk factor is a history of childhood sexual abuse (Bremner *et al.*, 2007). Pessimism is also a contributing risk factor for PTSD if the person subsequently experiences trauma. The incidence of PTSD in Dutch peacekeepers stationed in the former Yugoslavia during the civil war was significantly higher in those who had had a negativistic view of the world beforehand (McNally, 2003). Guilt and shame, as well as fear, are thought to be contributing factors to PTSD. Some examples of this are failing to protect a child from harm, causing an accident, or committing war time atrocities (McNally, 2003). Guilt and shame could also contribute to PTSD in sexual abuse victims. Many traumatic PTSD memories don't fit in with the affected person's trait self-knowledge, or their prior knowledge about how the world is (Brewin, 2001).

Longitudinal studies show that reporting of traumatic events varies over time, and those with the most PTSD symptoms, from the group who had experienced traumatic events, reported extra traumatic experiences not reported in the first interview in subsequent interviews, and also self-rated their trauma as more traumatic than they had initially (McNally, 2003).

Brain areas involved in the stress response are the medial prefrontal cortex, the hippocampus and the amygdala (Bremner *et al.*, 2007). Stress, through excitatory amino acids, decreased brain derived neurotrophic factor (BDNF) and increased glucocorticoids (see also the section on stress in Chapter 4) is associated with the loss of branching neurons in the hippocampus, and an inhibition of hippocampal neurogenesis, or the formation of new neurons. This may explain the range of memory deficits often associated with PTSD (Bremner *et*

al., 2007). Both antidepressants and enriched environments have been shown to mitigate this (Bremner *et al.*, 2007). There is an increased cortisol response to stress found in those diagnosed with PTSD (Bremner *et al.*, 2007).

Neuro-hormonal systems that act on the brain to modulate PTSD symptoms include norepinephrine and glucocorticoids. Dysfunction of these systems, including as a result of stress, produces PTSD symptoms and a failure of medial prefrontal cortex/anterior cingulate activation when re-experiencing trauma is thought to be the neural explanation for the failure of fear extinction in PTSD sufferers (Bremner *et al.*, 2007).

Neuroimaging studies of patients with PTSD show that there is a correlation with a low volume of hippocampal white matter in the brains of those patients (Hull, 2002; McNally, 2003). Twin studies showed that in identical twins, where one had been exposed to a traumatic event and developed PTSD and the other hadn't, the hippocampal volume of both twins was low, suggesting that low hippocampal volume is a risk factor for developing PTSD, not an outcome as was previously thought (McNally, 2003). Smaller hippocampal volume has been found in women who have experienced childhood sexual abuse, or other childhood abuse, who have also developed PTSD and/or other brain dysfunctions, such as dissociative disorders or depression (Bremner *et al.*, 2007).

One study, using a different imaging technique, found focal white matter lesions in a fifth of all the PTSD subjects studied (Hull, 2002). Higher IQ, or above average cognitive functioning prior to the traumatic event, has been shown to be a protective factor against the occurrence of PTSD (McNally, 2003). This could be related to better hippocampal volumes.

In those affected by PTSD there is decreased activity in Broca's area of the brain when triggers to the traumatic memory are experienced, which may limit the sufferer's ability to put the experience into words, as Broca's area is thought to be connected to semantic memory as well as speech (Hull, 2002). It has been shown that there is a decline in verbal declarative memory function after experiencing combat in war, compared to the base-line function before going to war (Bremner *et al.*, 2007).

Brewin (2001) proposes that the vivid flashbacks associated with PTSD are due to the memory being stored in a memory system that is not connected with verbalization or higher thought process, and is instead characterized by the re-experiencing of the traumatic memory at a lower sensory level. For this reason, it is thought that transferring the traumatic memory to oral or written form will help with both the organization and effective processing of the memory, so that it doesn't continue to be experienced at a purely sensory level.

The hippocampus does not function well under stress, while the function of the amygdala is advanced when stress is present. There are neural routes to the amygdala which can bypass the hippocampus (which helps with contextual learning) and this may be why traumatic memories have the potential to be stored differently. Declarative memory (which includes episodic and sematic memory, for events and facts respectively) is affected by a decrease in function of the hippocampus, and a procedural memory process may inappropriately store the memory. Procedural memory does not distinguish between past and present, which may be why flashbacks of past traumatic memory can be experienced as occurring in present time. The amygdala is strongly connected to the visual area of the brain, which could link with the visual nature of PTSD flashbacks. The amygdala, which is the area of the brain associated with fear, may be responsible for triggering the flashbacks (Bremner *et al.*, 2007; Brewin, 2001). PTSD sufferers have increased amygdala activation to triggers eliciting fear (Bremner *et al.*, 2007; Hull, 2002).

Flashbacks, although unpleasant, may be necessary to access the memory in order to transfer it into declarative memory (Brewin, 2001). Dissociation, commonly experienced alongside flashbacks, probably makes the transfer of the traumatic memory from procedural memory to declarative memory less likely (Brewin, 2001). But if memory can be correctly transferred, it is then subject to the normal inhibitory control of the hippocampus over the amygdala.

Desensitization, or the oral or written reconstruction of the traumatic event, is often an effective technique for treating PTSD (Brewin, 2001; Hull, 2002; McNally, 2003), but care must be taken not to re-traumatize the subject, and such treatment should be undertaken with care at a very slow pace. Some clients will re-experience the trauma as if it is currently happening while reconstructing it. This treatment only

has value if a client is having flashbacks or visual ruminations in non-verbal procedural memory, which need to be transferred to declarative memory (Brewin, 2001). Initial reconstruction of the memory via typing on a keyboard produces less arousal than writing out the traumatic memory longhand (Brewin, 2001). Entire narratives of the memory are not necessary, just the relevant 'uncontrolled' parts of the traumatic memory need to be reconstructed, and this may just involve snippets of memory. Sometimes only shapes or colours, textures or types of movement might need to be reconstructed so that they can be properly transferred to declarative memory (Brewin, 2001).

Other useful therapy involves re-storying (see the section on memory in Chapter 3 for a more complete explanation) and reframing the traumatic memory so that it fits in with the subject's sense of self, and trait self-knowledge (Brewin, 2001; refer to the section on memory and the self in Chapter 3).

What does this mean for counselling?

Early counselling is vital in the prevention of PTSD, before traumatic memories can be excessively rehearsed. Counselling allows organization of memory into a coherent narrative, and the counsellor can support the client to choose a strengths based perspective that focuses on the positive parts of their response, and normalizes the lack of agency that we can have in the face of external environmental factors and the human fallibilities which predispose us to mistakes (such as tiredness, not checking assumptions or failing to check for risk). Counselling allows the development of an organized, preferred perspective.

There is a possibility that traumatic memories which are excessively rehearsed could end up being stored in procedural memory; with a cue or trigger activating a neural shortcut that goes directly to the emotional experience, without the process of cognitive thought. If this is happening, counsellors will need to work with clients to practise pausing at the external cue and applying cognitive thought to choosing their reaction before they act. Desensitization is a similar technique where counsellors support the relearning of response to external triggers by starting off with very small doses of the trigger or cue,

helping clients experience safety in its presence, creating new learning and new neural pathways for this, and gradually increasing exposure to the trigger until the former automatic response is extinguished.

Internal cues are also triggers for PTSD flashbacks or uncontrollable memories, and in counselling desensitization will involve the very slowly paced recreation of the unbidden and/or flashback part of the traumatic memory into verbal or written form. Typing the memory may cause less stress for those whose flashbacks are particularly vivid. Extreme care needs to be taken not to re-traumatize the client when using desensitization techniques, and the client should set the pace and have the right to pause or desist at any point. The creation of an oral or written memory is important for the transfer of the traumatic memory from automatic procedural memory to declarative memory, where the hippocampus has control over the retrieval of that memory.

When guilt and shame are contributing factors in PTSD this may be caused by the mismatch of trait self-knowledge with the traumatic memory. Reframing the memory so that the client can see alternative reasons for their behaviour, other than that they failed in their duty or that they were a 'bad' person, can allow the memory to be properly integrated with the client's other neural networks that represent their sense of self.

Many clients affected by PTSD will have had traumatic childhoods and are likely to have experienced some form of abuse, such as sexual abuse. When treating people for PTSD, counsellors should remember that there is a high likelihood of vulnerability and low resilience. Early childhood trauma could be the cause of low hippocampal volume, which is common in those affected by PTSD. In turn, low hippocampal volume could be the cause of other risk and/or causal factors for PTSD, such as stress, mood disorders and anxiety, low IQ, low educational attainment and a negativistic outlook. Any or all of these could lead to the lack of social support that is also associated with PTSD. Dissociation (see the following section), common in PTSD, is also linked to sexual abuse in childhood, which is possibly developed as a coping strategy.

It may be valuable for the counsellor to check in to see whether it is the goal of the client to address any of these other features if they apply. Encouraging increased brain plasticity (through exercise, diet and good sleep), addressing mood dysfunction (using Omega 3, getting

sufficient exercise, practising mindfulness), and supporting the client to build social support networks are some of the ways in which this could be achieved (see Chapter 6). Exercise and better social networks are also likely to provide an enriched environment, which helps to reverse the impoverished hippocampus. Early childhood is the best time to increase the neurons in the brain (hence the lower volume in those who have had particularly traumatic childhoods) but we now know that the brain is still creating new neurons into adulthood, and that we can maximize the efficient use of whatever neurons we do have when brain plasticity is working at optimum levels.

For those who are experiencing mood disorders at the time of counselling, and for whom this creates a barrier to finding the energy to put their goals from the counselling session into practice, the counsellor should discuss with these clients the possibility of a referral to mental health services to see if some form of antidepressant treatment is right for them. The counsellor can also check in with the client as to any other strategies which they have found helpful in the past for managing or decreasing stress or depression. For some, a long hot bath with bubbles might do the trick; for others, it might be time out from everyday life, perhaps by going to a retreat, taking a holiday, or sending their dependent children to relatives for a short period.

Talking therapies, such as counselling, are likely to be helpful for those suffering from PTSD as they not only offer an opportunity to turn memory into a verbal reconstruction, they also offer an opportunity to increase verbal communication skills, so that any other past trauma, or any trauma experienced in the future can be appropriately processed. Accessing a more positive sense of self, and their capability to have agency in their lives through strengths based work and reframing and re-storying (as described in the section on memory) are likely to provide a good foundation for addressing and treating PTSD.

Dissociation

What do we know?

There are several permutations of dissociation. One form, known as Dissociative Identity Disorder (DID) used to be known as Multiple Personality Disorder (MPD). It has been concluded that rather than a single subject expressing many personalities, each seemingly different personality is a fragment of the same personality, poorly integrated. Dissociative subjects report difficulty with coherent memory and sense of self-identity across time (Spiegel, 2009). Dissociation is now defined as a failure to integrate aspects of identity, memory, consciousness and perception (Spiegel, 2009). Symptoms of dissociation include time distortion, a feeling of 'unrealness', or a 'dream-like state', seeming to experience leaving the physical body, or to be floating outside of it, observing the scene as if the subject was a third party rather than a participant, and viewing things as if through a tunnel, or conversely, through a wide angled lens (Bremner *et al.*, 2007). McNally (2003) also lists time distortion (speeding up, slowing down), bodily distortion and emotional numbing as characteristics of dissociation associated with PTSD (see the previous section on that topic). Dissociation is predictive of PTSD, and Spiegler (2009) proposes that dissociation is a chronic, severe form of PTSD.

International studies link dissociation with childhood abuse, including experiencing childhood sexual abuse, and/or childhood physical abuse. There was also a common history of drug abuse by the childhood caretakers of those affected by dissociation (Spiegel, 2009). Dissociation has its roots in trauma, making those who have experienced childhood trauma particularly susceptible. Dissociation from emotions, such as a numbing response to trauma, may help the subject to cope with the trauma while simultaneously impeding the correct cognitive processing and creating of neural pathways for the trauma which would allow it to be integrated into learning and memory systems in the brain (Spiegel, 2009). Examples of dissociation during the trauma experience include floating outside the body, time distortion and absorbing the self into an environmental detail, such as the pattern of the wallpaper (Spiegel, 2009). Although adaptive at the time, these coping mechanisms are likely to lead to subsequent disorganization in perception and the processing of perception.

Brewin (2001) proposes that traumatic memory can be inappropriately stored in the procedural memory that allows us to automatically react to external and internal triggers without the cognitive processing normally done through the neural pathways of declarative memory. Procedural memory is effectively a neural shortcut from trigger to action or reaction, which allows a lot of information to be stored in a very small space (or on a very short pathway) because the normal step by step neural connections have been condensed through habit over time (Klein and Lax, 2010; Wolfe, 1998).

Those who are affected by dissociation can experience the symptom of amnesia or memory loss. This can take the form of a 'fugue' where the person suddenly realizes that they are not where they should be, and that there is a blank space in their memory leading up to the present time. The fugue state is often associated with travel, and the sudden regaining of identity in an unexpected location. In some sufferers of dissociation, this fugue can last for years, and in some cases they have been living entirely different identities. At other times, the loss of identity is not accompanied by a new identity. The period of fugue can be quite short (Spiegel, 2009).

DID sufferers can find that one fragment of personality is aware of another, but not vice versa. Sometimes one fragment of personality will seek to punish another fragment, or to express anger towards them (Spiegel, 2009).

Spiegel (2009) says that pathological dissociation is most likely in those who have experienced trauma within their own families, rather than those who have experienced external trauma, and he believes that this is because of 'role conflict' where the child must rely on those who have harmed them for what they need to survive, such as food, shelter and protection from the outside world. Spiegel (2009) theorizes that needing to hold conflicting views of the same adults in their life can lead to tension and confusion, which complicates memory retrieval and provides a biological need for memories of the trauma connected with the caregiving adult to remain separate from other memories of them, which creates disconnection and a lack of integration among neural memory pathways. Higher levels of childhood trauma within the family correlate to greater levels of dissociation (Spiegel, 2009).

What does this mean for counselling?

Early trauma can mean disorganization of neural pathways, as the client has made use of distorted perception and incorrect neural storage of memory (probably in procedural memory rather than in episodic and semantic memory, which are subtypes of declarative memory; see also the section on memory in Chapter 3) in order to cope with the traumatic experience. Our learning is represented by the neural pathways we build in the brain, and when this is subverted by trauma, especially persistent experience of trauma, what we know and understand of ourselves, our identities and our experiences becomes confused.

Storage of traumatic learning and memory in procedural memory means that the affected person can become instantly reactive to trauma cues, which may not have specific connection to trauma in present time, such as a scarf, a certain coloured car, a spoken phrase or stress in general. The opportunity to process the cue by thought and to put it into context using the hippocampus and higher executive thinking is not available. The cue automatically leads to the behaviour or behaviours, which are in turn automatic. Many clients have also habituated dissociation into procedural memory as a stress response.

Our learning about ourselves and about the world is based on the neural connections that we store in our long term memories. (See also the sections on memory and memory and the self in Chapter 3.) If these are disorganized, or not wholly integrated and cross connected with each other in a coherent way, it will be difficult to form a coherent sense of self, hence the self-reports from those who experience dissociation of struggling to hold themselves together in one coherent identity. Those who are affected by what used to be known as multiple personalities are probably using each fragment of personality to experience the unintegrated emotions associated with the abuse they have experienced. One fragment may express anger, while another may be more loving. Sometimes a personality fragment may blame themself and wish to punish themself, and this is an aspect of response to abuse which may respond to reframing of the trauma within the counselling session.

Distortion of perception occurs as a coping mechanism for trauma or cues that have preceded trauma or been concurrent with it in the

past. Dissociation of perception is likely to also be stored in procedural memory, as automatic as walking or reaching out to grasp an object.

Memory loss and non-integrated memory and learning are going to create a disorganized context for counselling where the client brings themselves and their perceptions to the table (in this case to the session) and counsellors will need to be patient with inconsistency of narrative and world view in any clients who may have been, or are suspected to be, affected by dissociation. Indeed, such symptoms regularly displayed in counselling should alert the counsellor to the possibility of dissociation in the client.

Visual technique may be helpful to assist the client to begin to integrate and fit the pieces of their identity and experience together. This could take the form of a timeline, a diagram, a chart or even art work, where the client can begin to reconcile seemingly paradoxical information or beliefs. The counsellor can help the client to identify and record these. The client may not initially be aware of the conflict between two statements that they make at different times.

Counsellors should be aware that this is difficult work for clients, and can disrupt delicate but poorly balanced equilibriums that the client uses to hold themselves in the interim. These unintegrated neural pathways for learning and memory are what make the client susceptible to stress and further trauma, as well as the continuing effects of dissociation, but the goal to work towards a different state of integration and progress towards healing must be that of the client. It is not up to the counsellor to decide to 'fix' the client, or to fix them on behalf of someone else's behest. Dissociation is a survival strategy, and the client must be ready to let go of it and replace it with something else, or they will suffer further trauma.

Counsellors should acknowledge the client's strength and will to survive as evidenced by the dissociative coping strategies, and counselling should be a safe place to explore what these are, including differences in their perception, without the client being made to feel that they are 'crazy'. Counsellors have a role to play in normalizing dissociation as an adaptive response.

For many clients, dissociation is going to be connected with their memories and associations, past and current, of childhood caregivers. The client may, for a variety of reasons, need or want to have on-going contact with that person. They may find it difficult to cut contact with

family, particularly if they have limited support networks; they may not be assertive enough to resist that person; or they might have common connections with them, such as their other parent (i.e. the partner of the first abusive caregiver), which may mean that the relationship continues to be sustained. If the client is a child or teenager, they may have continued contact through access arrangements, or because the caregiver has made changes in their life that make them safer in that role. Sometimes, due to legal or statutory decisions, children remain in what a counsellor may consider to be unsafe care.

Even if the client no longer has any contact with that caregiver, for reasons of choice, location or the death of the caregiver, the client may still experience significant feelings of ambivalence and paradoxical emotion connected with this person. The role of the counsellor is not to persuade the client to see the caregiver as 'evil', but to help them to understand that most people are a complicated collection of personality traits and behaviours, and that while the behaviour of their caregiver towards them was unacceptable, it is possible to hold many complex perceptions of the same person and to integrate them all. The counsellor may be able to support the client to experience and express the negative emotions that they have toward this caregiver while they continue to hold any positive perceptions or memories that they also have.

Clients who no longer depend on their former caregiver for protection or sustenance may wish to fully acknowledge their formerly repressed anger, pain or betrayal, and not be interested in any positive focus regarding that caregiver, and if this is their choice, counselling should be a safe space for this to occur, a safe space that was not available to the client when they were younger. Many counsellors are of the opinion that forgiveness is necessary for a client to be whole and healed. Forgiveness is a process that begins with anger, and it is only in the later part of this process that a client is ready to forgive. Clients set their own pace, and they will know if they arrive at the place where forgiveness is right. Counsellors should allow for other parts of the process such as acknowledgment, anger, questioning and grief to come first, and different clients will progress through these stages at different paces, with some taking only a short period, and others taking many years.

For those who are still in regular contact with the abusive caregiver (particularly children and young people) the counsellor should co-construct a comprehensive, practical and achievable safety plan with the client, which is also age appropriate, and make sure that the client has a clear vision around this safety plan. The plan should include safe people and safe places as well as immediate actions towards safety.

If dissociation is to be regarded as a long term form of PTSD, as suggested by Spiegel (2009), then desensitization (which is currently the most effective treatment for PTSD) and the reconstruction or recreation of the traumatic memory, or cue to the memory which provokes dissociation should be considered by the counsellor if the client is a willing participant. Once again, the counsellor should take care to proceed very slowly and at the pace comfortable for the client. This reconstruction could be a typed or written story, it could be a verbal reconstruction, or it could be a representative art work. Sometimes a sentence or a line on a piece of paper is enough progress for one session.

It could also include exposure during counselling to a particular trigger, such as a smell or a texture, initially for a very short period of time, which is gradually increased over subsequent sessions until the client is confident of safety with that trigger present. Once again, moving at the client's pace and having client consent are of the utmost importance. New learning in connection with that trigger can rewrite neural pathways, but as per all effective learning this needs repetitive practice, and then needs to be followed by an interval after which the skill is once again practised. (See also the section on learning and attention in Chapter 3.) Care should be taken by the counsellor to ensure that the client is adequately debriefed and not experiencing any dissociative symptoms before the session ends.

Counsellors can also work with clients to develop detailed plans for what to do in response to problematic stress, including consciously pausing and choosing not to react in automatic ways, defining the problem, and developing a simple action plan to address the situation. This problem solving technique is a good general technique to teach clients as it can be replicated in any situation of stress.

As new learning takes place, whether through integration of neural pathways which have been kept separate and have been seemingly unable to be logically fitted together, or through reconstruction and

transfer of memory from one type of memory to a more appropriate and accessible type subject to processing through higher executive thought processes, the client can begin to organize and control memory more efficiently and to practise new responses to former triggers and stress.

Finally, it is important to remember that not all clients with dissociative symptoms will disclose sexual or physical childhood abuse. Some may have experienced a different trauma. Others may be experiencing poorly integrated memory as a result of coping skills, may not be ready to discuss it, or may just not think that the counsellor is the right person, or that counselling is the right forum for them to process this.

If a counsellor is aware that a client is experiencing dissociative symptoms, they can say that dissociation can be the result of childhood abuse, and then the ball is in the client's court and the decision to start a conversation about this is entirely up to them. Some may choose to do so several sessions later, when a relationship of greater trust has been formed. Others may never do so at all. It is not the counsellor's responsibility to 'uncover' hidden things about the client. Clients deserve the dignity and the agency of choosing when or if to share knowledge about themselves.

Depression

What do we know?

Depression can range from low level depression to major depressive disorder (MDD). It is characterized by feelings of sadness, worthlessness and hopelessness, loss of motivation, and a lack of pleasure in activities that were formerly enjoyed (Manji *et al.*, 2003; Martinowich, Manji and Lu, 2007; Porter, Bourke and Gallagher, 2007). It can include sleep dysfunction, a slowing of physical reactions and disruption to normal appetite (Manji *et al.*, 2003). Memory and attention can also be affected (Porter *et al.*, 2007). Depression without mania is sometimes known as unipolar depression, as opposed to Bipolar Depression, Bipolar Disorder or BAD (Heilig, 2004; Manji *et al.*, 2003; Porter *et al.*, 2007). (See also the following section on BAD.)

Major depressive disorder is often co-morbid with other dysfunctions, such as anxiety, personality disorders and psychosis (Porter *et al.*, 2007) (see also those sections in this chapter). Many of the underlying brain mechanisms thought to contribute to depression are also thought to contribute to anxiety (Heilig, 2004; Martinowich *et al.*, 2007; Reul and Holsboer, 2002). Often, depression is triggered by stressful situations (Heilig, 2004; Martinowich *et al.*, 2007; Reul and Holsboer, 2002), but a genetic predisposition also exists (Manji *et al.*, 2003). It is thought that impairment to neuroplasticity and cellular resilience may be the underlying causes of mood disorders, such as depression (D'sa and Duman, 2002; Manji *et al.*, 2003). Poor cellular resilience may be contributed to by both genetic factors and repeated depressive episodes, as well as by stress and elevated levels of glucocorticoids (Manji *et al.*, 2003).

People diagnosed with depression exhibit changes in regions of their brain, such as the limbic system, the hippocampus, the basal ganglia and the amygdala, and other cortical areas associated with emotion and thought processing. Postmortem studies suggest that there is a reduction in the size of the neurons in the orbitofrontal cortex, a reduction in the number of glia (a second type of brain cell) and the size of the glia in the prefrontal and orbitofrontal cortex, as well as a decrease in the thickness and volume of the basal ganglia (grey matter) in the brain of depressed subjects (D'sa and Duman, 2002; Kempermann and Kronenberg, 2003; Manji *et al.*, 2003).

Glia are responsible for regulating synaptic glutamate concentrations, including the clearing of excessive glutamate, and energy homeostasis (homeostasis is the constant balancing of biochemical and physical processes necessary for healthy function), as well as for releasing trophic (nurturing) factors that participate in the development and maintenance of synaptic networks. Impaired function of glial cells may prove integral to dysfunctions in synaptic plasticity and to the underlying causes for mood disorders (Manji *et al.*, 2003). Abnormal metabolism in the caudate (rear) nucleus of the basal ganglia and in some frontal regions of the brain has been observed in those affected by depression (Porter *et al.*, 2007).

Imaging studies show impairments in the cerebral blood flow (blood flow within the brain) and with the glucose metabolism in limbic and cortical structures described above (D'sa and Duman, 2002;

Manji *et al.*, 2003). There is a reduction in prefrontal cortex, ventral striatal and hippocampal volume as well. The hippocampus is a core component of the higher executive brain functioning necessary for memory, attention and other cognitive processing, and its role in the pathology of depression is likely to affect these abilities (Porter *et al.*, 2007). The limbic system, associated with the regulation of emotion, includes the hippocampus, as well as the amygdala, and is believed to be associated with the dysfunction of depression (Porter *et al.*, 2007).

The overall picture is that cellular loss and volume decrease are associated with depressive disorders (D'sa and Duman, 2002; MacMaster *et al.*, 2008; Manji *et al.*, 2003; Martinowich *et al.*, 2007). It is not known whether this is a cause or an effect of mood disorders, such as depression (Manji *et al.*, 2003). It is thought that a failure of neurogenesis, or cellular renewal, plays a large part in the pathology of depression (D'sa and Duman, 2002; Kempermann and Kronenberg, 2003).

New pharmacological treatment research is focusing on stimulating neuroplasticity and cellular resilience rather than on the neurotransmitter system of the brain. Those antidepressants which focus on neurotransmitters such as serotonin and norepinephrine only show efficacy after an initial period of repeated administration (days to weeks) suggesting that a cascade of downstream effects is responsible for their therapeutic effect (Manji *et al.*, 2003). Kempermann and Kronenberg (2003) contend that the system which regulates serotonin may be connected to neuroplasticity, as explained later in this section.

Increased levels of neurotrophic factors such as BDNF, which promotes brain plasticity and cellular health, have been shown to be associated with response to treatment with antidepressant medication, or to recovery from depression (Gonul *et al.*, 2005; Martinowich *et al.*, 2007). People diagnosed with depression have lower levels of BDNF prior to being treated for this mood dysfunction (not prior to its onset). The extent of the low levels of BDNF corresponds to the severity of clinical depression (Gonul *et al.*, 2005; Manji *et al.*, 2003). BDNF also facilitates the release of the neurotransmitters glutamate, GABA (y-aminobutyric acid), dopamine and serotonin in the brain (Manji *et al.*, 2003).

Stress can induce down-regulation of BDNF in the hippocampus (Gonul *et al.*, 2005). Chronic stress also reduces the levels of BDNF

in the dentate gyrus, the part of the hippocampus that is the site of the formation of new neurons (Smith *et al.*, 1994). The promoter of the BDNF gene contains CRE, and can by induced by CREB (a gene transcription factor). Antidepressant treatment increases CREB phosphorylation (a biochemical process involving the addition of phosphate) and CRE mediated gene expression in the limbic brain regions (Gonul *et al.*, 2005).

Stress can also induce LTD (long term depression, or a rigidifying of synaptic connections) in the adult hippocampus, which is not usually subject to LTD. In general, various forms of stress impair LTP (long term potential, or a capacity for synaptic change and flexibility). Mature BDNF is widely recognized as a regulator of LTP in the hippocampus. It is likely that currently available antidepressants encourage BDNF signalling and expression, leading to an increase in hippocampal LTP (Martinowich *et al.*, 2007).

Stress can also cause the atrophy (wasting away, decrease in size, deterioration, etc.) of neurons. (See also the section on stress in Chapter 4 for a more in-depth explanation of how this process works.) Glutamatergic transmission, to which glia are integral, is implicated in stress induced hippocampal atrophy. Corticotropin-releasing hormone (CRH) made in the hypothalamus is also activated by stress, producing corticotrophin, which releases corticosteroids (Reul and Holsboer, 2002). CRH is thought to be associated, along with excessive glutamate, with neuronal atrophy in the hippocampus (Manji *et al.*, 2003; Porter *et al.*, 2007).

Stress also affects the neurons in the amygdala (Vyas *et al.*, 2002), suggesting a reason for the common overlap between depression and anxiety. (See also the sections on stress in Chapter 4 and on anxiety in Chapter 5.) There is a large subset of people affected by depression who have glucocorticoid hyper secretion (over production) or who exhibit a hyperactivity of the hypothalamic–pituitary–adrenal axis (HPA axis) (D'sa and Duman, 2002; Porter *et al.*, 2007; Reul and Holsboer, 2002).

Glucocorticoids are a class of adrenal steroids which usually maintain the granule neurons in the dentate gyrus, the site of neurogenesis, but which can also inhibit glucose transport (and the capacity for energy production in hippocampal neurons) (Manji *et al.*, 2003; Smith *et al.*, 1994). Cortisol is the major glucocorticoid and the

primary stress hormone. High cortisol secretion levels are present in depressed subjects and are associated with impaired memory (Porter *et al.*, 2007). The HPA axis works overtime in reaction to chronic stress because a loss of inhibitory hippocampal control is combined with a gain in the excitatory control exerted by the amygdala. (See again the section on stress.) This leads to a deterioration in cognitive function coupled by a heightened fear response (Smith *et al.*, 1994; Vyas *et al.*, 2002).

An under-activation of Neuropeptide Y, which is mediated by both the hippocampus and the amygdala, leads to increased emotional response, and an over-activation of this neuropeptide has the opposite effect. (Neuropeptides are molecules of amino acids that comprise proteins in the brain.) Evidence suggests that a network of circuits utilizing Neuropeptide Y signalling, including the hippocampus and the amygdala, acts as an internal 'alarm system' in the face of stress. In the long term, this serves as an adaptive mechanism in response to chronic stressful stimuli. Neuropeptide Y levels are likely to contribute to the pathology of depressive disorders, and an up-regulation of the Neuropeptide Y gene may signify an ability to cope successfully with stressful situations (Heilig, 2004). Many of those affected by depression are also affected by what is called 'catastrophic thinking', where they are overwhelmed by perceived failure or perceived negative feedback and are unable to cope with the stress of that situation (Porter *et al.*, 2007).

Reduced neurogenesis (the production of new neurons) occurs in response to both acute and chronic stress. Ageing also decreases the rate of neurogenesis, probably as the result of the up-regulation of the HPA axis, and an increase in corticosteroids, such as glucocorticoids. The rate of neurogenesis can also fluctuate in tandem with lower estrogen levels in women (Manji *et al.*, 2003). In addition, the serotonergic system, regulating the neurotransmitter serotonin and its distribution in the brain, affects the regulation of adult hippocampal neurogenesis. Several current antidepressant medications regulate the uptake of serotonin in the brain (Kempermann and Kronenberg, 2003).

HPA axis hyperactivity is measurable by increased cortisol levels in plasma, urine and spinal fluids, enlarged pituitary and adrenal glands, and down-regulation of frontal cortex CRH receptors. Decreased levels of corticosteroid receptors are present in the hippocampus of

those diagnosed with depression or Bipolar Disorder (Manji *et al.*, 2003). Postmortem studies have found decreased levels of extra cellular signal related protein kinase (ERK), which is part of a neurotrophic dependant mitogen-activated protein kinase (MAP) cascade, in the hippocampus and cerebral cortex of depressed people who committed suicide (Kempermann and Kronenberg, 2003). (A mitogen is a substance which induces cell division and transformation.)

The anterior cingulate, a region of the brain near the hippocampus thought to direct attentional focus, may also have a role to play in depression (Etkin *et al.*, 2005; Porter *et al.*, 2007). The anterior cingulate can be divided into three parts: the dorsal (top), ventral (bottom), and rostral (front) sections. In those affected by depression, changes in both the dorsal and ventral regions have been observed through functional magnetic resonance imaging (fMRI) brain imaging techniques. The rostral section of the anterior cingulate lies between the other two sections. It receives input from both of them and its function is thought to be to integrate the two. The dorsal region is activated by non-emotional tasks, and the ventral region by processes involving mood and emotion. The rostral section is therefore needed for integrating emotion and cognition and deciding what to focus attention on. Its role is probably to resolve discrepancy between emotion stimuli and conflicting mental content.

In MDD the rostral area of the anterior cingulate may be needed to regulate the effects of negative mood over perception, thoughts and behavior. People with higher levels of activity in the rostral area of the anterior cingulate are probably in a better position for recovery through talking therapies and vice versa (Etkin *et al.*, 2005). Because the anterior cingulate has a major role in the focusing of attention, this may provide the link to the decreased motivation experienced by those who are affected by depression.

White matter abnormalities have been observed in the brains of depressed patients, and also those with BAD (see also the following section on that topic). These abnormalities can be caused by a variety of factors: demyelination (the loss of myelin; a combination of fat and protein which usually coats the cell, and assists in the speed of neuronal connectivity), loss of axons, minute brain cysts, dilated vascular (blood vessel) space, the death of living cell or tissue, or small stokes or malfunctions of the blood vessels in the brain. They

are likely to disrupt normal neuronal connectivity. The exact role that they might play in mood disorders, such as depression and Bipolar Disorder is unknown (Manji *et al.*, 2003). (See also the following section on BAD.)

Elderly subjects who experience late onset depression are likely to have white matter abnormalities (Porter *et al.*, 2007). Because neurogenesis decreases with age, this could also contribute to late onset depression (Kempermann and Kronenberg, 2003). All known pharmacological antidepressant treatments which have been demonstrated to have efficacy against depression stimulate the first stages of adult neurogenesis (Kempermann and Kronenberg, 2003). A failure of neurogenesis may be related to a failure in cellular plasticity (Kempermann and Kronenberg, 2003).

Early and prolonged treatment of depression may be necessary to prevent the structural deterioration and cell atrophy associated with mood disorders if these things turn out to be the result of depressive episodes, rather than a pre-existing vulnerability towards it (Manji *et al.*, 2003). Overall, studies indicate that a reduction in hippocampal volume appears to be related to the number of severe depressive episodes experienced (Porter *et al.*, 2007). Etkin *et al.* (2005) say that combined use of medication and talking therapies in the treatment of MDD has been shown to be more effective than either of those approaches alone.

What does this mean for counselling?

Many counsellors have their own preferred modes of counselling and preferred strategies for working with people affected by depression. The techniques and strategies contained in this section are not intended to replace what counsellors are already doing, but instead to complement and add to what therapists are already finding effective.

Depression is very closely linked to the experience of stress in a person's life. A poor coping response to stress is likely to trigger or exacerbate depression. Stress can be combated on many fronts. The first potentiality is for counsellors and clients to do an environmental audit together, to see what potential stressors can be eliminated from the client's life. These might be associated with work or the client's

living environment, including both the home and the neighbourhood in which they live. Stressors can be as simple as loud noise or bright lights or unsuitable routines, such as staying up late at night.

Some stresses involving other people may not be able to be eliminated, or the client may choose not to separate themselves from these (e.g. children or an elderly parent). In some cases, counsellors can work with clients to explore whether or not the client should distance themselves from stressful people in their life, such as an abusive partner or an adult son or daughter who is a source of stress. A plan for change should emerge from an environmental audit if the client identifies goals for eliminating stressors which are affecting them.

The second way in which stress can be combated is through the development or extension of social support networks and social connections for the client. Clients who have support people in their lives can not only relieve stress by talking it through with people who are a part of their lives on a regular basis (a trouble shared is a trouble halved!) but also share some of the stress load that they are under with these people. For instance, a neighbour might help with childcare so that the client can get some respite, or an uncle might help with tasks around the house or garden that are overwhelming the client.

Another way of managing stress is to break it down into manageable tasks. The problem solving technique currently in vogue is perfect for this, and this strategy is associated with positive outcomes for depressed clients, including those at risk of suicide (Bell and D'Zurilla, 2009). In essence, it involves identifying the problem (specifically what is happening versus what the client would like to happen), brainstorming potential solutions without worrying about whether the solutions are feasible or not (to encourage lateral thinking), choosing one or two solutions from this brainstorm to move forward with, and detailing the small, specific steps which are necessary to make them happen. This process should be kept simple and brief. Clients also need to practise consciously pausing and choosing to use this technique, rather than moving into habitual brain pathways connected with internal messages of powerlessness, hopelessness and being overwhelmed (Bell and D'Zurilla, 2009).

As ever, repetitive practice of this technique is required for it to become automatic. Defining a course of action leads to a sense of control and agency over the source of stress and combats the sense of

powerlessness. Reflecting back (possibly during a counselling session) also means that clients begin to build brain connections and pathways for recognizing an ability to manage in stressful situations.

In addition to focusing on stress, counsellors can suggest that clients manage the depression that is affecting them through the use of cardio exercise (exercise which raises the heart rate) because it is a powerful promoter of the production of the neurotrophic factor BDNF (see Chapter 6). Identifying a support person who will encourage this exercise plan, and maybe even participate in it alongside the client, could help to mediate the lack of motivation often experienced by those struggling with depression.

Clients may also want to investigate the use of Omega 3 to manage depression (see Chapter 6). Omega 3 fatty acids provide the brain with docosahexaenoic (DHA), which supports the growth and function of nerve tissue and cells, increasing memory, learning and cognition. Omega 3 may also have a role in the myelination of axons and therefore the repair of damaged white matter in the brain. Omega 3 is required for the activation of Syntaxin 3, which is essential for dendrite growth. The section on Omega 3 in Chapter 6 provides evidence of its efficacy in the management of depression and its contribution to brain health. It is widely available without medical prescription and has no documented side effects.

Counsellors should be mindful of the tendency for depression to be co-morbid (co-exist) with other brain dysfunctions, and to be especially closely linked to anxiety. Asking the right questions, and carefully listening to the information that the client has to offer about what is happening for them, will ascertain whether this is the case or not. Assumptions should not be made without hearing what clients, who are the experts in their own lives, have to say. Some clients may experience depression on its own; for others, it may be linked with other mood disorders and/or mental health dysfunctions, and the counsellor should modify or add to what they are doing together with that client accordingly. (See the relevant sections in this book, according to client information, and/or consider suggesting to the client that the counsellor make a referral to mental health services.)

If there is a genetic predisposition towards mood disorders in the client's family, the counsellor can give information on preventative measures the client can take against the onset of depression: exercise,

Omega 3 supplements, good sleep habits and the effective management of stress. Counsellors should also keep in mind the risk factor of late onset depression in elderly clients, and the fact that BDNF production can be linked to women's menstrual cycles, and not overlook depression in these instances.

Those with depression are likely to have a more negative focus and perspective on their situation because of the workings of the anterior cingulate. Reframing and refocusing by the counsellor, and the client practising doing the same, may help to build new neural pathways for a more positive focus. For instance, 'he was rude to me, so I'm a loser' can be reframed as 'he's a rude person because he is unhappy, and the way he talked to me was no reflection on me'. Counsellors can encourage clients to do this by halting the conversation when the client brings forward a negative perception and getting the client to brainstorm alternative perspectives, including those which put the client and their situation in a more positive light. This will usually take prolonged, repeated practice, as depression puts the brain into a state of LTD where the brain connections are more rigid and fixed, rather than LTP where the brain connections are softer and ripe for synaptic change.

Finally, there is a great deal of debate about the efficacy of pharmacological medications for depression, and people tend to have fixed positions on this. Counsellors should encourage exploration of the possibilities of people seeking medical treatment from their GP or mental health service, especially for more severe depression. Pharmacological treatment is not right for everyone, but many people are significantly helped by it. Good evidence shows that early and prolonged treatment can prevent the brain deterioration that comes with successive depressive episodes. There is also evidence to show that a combination of medication and therapy works better than either alone in the treatment of depression. It is possible that correcting brain function in the short term through medical intervention means that clients are better placed to uptake and comprehend the learning they do in counselling, such as strategies for managing stress. Such techniques, once learned, are likely to help prevent further depressive episodes.

Bipolar Affective Disorder

What do we know?

Note: For the purposes of this book, Bipolar Disorder, which sometimes uses the acronym BPD, will be know by its alternative acronym, BAD (Bipolar Affective Disorder) to distinguish it from Borderline Personality Disorder, which also uses the acronym BPD in the literature.

BAD is likely to arise from the complex intersection of multiple gene susceptibility and environmental factors (Schloesser *et al.*, 2008). The dysfunction has a chemical basis in the brain (Zarate and Manji, 2006). BAD is characterized not only by mood disturbance (commonly cycling between a low, depressive mood and a hyper, manic mood) but by a cluster of cognitive (thought), motor (movement), autonomic (nerve system), endocrine (hormones and the glands that make them), and sleep/wake disorders (Schloesser *et al.*, 2008). Research into BAD is moving away from focusing on trying to affect change in neurochemicals, and into highlighting the role that neural circuits and synapses, which make up neural pathways, and brain plasticity play in controlling the function of the neurochemicals (Schloesser *et al.*, 2008; Zarate and Manji, 2006).

A growing body of evidence supports BAD arising from abnormalities in cellular plasticity cascades, leading to dysfunctional information processing in the neural circuits and synapses. Many of the cellular plasticity abnormalities in BAD play important immediate roles in synaptic plasticity as well. This is also likely to explain the fact that BAD often co-exists with other brain dysfunctions (Schloesser *et al.*, 2008; Zarate and Manji, 2006).

Postmortem studies have showed reduced density of both neuronal and glial brain cells in some areas of the brains of people who have been affected by anxiety. Glia have roles in synapse function, myelination, neurotransmitter energy burning processes, immunity and providing trophic support to neurons (Schloesser *et al.*, 2008; Zarate and Manji, 2006). These characteristics may disrupt the neuronal connectivity necessary for normal brain functioning. Neurogenesis, or the creation in the brain of new neurons, appears to be an important role of effective treatment (Schloesser *et al.*, 2008). The reduction in glial cell counts could contribute to a reduction

in energy supply to the neurons and a reduced clearing of excessive synaptic glutamate (Zarate and Manji, 2006).

Glutamate is also believed to have a role in BAD. Neuronal atrophy in the HPA axis is thought to be caused by exposure to high concentrates of glucocorticoids. This can cause a reduction of cellular resilience in the hippocampus, and probably causes a related reduction in the expression of BDNF, which is necessary for the survival and function of neurons. Glucocorticoids also regulate the glutamatergic system. Glutamate is the major excitatory synaptic neurotransmitter in the brain and regulates functions such as synaptic plasticity, learning and memory. Abnormal activity in this system is likely to contribute to impairments in brain neuroplasticity and cellular resistance in those with BAD (Zarate and Manji, 2006).

Cell loss and atrophy of both neurons and glia (non-neuronal brain cells) are likely to represent a pre-existing vulnerability to BAD (Schloesser *et al.*, 2008) as well as being a long term outcome (Schloesser *et al.*, 2008; Zarate and Manji, 2006). Volumetric (size) abnormalities in the brain can be seen in first episode BAD patients and also in unaffected siblings (Schloesser *et al.*, 2008). Abnormalities in the white matter in the brain have been observed in both elderly and young populations of patients with BAD (Schloesser *et al.*, 2008). Grey matter volume is also decreased in the frontal cortical area in those with anxiety, and this is matched by increased ventricular size (cavities separating discrete regions of the brain) although grey matter volume over all is similar to that of healthy subjects. (Increased ventricular size may mean that this has been incorrectly calculated, and that grey matter volume is actually reduced in those with BAD; Schloesser *et al.*, 2008; Zarate and Manji, 2006.)

Several proteins implicated in neurotransmitter release and transfer between synapses, have been studied in relation to BAD, and some have been found to be reduced or increased in the brains of those affected (Schloesser *et al.*, 2008). Excessive activation of protein kinase C (PKC) dramatically impairs the cognitive functions of the prefrontal cortex. Regulating PKC results in the reversal of risk taking behaviour, hyperactivity and hedonistic drive, all of which are characteristic of the manic phase of BAD (Zarate and Manji, 2006).

Glycogen Synthase Kinase-3 (GSK-3) is a protein which is a key component of multiple neurotransmitter and signalling pathways

which are dysfunctionally implicated in BAD. It has an important role in many cellular processes including metabolism/energy conversion, proliferation/cell division, cell specialization, the creation of new axons and synapses, cell development, and the elimination of old cells (Schloesser *et al.*, 2008; Zarate and Manji, 2006).

GSK-3 is regulated by serotonin and dopamine and is responsive to psycho-stimulants and antidepressant. GSK-3 is in turn a major regulator of cellular plasticity and resilience. Manipulations of GSK-3 signalling cascades in animals produce both manic and depressive effects (similar to those experienced by those with BAD) and this is the only thing so far shown to produce these effects (Zarate and Manji, 2006). GSK-3 also has an effect on regulating circadian rhythm (a genetic body clock, regulating physical cycles including sleep/wake cycles; see the section on sleep in Chapter 6) in diverse species, and lithium, an effective pharmacological treatment for BAD in many cases, shares this effect (Zarate and Manji, 2006).

Neurochemically, fMRI has found low levels of n-acetyl-aspartate (NAA) in the brains of those diagnosed with BAD, particularly in the hippocampus, and in the limbic and limbic related areas of the brain which regulate emotion. NAA is localized to mature neurons and synthesized within mitochondria (the energy producing components of cells). Mitochondrial function has major effects on neurotransmitter function. Once again, it is believed that 'upstream' brain functioning, which may be genetically coded, affects the function of the mitochondria. The mitochondria themselves are not the original site of dysfunction (Quiroz *et al.*, 2008; Schloesser *et al.*, 2008).

Increasing evidence shows mitochondria to be important in the process of synaptic plasticity and increased synaptic strength. Synaptic activity increases expression of mitochondrial encoded genes. Calcium sequestering cells in the brain, regulated by mitochondria, play a key role in many regions of the brain, including those implicated in mood disorders (Quiroz *et al.*, 2008). Mitochondria are also likely to affect the synthesis of GSK-3 (Quiroz *et al.*, 2008).

Genes differentially expressed in those with BAD include those involved in the mitochondrial electron transport chain. There is also a down-regulation of antioxidant genes in BAD affected people (Quiroz *et al.*, 2008). Lower pH has also been observed in the brains

of those diagnosed with BAD. This may indicate a shift from oxidative phosphorylation to glucolysis reducing efficiency and total energy output, possibly as a result of mitochondrial function, as mitochondria are normally responsible for oxidative phosphorylation (Quiroz *et al.*, 2008). Histone deacetylase, which affects gene regulation, is also believed to play a role in BAD, and some pharmacological treatments target this by acting as histone deacetylase inhibitors (Zarate and Manji, 2006).

It is likely that a major neural defect is the inability of those with BAD to adjust to normal loads of stress (including environmental stress, hormonal and neurochemical stress and pharmacological induced stress) without reactively overcompensating with related processes of the brain, leading to a pendulum affect in terms of brain states, including mood (Schloesser *et al.*, 2008; Zarate and Manji, 2006). Environmental stressors are associated with a substantial increase in risk for the onset of mood disorders, such as BAD, in susceptible individuals (Zarate and Manji, 2006). Stress has a direct link to mood disorders, including BAD (Vyas *et al.*, 2002).

Evidence suggests that the earlier a patient receives appropriate medical treatment intervention for BAD, the lesser the long term deterioration of neurons, glia and both white and grey brain matter, all of which affect neuronal and synaptic plasticity (Zarate and Manji, 2006). Lithium and other mood stabilizers appear to have the beneficial effect of promoting neurogenesis in the dentate gyrus cells of the hippocampus and there is a resulting increase in the volume of grey matter in the brain (Schloesser *et al.*, 2008; Zarate and Manji, 2006). Current antidepressants are only effective after a few weeks of usage, suggesting that it is the downstream effects rather than the neurochemical state on which they are acting (Schloesser *et al.*, 2008).

A growing accumulation of evidence also suggests that BAD is a systemic condition with downstream physical effects. This means it affects people in a holistic way; besides having an impact on brain function/dysfunction, it is associated with cardiovascular disease, diabetes, obesity and thyroid disease (Schloesser *et al.*, 2008; Zarate and Manji, 2006).

What does this mean for counselling?

It seems that there is a genetic predisposition to BAD and this is not something that a counsellor can affect. However, early treatment of BAD is important to arrest further deterioration of the brain through atrophy and loss of neuronal density. Providing clients with pathways to diagnosis through prompt referral to psychiatric services if BAD is a possibility, or suggesting that they visit their GP for this referral, will make a big difference to the course of the brain dysfunction if BAD is confirmed. Medications such as lithium (which promotes neurogenesis, and is a generally effective treatment for BAD) are available through those psychiatric services.

Counsellors can also suggest avenues through which clients and families can find out more about the condition of BAD in order to determine whether this might be what is affecting them. People provided with clear information, or sources of information, are able to make informed choices about whether to seek psychiatric help or not, preferably while their brains are still in a position to make good decisions.

Counsellors can support clients to do anything else which might encourage neurogenesis (the formation of new neurons) and good brain health through the encouragement of trophic factors such as BDNF, which nurture and maintain the health of neurons and other brain cells. This might mean helping clients get the support to develop exercise programmes, eat well and get good sleep (see Chapter 6). In particular, good sleep, if it is achievable, may have positive implications for the function of GSK-3. Melatonin supplements can regulate the same circadian clock gene in the brain that is affected by GSK-3, and exercise is effective in encouraging the production of BDNF.

Something counsellors can, and do, work with is supporting clients to manage stress, which is believed to be a major contributor to BAD, often triggering a first episode. Those who have read the previous section on stress will know that stress can cause neuronal atrophy in the hippocampus, reduces BDNF levels in the dentate gyrus (the part of the hippocampus where new neurons are formed) increases dopamine release (which is one of the regulators of GSK-3), affects the ability to sleep, and has an impact on the immune system (which could contribute to the physical dysfunctions correlated with BAD).

Counsellors working with clients affected by stress can support changes in behaviour or cognition to lower a client's stress levels, or alternatively encourage clients to think about how they might make environmental changes towards the same ends. External stress is not always controllable, so putting together a safety plan for the management of stress is something that counsellors and clients can do together. This could include the use of techniques such as time out, mindfulness and mediation (see those sections in Chapter 6), talking through and processing worries with members of the client's support network (or even the building of support networks, depending on where the client is at), the use of exercise, participating in activities that are pleasurable for the client, and the use of sensible self-reward for appropriate stress management.

For those diagnosed with BAD, or for those who might possibly be diagnosed in the future, general health check ups by GPs or similar should be encouraged by counsellors on the 'everything affects everything' basis, because of the strong links between BAD and diabetes and heart disease, and so on. Being physically well contributes to good mental and brain health.

Anxiety

What do we know?

Anxiety is a trait which can help us cope with dangerous situations, allowing us to anticipate them and to respond to them quickly and efficiently (Venault and Chapouthier, 2007). Fear, the emotion most strongly linked with anxiety, is an aversive state which readies us biologically to take action to avoid threat. As part of the fear response, the metabolism changes the way it works to allow us to fight, take flight or freeze, depending on which is most appropriate (Ohman, 2005). Anxiety disorders represent biases in information processing of external stimuli to become focused on potential threat in inappropriate contexts (Pine, 2007). Because anxiety is linked to other processes such as learning, memory and emotion, it involves many parts of the brain and many brain transmitter systems, thereby involving brain plasticity

on many levels (Venault and Chapouthier, 2007). Both genetic and environmental factors are believed to contribute to the neural circuitry which shapes threat responses (Pine, 2007).

MacMillan *et al.* (2003) have found that children and adolescents aged 8–17 years affected by anxiety had a significantly enlarged amygdala on both sides of the brain when compared with controls. Anxiety is often co-morbid with MDD but the amygdala was not enlarged in those with depression alone. The extent of the volume increase was correlated with the severity of the anxiety experienced. Pine, Cohen and Brook (2001) found that having high levels of anxiety in adolescence was a risk factor in developing MDD later in life, and that anxiety in childhood and adolescence was predictive of experiencing anxiety in adulthood.

The amygdala is implicated in emotional behaviours relating to both fear and aggression (Adolphs, Tranel and Damasio, 1998; Davis, 1992). The amygdala also helps to play a role in social judgement of others, based on facial appearance. People with impaired function of the amygdala are more trusting of others when asked to make social judgements based on appearance characteristics (Adolphs *et al.*, 1998). Conversely, it is probable that people with enlarged amygdala would be more fearful when making the same social judgements.

Increased amygdala activation is associated with anxiety states (Pine, 2007; Shekhar *et al.*, 2005) and this has been evidenced by fMRI scans (Anand and Shekhar, 2003). The activity of the amygdala is regulated by the excitatory action of glutamate, and the inhibitory action of GABA, both chemical neurotransmitters (Shekhar *et al.*, 2005). Many of the areas to which the amygdala projects are involved in the processing of fear, anxiety and emotion in general and the behavioural response to these. In particular, the closely connected lateral hypothalamus activates the bodily responses to fear. Also implicated are other areas, which along with the lateral hypothalamus are associated with the ability to produce an increased startle reflex, panting of breath, hyper arousal, pupil dilation, increased blood pressure, pale skin tone and sweating (Davis, 1992).

Stimuli already associated with fear are more readily located by subjects who seem primed to search them out, despite distractions (Ohman, 2005). These stimuli automatically activate a fear response through a fast subcortical brain network centred on the amygdala

(Bishop, Duncan and Lawrence, 2004; Ohman, 2005) because the neural wiring for the processing of these fear associated stimuli has become more efficient through a process known as 'conditioning', an automatic response to a previous repeatedly experienced stimulus routinely associated with a specific outcome (Pine, 2007).

In the absence of conditioning, when the duration of the stimulus allows a longer, more continuous processing, the amygdala response is enhanced, and a slower, more comprehensive network which involves the anterior cingulate (involved in directing attentional focus) and the anterior insula (involved in consciousness, self-awareness, emotion, perception and motor control) is activated (Bishop *et al.*, 2004; Ohman, 2005; Pine, 2007). It is thought that attention constantly directed to the detection of threat and to fear response neural circuitry by the anterior cingulate may reduce the amount of attention available for normal goal directed behaviour (Pine, 2007). The amygdala itself also has an important role to play in normal learning, so abnormal amygdala activation may affect the capacity to learn and remember, or be responsible for a different neural learning profile (Pine, 2007).

When the activation of the amygdala is mediated by the faster subcortical pathway, passing only through the superior colliculi (involved in spatial orientation and the processing of and response to sensory stimuli) and the pulvinar nucleus of the thalamus before accessing the amygdala, it can operate on low spatial frequency information, or incomplete perceptual data, such as might be collected when vision is impaired for any reason (Ohman, 2005). Bishop *et al.* (2004) found that potential threats outside the field of spatial awareness were more closely attended to by highly anxious subjects than by subjects judged to be low anxiety, and those affected by high anxiety gave more attention to peripheral threat than to potential threat within their field of focused attention.

BDNF, a neurotrophic factor, is also implicated in the pathology of anxiety. Mature BDNF (as distinct from its precursor, pro-BDNF) facilitates LTP priming neural circuitry for synaptic plasticity. Low levels of mature BDNF are observed in the dysfunctions of both anxiety and depression (Martinowich *et al.*, 2007). (See also the section on depression in this chapter.) BDNF production can be triggered by antidepressant medication (Martinowich *et al.*, 2007).

Stress plays a role in preparing us to adapt to new environmental challenges (Shekhar *et al.*, 2005) but chronic stress, which activates the HPA axis (Smith *et al.* 1994; Vyas *et al.*, 2002), and the resulting overproduction of the neuropeptide CRF can lead to anxiety and other mood disorders, such as depression (Reul and Holsboer, 2002; Shekhar *et al.*, 2005). (See the sections on stress in Chapter 4 and depression in this chapter for more detailed explanations.) The amygdala expresses high concentrations of CRF receptors, and is also a major source of CRF containing neurons. Within the amygdala, the basolateral nucleus is important in the regulation of anxiety and its symptomatic responses. The synaptic plasticity of the basolateral nucleus plays a role in mechanisms regulating emotion. Stress induced plasticity in the amygdala (and in particular, the basolateral nucleus), which is subject to LTP, and the resulting neural wiring arising from the plastic changes, probably plays a large part in the transition from normal vigilance responses to pathological anxiety (Davis, 1992; Shekhar *et al.*, 2005). Disrupting the function of the basolateral nucleus leads to a disruption in conditioned fear responses, and an inability to retrieve the emotional information associated with these. One of the roles of the basolateral nucleus is assigning emotion, including anxiety, to input from sensory stimuli (Shekhar *et al.*, 2005).

As well as dysfunction of the amygdala, anxiety is also linked to functional and/or structural deficits of the hippocampus and the medial and ventral prefrontal cortex, which act in conjunction with the amygdala. The amygdala is connected to the prefrontal cortex. Better prefrontal cortex-amygdala coupling is associated with lower levels of generalized anxiety disorder (Pine, 2007). The fear response of the amygdala can be inhibited by the activation of the dorsolateral and orbitofrontal cortices, which form part of the higher order, executive functioning network of the brain, located in the prefrontal cortex (Ohman, 2005).

What does this mean for counselling?

Anxiety is a dysfunction with a unique characteristic; being affected by it can be the very thing which precludes seeking (or following through) treatment to manage or eliminate the dysfunction. A large subtype of

anxiety dysfunctions are characterized as 'social anxiety disorder' in the DSM IV (American Psychiatric Association, 2000) which can be summarized as anxiety involving interaction or proximity to other people. Someone experiencing anxiety will find it very difficult to approach a practitioner to discuss their condition and very difficult to attend follow up appointments, whether in the practitioner's space or in their own home. (Even at home, someone experiencing anxiety can be triggered by the situation of someone unfamiliar in their private space. Some clients, however, may find that their home environment is less anxiety-provoking than an unfamiliar one. If this is the case, home visits may be indicated, if these are possible for the counsellor to arrange with the client.) Anxiety often leads to avoidance of potential treatment to mitigate the condition.

Much of this is outside of a counsellor's control. What counsellors can do is create conditions which are perceived as safe enough for someone affected by anxiety to bring themselves to return, if they do initially present for service. Anyone who seems shy, private, withdrawn, who constantly checks the environment around them (including their watch) or who is eager to leave should be treated as if they could be affected by anxiety. The counsellor should also keep in mind that people who do not display these characteristics, but who have been under chronic stress or who have experienced trauma, may also be affected by anxiety. Often the presence of anxiety is enough to keep the client from declaring it, so counsellors may not be aware that anxiety is a factor in the client's life.

On a personal level, counsellors can keep their voices low and soft (as if speaking to a frightened child) and their movements sedate rather than sudden or exaggerated. Clients should be allowed to position themselves in the counselling session room, and a chair or similar should always be available close to the door, where the client can keep the door in sight and within easy reach as they speak. Clients may also wish to put space between themselves and the counsellor to create a feeling of safety.

Additionally, counsellors can offer the client a measure of control over the environment, such as whether the lights are turned on or off during daylight hours (some clients might feel more comfortable in bright light where they can see potential threat more clearly, others may prefer a dimmer light, where they feel safer and better concealed),

whether the blinds or curtains are semi closed and/or drawn or whether the windows are open or closed, and the same for the door. As the experts in themselves, the clients are in the best position to know what makes them feel most comfortable, and what sort of environment is the most effective strategy against anxiety.

Some clients may wish to bring a support person or people to the session, at least to the initial visit. As a bond is built between counsellor and client and the client creates new brain connections for safety in the counselling environment, this might be able to be systematically and slowly withdrawn as subsequent sessions progress. For instance, the support person might stay for the entire appointment for the first few times, and after that, only be present for the initial part of the appointment but wait close by outside. Later, they might only accompany the client to the place of counselling and wait in the waiting room till the session is over. Later still, they might just drop the client off and then collect them again, followed by a time when the client can come to counselling unaided.

The time set for the appointment can be crucial. If a client is affected by anxiety, an appointment which is scheduled early or late in the day can mean that the client is less likely to encounter other people in the waiting room, or on the way to the counselling service. This is particularly important when the counsellor shares space with other practitioners or services. The timing of the sessions should also be considered, and a client may also find it easier to start with short sessions which can become longer as the client becomes more trusting of the counsellor and the counselling environment.

When the counsellor becomes aware that anxiety is a factor in the client's life, they should enquire about what the client's particular triggers are, rather than just accepting the general term anxiety. The research in the section above shows that people are primed, or conditioned, to be over vigilant for pre-established cues when they are experiencing anxiety. If any cues like this which might be present are eliminated then the client will be in a better position to attend to the work of counselling and to remember and integrate any learnings.

Some areas where a client is monitoring for threat may be outside the area of current focus, for example, outside the room, in the hallway, out of the window or in corners of the room. Counsellors can allow time for potential threat to be investigated – for example,

by looking out of the window together, checking the hallway for others – and allow time for the client to ask questions which can provide reassurance when answered. These questions might be about confidentiality (which counsellors will naturally cover in an initial contract, but which may need to be discussed in more complex detail), whether the session is being taped or observed (e.g. through a two way mirror) and who might come into the room.

Some cues might involve the client themselves, such as the discomfort of being observed by the counsellor. This may relate to how they feel about talking in front of others, or how they feel about what they are wearing, or how they look in physical terms. In this case, a counsellor may choose to accommodate the client by placing themselves in a way where they are not directly observing the client, such as a position where both the client and counsellor face in the same direction and are in line with each other. Most counsellors will recall a time when they were talking to someone in a situation other than face to face (perhaps in a non-counselling role), for example, doing dishes alongside someone, driving while someone else is a passenger or walking alongside someone, where the other person was surprisingly forthcoming with what they shared verbally. This sort of positioning can help to bypass anxiety triggered by eye contact, or the feeling of being observed.

Anxiety is often co-morbid with other dysfunctions, such as depression, Bipolar Disorder, Obsessive Compulsive Disorder (OCD) and borderline personality disorders (see also those sections in this book). It is helpful during the information gathering process of counselling, for counsellors to ask naive screening questions which will elicit answers that would indicate if any other dysfunctions are also present, so that the counsellor can refer on, or do their own work from a more holistic position. Other dysfunctions, if the counsellor is unaware of them, might prevent effective counselling work being done. For instance, if a client is affected by depression and suffers from loss of motivation, this may lead to changes and strategies against anxiety chosen by the client not being implemented by the client.

Anxiety can also lead to aggressive behaviour by some clients, as it is a state which can trigger 'flight' (avoidance), 'freezing' (being incapacitated like a deer in the headlights), but also 'fight' (aggression). If aggression is triggered by anxiety (and aggression does of course

have other triggers), the best way to manage it is to minimize threat for the client, either by making the space safer, giving them the option to exit the space, or by keeping very still and quiet while they work on their emotional regulation, if it is safe for the counsellor to do so.

The important thing to remember about anxiety is that it involves regions of the brain (the amygdala, and other prefrontal regions) which are subject to brain plasticity, and as such has the potential for change. CBT is perfect for identifying triggers for anxiety and the accompanying feelings, and consciously choosing different behaviours in response, practising them repeatedly to build new brain connections and pathways which can in time become the automatic response from procedural memory (see also that section in Chapter 3) rather than the automatic response of the pathological anxiety circuits involving the amygdala. Reframing the potential threat during the process of counselling (the cognitive dimension of CBT) can also add to this process.

The management of stress is an important topic of counselling conversation when a client is affected by anxiety (see also the section on stress in Chapter 4). Because of the over-activation of the HPA axis during chronic stress (an axis which includes the adrenal glands located above the kidneys) some clients affected by anxiety might find themselves nauseous, particularly in the mornings, as the body works to clear an over supply of adrenalin. The management of stress includes working to minimize environmental stress, and where this is not possible, organizing some respite from that stress. Building natural support networks comprised of people who will be in the client's life in the medium and long term can be a key component in this process. Counselling can help clients to identify goals and plans to address chronic stress.

Neural resilience to stress can be provided by increasing the brain's production of the trophic factor BDNF (shown to be in low concentrations in its mature form in those affected by anxiety) and an accessible way to do this is through regular cardio exercise which increases the heart rate. (See the section on exercise in Chapter 6.) BDNF also increases the brain's plasticity, allowing new learning to take place and new brain pathways to be built, perhaps in counselling, or in the integration of learnings from the time spent in counselling.

For those younger clients (under the age of thirty) affected by anxiety, as well as enhanced potential plasticity there is the advantage of the higher areas of the brain in the prefrontal cortex continuing to mature. The more developed these areas become, the more likely they are to have the ability to inhibit anxiety. (For ideas to promote optimum brain function and plasticity, see Chapter 6.) This knowledge can provide extra hope to these clients and their families.

Finally, it is important for counsellors to remember that other members of the client's family may require support. They may themselves be affected by anxiety, or by the same stresses as the client, but in addition, if they are the support person/people for the client they may have the responsibility of doing all the life tasks for the client which the client is too anxious to undertake themselves, anything from shopping to organizing and facilitating appointments. Some support people also find themselves on the receiving end of the client's anxiety based aggression, such as when they need to encourage the client to attend a compulsory appointment, for example, with a government department or a medical or helping professional. Counsellors should make sure that support people in these situations are offered referrals to another counsellor or support person who can work with them to identify and meet their own goals.

Attention Deficit (Hyperactive) Disorders

What do we know?

ADHD is associated with developmentally inappropriate capacity for attention and impulse control and motor or physical restlessness (Bush, Valera and Seidman, 2005). Attention Deficit Disorder (ADD) is regarded by some to be a variation of ADHD, but without the hyperactivity. Most diagnosticians now assess both across several axes, such as impulsivity, attention deficit, physical restlessness, difficulty identifying and planning for consequences, low empathy and inappropriate behaviour, with those positively diagnosed with ADHD scoring highly on most of the axes, which may or may not include hyperactivity (Lahey, Carlson and Frick, 1997).

Although poor parenting is often blamed, ADHD has a biological basis in the brain. Among the various current categories of psychiatric dysfunction, ADHD is one of the most heritable (as evidenced by studies of twins raised apart) and is therefore one of the most prevalent (Forero *et al.*, 2009; Lesch *et al.*, 2008). Future genetic analysis should identify the genes involved, expected to be variations of genes involved in synaptic plasticity (Forero *et al.*, 2009). More work also needs to be done to find out under which environmental conditions the genes responsible for ADHD are most likely to be expressed (Lesch *et al.*, 2008). This could include parenting styles, but may also include the richness of the environment provided during key early developmental stages, the types of food eaten, sleep patterns, stress levels, etc.

ADHD is associated with a reduced volume of grey matter in the brain (including neurons, axons, dendrites and synapses) especially in the frontal lobes, striatum and the cerebellum. Volume loss is primarily in the prefrontal cortex, which supports higher order thinking, such as the ability to pay attention, the ability to learn and remember, the ability to plan for and conceive of the future, impulse control and empathy (Aron and Poldrack, 2005; Bush *et al.*, 2005; Hoekzema *et al.*, 2011). Even small bursts of cognitive training for children diagnosed with ADHD and not currently on medication have been shown in functional imaging studies to increase the grey matter in the relevant areas of the brain. This is an example of brain plasticity. It has not yet been shown whether this increase in the volume of grey matter is stable or just transient. The higher the IQ of the child with ADHD to receive the cognitive training, the more the increase in grey matter (Hoekzema *et al.*, 2011).

Studies have also shown dopamine transporter/dopaminergic abnormalities in the striatum, which links the grey matter of the basal ganglia (Bush *et al.*, 2005). Dopamine receptors numbers are raised significantly in those affected by ADHD (Anderson and Teicher, 2000; Dougherty *et al.*, 1999; Li *et al.*, 2006). It is possible that this accounts for gender differences in the prevalence of ADHD (more boys than girls are affected) as pubescent males experience a rise in dopamine receptors, where pubescent females do not, and the symptoms of ADHD are often at a peak at this time of life for male adolescents, although for some, ADHD persists into adulthood (Anderson and Teicher, 2000; Goldstein, Ngalieri and DeVries, 2011). A connection

between ADHD and dopamine receptors may have relevance for the medical efficacy of current treatments, as these are the target sites for those medications (Dougherty *et al.*, 1999).

A meta-analysis of studies conducted on groups of people diagnosed with ADHD found that executive functions of the brain associated with the prefrontal cortex and the orbitofrontal cortex, such as impulse control, vigilance and sustained attention, spatial working memory and the ability to plan were significantly lesser than in the general population, but the authors concluded that this was not necessarily causal of ADHD and might instead be only partial causal, or even a consequence, because these things were not sufficient to explain ADHD in all the subjects of the studies (Willcutt *et al.*, 2005).

Mangina and Beuzeron-Mangina's study (2003) showed that children with ADHD had either significantly underactive brain electrical activity (50.42% of their sample), significantly overactive brain electrical activity (28.15% of their sample), or a combination of the two at different times (21.43% of their sample), when this was measured while they were performing cognitive tasks. Mangina and Beuzeron-Mangina (2003) were able to artificially manipulate these children's brains into normal range for measurable electrical activity during cognitive activity, which not only significantly improved their ability to learn during the trial tasks, but the improvements in concentration, memory and learning were still evident two years later when this group of children was retested.

Two control groups of children – one group diagnosed with ADHD who received only learning interventions and not electrical manipulation of the brain, and a second group without ADHD – were used in comparison to allow effects such as the maturation of the brain to be discounted. The electrical activity measured by Mangina and Beuzeron-Mangina (2003) is mainly moderated by the limbic system, the amygdalae, the hippocampus and the anterior cingulate gyri, which regulate attention. They suggest that electrical manipulation of the brain allowed new neural pathways to form, taking advantage of brain plasticity and correcting a defective, prefrontal inhibitory control mechanism, and/or sub-normal activation of the prefrontal brain systems.

What does this mean for counselling?

While it is not the case that poor parenting causes behaviours that should be more properly ascribed to an ADHD heritability, because of the heritable nature of the condition it is likely that a biological parent and/or one or more close family members also has ADHD, leaving them also with deficits of attention, planning, memory, impulse control (including poor emotional regulation) and empathy. If someone with ADHD is in the role of caregiver to a child with ADHD, it makes the family very vulnerable to the full effects of ADHD expressing themselves in the child, both because of poor behaviour modelling by the adult with ADHD, and because the adult is likely to be unable to plan, organize and follow up limits and boundaries for the child's behaviour.

It is also my observation that adults with ADHD are exceedingly poor at planning, remembering and keeping appointments – including appointments with counsellors, doctors, paediatricians, psychologists and psychiatrists – for either themselves or their children. Many children with caregivers who also fit the ADHD profile therefore go undiagnosed and unsupported, perpetually regarded as 'naughty children' and punished as such. This leads to a hopelessness of ever succeeding in society's systems (especially the education system) and a consequential disregard for those boundaries as they grow to become teenagers and later adults, often even behaving in active opposition to those systems (including the justice system). Coupled with a poor ability to suppress impulses and regulate emotions, this means that many children with ADHD grow up to spend time in prison, seemingly unable to learn from mistakes, or attend to future consequences when they act.

Even competent parents or caregivers who have children with ADHD may struggle to get adequate support for their children, blocked by the perceptions of professionals in the agencies who should be supporting them, peripheral support agencies and the public in general, such as that if children regarded as having ADHD were parented correctly (which ranges from 'a damn good smack' to more liberal 'positive parenting' techniques, depending on who is offering the opinion), they wouldn't have a problem and their behavioural issues would cease. This weight of perception often causes self doubt

in parents and they can swing between quite different parenting approaches, trying to get things right while finding nothing really works. Care should, however, always be taken by all involved in assessment of children with possible ADHD to differentiate children who truly do receive poor parenting and whose symptoms would be alleviated if they were parented in better ways, from those children who truly are affected by ADHD. Some children, of course, may be affected by both poor parenting style and ADHD.

Parents who are unaware of the signs that can indicate a child has ADHD may not push for an assessment or diagnosis because they too believe that there is something 'wrong' with their parenting. There is a lot of stigma still attached to medicating a child with ADHD with stimulants such as methylphenidate (Ritalin) and the common perception is that parents who do so are 'turning their children into glassy eyed robots' who behave well at the expense of the child's free will.

This, of course, is not the case, and for those whom Ritalin and related stimulant medications work, concentration and attention become achievable for the duration of the dosage, allowing a child to learn and remember and to succeed educationally. Counsellors provide talking therapy but sometimes their role must be in providing information, to both adults with ADHD and adults who have children who may have ADHD, in terms of symptoms that can indicate a psychiatric or paediatric assessment is worthwhile, and if ADHD is indicated after that, what options for treatment may be available to them.

Medication is not right for every child, and parents have the right to make their own decisions on behalf of their children, but they should do so with enough information to make informed choices. Counsellors need to avail themselves of enough basic information to educate clients about ADHD on a basic level, and also to be able to direct clients to more detailed sources of information if they wish for this. Because neuroscience is moving forward at a fast pace, it is also important to make sure that the information being given is relevant and current.

Counsellors can also play a role in providing referrals to mental health services which contain clear, objective observations by both parent/caregiver and counsellor (or counsellor and possibly other family

member if the client is an adult) that will assist mental health services to gain a clear picture of the symptomatic behaviours experienced by the client, affecting them and therefore those around them. Mental health services can be busy and overworked and often clients don't know what information is helpful to give in the appointment time allocated to them. A referral containing good observations (but not a diagnosis, because that is not the counsellor's job, or area of expertise) is more likely to lead to clients receiving the service that they need from mental health agencies. This is also helpful where the parent or caregiver also has symptoms of ADHD and presents as disorganized and lacking in concentration, because this can affect their own ability to give medical professionals clear information.

Counsellors who work holistically with families should consider it a likelihood that a parent or a close biological relative of the client will have ADHD. If the client is a child, this might mean providing support to the parent or caregiver. If the client is an adult, this could mean providing written information that can be passed along to the affected person if the client chooses to and remembers to do so. Parenting support to set limits and boundaries coupled by realistic logical consequences and positive parenting can be helpful for the parents or caregivers of children with ADHD, but all concerned should remember that difficulty with learning, impulse control, consequential planning and memory will mean that keeping to rules and boundaries will always be a struggle for ADHD children, and that keeping to routines and consistently following up limits and boundaries will always be a struggle for an ADHD parent or caregiver.

Counsellors can provide information to clients and their families about the best environmental conditions for healthy brains: healthy food, good sleep, sufficient exercise, the absence of stress, the use of relaxation and mindfulness techniques, and so on (see also Chapter 6).

In some places, it is possible to access 'neuro feedback' as a treatment for ADHD, where the client practises moderating the electrical activity of their own brain through manipulating images on a computer screen during sessions. This treatment is still expensive, but may become more affordable in the future.

Some clients who are diagnosed with ADHD, and who choose to trial medication, will find it very successful in helping them live lives in better possession of the executive functions of their brains.

A smaller percentage of those who try medication will find that it makes no difference to them. Whether this is due to the density of their dopamine receptors, or to the measurable electrical frequency of their brains when they perform cognitive tasks (there may be a difference in medication efficacy between those who have a low electrical frequency, and those who have high or mixed frequencies), to live fulfilling lives they will need different support. (Remembering that counsellors do not have the option of electrically stimulating their client's brains into a normal electrical frequency range!)

There will also be some clients, or parents/caregivers of clients, who do not feel that medication is right for them or their child. Ideally, mental health services will provide non-pharmacological therapy to these people, but in practice, busy mental health services often don't seem to have the time or resources to do so. Such services concentrate mainly on those most affected by mental health dysfunction, and are mandated to only accept the top percentage of the population with mental health dysfunction into service.

This means that much of the therapy needed to support those with ADHD will fall to counsellors and other practitioners of talking therapies. Here, counsellors have multiple roles. The first is to help the client (and their family in the case of children) to understand ADHD as a condition with a biological basis and not a wilful laziness, 'naughtiness', anger management problem or a lack of consideration for others. ADHD should be considered an 'invisible disability'. When people are in wheelchairs, no one is angry if they do not walk. If someone has ADHD, others should remain patient if they lack consequential thinking, find it difficult to resist satisfying their impulsive desires, have sudden emotional meltdowns, fail to consider others, are physically restless or easily distracted, have bad memories, are disorganized as a consequence of their lack of memory retention, and concentrate poorly on most things, failing to learn much from past mistakes. Because of difficulty with learning and attention, literacy skills are also likely to be poor. Not all those who are affected by ADHD will show all of these symptoms, but any cluster of these symptoms should be expected. People who have this invisible disability often find themselves on the wrong side of society's rules.

Lack of consequential thinking and impulse control can lead to those with ADHD developing addictions, and if this happens they

can be connected with an alcohol and drug counsellor who specializes in addiction counselling. Alcohol and drug counsellors working with clients with ADHD need to be especially patient, because often the lack of executive brain function symptomatic in those with ADHD (such as having poor impulse control) gets in the way of clients overcoming their addictions. It is likely to be most valuable in conjunction with cognitive therapy as described below, and these two types of counselling (addiction and cognitive) may or may not be provided by the same counsellor.

Those with ADHD who have been prescribed methylphenidate/Ritalin and who have addictions or poor impulse control, or who may be tempted to exceed the dosage of their medication or to sell it to others who will use it for this purpose, now have the option of a variant which cannot be used to get 'high' and therefore has no street value. (Those who truly have ADHD find an excess of the medication calming rather than stimulating. Any taking of medication above recommended dosage or without prescription should be regarded as dangerous.)

So what help or support can counsellors give other than information? Just as for those who have visible physical disabilities, support networks for those affected are vital and counsellors can work with clients and/or their families to develop these. Support networks might consist of family members, social services workers, or people connected to the client in other capacities, such as church members or members of cultural groups to which the client belongs. Counsellors can also help clients to identify which areas they need the most support in so that they can ask for specific support from these people. An example might be that a man asks his wife to make record all his appointments for him on the calendar and remind him, or a school might put a plan into place to provide one on one support to a non-medicated student with ADHD to help them focus their attention and learn better, or that a fellow church member reads letters received out loud to a person affected by ADHD.

Cognitive therapy within counselling can be provided to take advantage of brain plasticity and build new neural connections. For these new neural pathways to become permanent, repetition is required, and this means that several sessions with space between them will be needed (see also the section on learning and attention in

Chapter 3). As the name suggests, CBT is particularly suited to this type of counselling. Getting a client to enlist the support of a family member to encourage and remind them to practise at home can be helpful, especially since lack of consequential thinking can mean that the client fails to value the therapy in the moment that they should be practising it, and/or that affected clients can forget to practise in the first place, or fail to plan for that practice, commonly known as 'homework' among CBT practitioners. ADHD is not an excuse to avoid making positive behavioural change. It makes the process of change more difficult, but the plasticity of the brain means it is possible.

Examples of relevant cognitive therapy could include: practising willpower by suppressing the urge to gratify an impulse (such as eating cake or chocolate, having a cigarette, or scratching an itch) for longer and longer periods of time; practising memory tasks (such as remembering a list of words, or a sequence of numbers, which gets progressively harder); practising focusing on one thing (such as a black dot on a white piece of paper for longer and longer time spans); or practising drawing up flow diagrams of behaviours and their downstream consequences (particularly behaviours that the client has or might engage in). Counsellors can use their creativity to come up with ideas that specifically address the higher order cognitive deficits of the client.

As is often the case, counsellors need to remember that because of the neural biological basis for this dysfunction, the therapies (both addiction and cognitive) will be hard work for the client and should only be undertaken if the client is motivated to do so in order to make a change in their lives. It should also be remembered that continuing brain development over an extended period of time can relieve or diminish many of the symptoms of ADHD as the prefrontal cortex and the orbitofrontal cortex mature.

Autisic spectrum disorders, including Aspergers

What do we know?

Autistic spectrum disorders (ASD) range from Asperger's syndrome at the milder end of the continuum, to autism at the more severe (Belmonte *et al.*, 2004; Tantum, 1988). ASD is defined by dysfunctions which result in impaired social interaction and communication, obsessive interests and repetitive behaviours. Speech is often affected in those at the more extreme end of the ASD continuum, and other motor deficits such as physical clumsiness and dyspraxia may be present. Those with ASD often find it difficult to execute or interpret body language and facial expressions. The ability to empathize with and understand the motivations of others is poor. They also tend to be rigid in their logic and understanding, and to comprehend language in a literal way (Belmonte *et al.*, 2004; Mundy and Neal, 2000). Baranek (2002) also notes auditory processing problems (not a hearing difficulty, but problems with the brain's interpretation of the sounds heard by the ear) in most children and teenagers with ASD, as well as compulsive, repetitive actions.

Neurologically, one of the differences between the brains of those with ASD and the brains of those who do not, appears to be an abnormally high level of localized neural connectivity within discrete regions of the brain, which develops in tandem with an abnormally low connectivity between the differing regions. The cerebellum is particularly implicated in this failure of connectivity (Belmonte *et al.*, 2004). This may account for the effect of processing too much sensory information and the inability to filter out what is relevant and what is not. Those with ASD often have accompanying sensory sensitivities and/or sensory obsessions from a young age (Baranek, 2002; Belmonte *et al.*, 2004).

Low regional connectivity of the brain is probably the reason why those with ASD can experience an inability to integrate different functions of the brain, for instance, the function of language processing with the motor function of the tongue (Belmonte *et al.*, 2004). Physical clumsiness of fine motor muscles in the hands and fingers appears to stem from poor neural pathway preparation through initial stages of the neural connectivity rather than an inability to physically execute the

required movements (Baranek, 2002). High local neural connectivity combined with weak regional connectivity potentially incurs a higher load at later processing stages of perceptual integration, and higher order brain functions such as learning and memory (Belmonte *et al.*, 2004) (like a traffic jam on narrow, congested roads). There is some similarity in EEG measured brain activation between siblings who have ASD and those who don't, suggesting a genetic predisposition to low activity and integration between different regions of the brain (Belmonte *et al.*, 2004).

In those affected by ASD, there is a documented reduction in the number of cerebellar Purkinje cells in the cerebellum, and incomplete development of the cerebellar vermis (a narrow structure between the two hemispheres of the cerebellum) and the cerebellar hemispheres. (Purkinje cells are large complex neurons typically involved in receiving synaptic input and with outputs relating to motor function, learning and attention, and cognition.) Functionally, cerebellar activity is abnormally low on selective attention tasks and abnormally high on motor tasks, and this correlates with smaller sized subregions and the reduction in the number of Purkinje cells (Belmonte *et al.*, 2004). Purkinje cells are also associated with processes that prune unnecessary synaptic connections at specific stages of brain development (Belmonte *et al.*, 2004).

The brains of those on the ASD scale typically have an excess of localized synaptic connection. Despite the reduction in Purkinje cell numbers, fMRI imagining shows an excess of white matter and overall cerebral matter in very young children. (The cerebrum is the largest part of the brain, with its two hemispheres connected by the corpus callosum.) This is not replicated in adults, suggesting that the excessive growth occurs during early developmental periods in those with ASD, while more measured growth in those unaffected by ASD allows their brain size to catch up eventually. This is backed by measurements of head circumference. Frontal lobes show the most significant overgrowth when ASD is present. Frontal lobes are the regions essential to complex, higher executive function of the brain, such as learning and attention, social regulation and impulse control, and also to functions, such as language (Belmonte *et al.*, 2004; Mundy and Neal, 2000).

There appears to be an overlap between ASD and the genetic condition of Fragile X syndrome, with many of those affected by Fragile X showing symptoms of ASD. In the brains of those with Fragile X, dendritic spines in specific cortical regions have been observed to be dense, but abnormally long and thin, suggesting an immature development which may produce neural over-connectivity (Belmonte *et al.*, 2004).

There is also believed to be some connection between immune signalling and ASD, and maternal viral infection in mid-pregnancy has been said to be a leading non-genetic cause of ASD. Normally developing neurons express proteins known for their role in the immune system, and which are needed for specific forms of developmental and functional plasticity. These proteins are linked to Purkinje cells. Changes in the expression of these proteins may contribute to the development of ASD (Belmonte *et al.*, 2004).

There is a thinning of grey matter in the cortical areas where mirror neurons (see also Chapter 4 on mirror neurons) should be present, in those affected by ASD (Mundy and Neal, 2000). Mirror neurons are a special class of neuron found in the posterior part of the inferior cortex and the anterior part of the parietal lobe in the human brain; regions which are closely connected, forming the mirror neuron system (Iacoboni and Dapretto, 2006; Mundy and Neal, 2000). The same mirror neuron pathways are activated when a person observes the action of another person as those pathways which would be activated if the person performed that action themselves. They allow learning through watching and imitating the actions of other people (Iacoboni *et al.*, 2005; Mundy and Neal, 2000; Rizzolatti and Craighero, 2004). Those with ASD are less able to successfully imitate the actions of others. Additionally, mirror neurons are able to code an action for intention, allowing subsequent actions to be predicted by the watcher (Iacoboni *et al.*, 2005; Mundy and Neal, 2000).

The mirror neuron system is also directly connected to the limbic system, which is the emotional processing centre for the brain. This means the mirror neuron function is linked with not only imitation and understanding action motivation, but also with understanding the emotional motivation of others' actions, which is the basis for empathy (Dapretto *et al.*, 2005; Iacoboni and Dapretto, 2006). Iacoboni and Dapretto (2006) found that the mirror neuron system is linked to

language (an inability to communicate successfully via language is a common feature of the more severe end of the ASD continuum) and to facial expression. A reduction in the number of available mirror neurons, or damage to the mirror neuron system, results in an inability to predict the action or intention of others through body language or facial expression (Dapretto *et al.*, 2005). This dysfunction is also apparent in those affected by ASD (Mundy and Neal, 2000).

There are time delays in neural connection between the regions of the brain involved with the mirror neuron system in those diagnosed with ASD (Iacoboni and Dapretto, 2006). (This could be due to the weak connectivity between regions, as discussed above.) Healthy mirror neuron activity is associated with Mu rhythm suppression in the brain, and this is lessened in those with ASD (Iacoboni and Dapretto, 2006). There is a direct relationship between a lack of mirror neuron system activity and ASD symptom severity and conversely, there is a correlation between healthy functioning mirror neuron system activity and those with less severe symptoms or no symptoms (Iacoboni and Dapretto, 2006).

Thinning of the grey matter in areas involved in facial expression production and recognition (regions representing the face in the sensory and motor cortices, and in the middle temporal gyrus) has been documented, as well as grey matter thinning in other areas of the brain involving social cognition: the prefrontal cortex, the anterior cingulate, the supramarginal gyrus (part of the parietal cortex), and the middle and inferior temporal cortex. All of the regions where thinning was observed are related to the larger mirror neuron system (Mundy and Neal, 2000).

The superior temporal sulcus (part of the temporal cortex), which forms part of the mirror neuron system, is associated with eye gaze direction activation and the processing of social information related to the shifts and subtleties of eye gaze in others. This area is also subject to grey matter thinning in those affected by ASD. Consequently, those affected are less able to process social intention arising from eye gaze and are poor at meeting and holding the gaze of others, i.e. making eye contact (Mundy and Neal, 2000). There are also aural mirror neurons, related specifically to the auditory system and hearing (Iacoboni *et al.*, 2005; Rizzolatti and Craighero, 2004). This may relate to difficulties

in auditory processing experienced by many of those with ASD, in conjunction with excessive auditory sensory sensitivity.

Current findings cannot determine whether brain abnormalities in the mirror neuron system are a cause or a result of ASD. Early dysfunction of the mirror neuron system could generate abnormal development in other areas of the brain. Cortical thinning of the areas of the brain involving the mirror neuron system could also be a result of either a lack of input to specific brain areas consistent with weak regional connection, developmental abnormalities involving defective neuronal proliferation or migration and/or cell density and connectivity, or white matter abnormalities (Mundy and Neal, 2000).

Schumann *et al.* (2004) report an enlarged amygdala in children up to 12 and a half years old (but not adolescents) with ASD, and an enlarged right hippocampus at all ages in those affected. The amygdala is associated with the detection and response to stimuli which may need an avoidant response because they represent potential danger. It is strongly connected with anxiety (see also the section on anxiety in this chapter). ASD symptoms often include increased fear and anxiety in those affected (Schumann *et al.*, 2004). The amygdala develops significantly up until the age of eighteen, so while those diagnosed with ASD had more initial development of the amygdala, the amygdala of those who were not affected by ASD gradually reached the same volume but over a longer period (Schumann *et al.*, 2004).

What does this mean for counselling?

Early overdevelopment of the amygdala may mean that neural pathways for increased fear and anxiety are developed during pre-adolescent development stages, and that these pathways become reinforced and strengthened through constant and habitual use. Higher fear and anxiety are likely to be focused on social situations, where the interactions between other people can be a mystery to those with ASD as if everyone else is aware of a code to which they have no key: the code of subtle facial expression, body language and understanding the social motivations of others through these things, and also through making predictions of other people's future actions based on their prior actions. An enlarged amygdala in childhood

may preclude the opportunity to develop neural connections for pleasant or unthreatening social experiences, and this is likely to be intensified by the difficulties experienced in social understanding and communication. Communication difficulties can, in turn, be intensified by difficulties in verbal communication skills and/or auditory processing for some of those with ASD.

Counselling is usually a situation of emotional intimacy, where clients share things that they would not share with the general population, or even sometimes with their nearest and dearest. It can be a situation of intense social connection where clients and counsellors interact one on one, with the primary mode of communication being verbal interaction. Counsellors are used to interpreting subtle facial and body language cues and to making and holding eye contact with their clients.

All of this poses problems for clients with ASD, and by extension, impacts on the counselling experience. It is extremely important for counsellors to do all that they can to reduce anxiety levels for those clients on the ASD scale, particularly in relation to the interaction between client and counsellor. This applies to not only those who come in with a diagnosis of ASD (no matter which end of the scale) but to those whom the counsellor observes as having symptoms which might relate to this dysfunction (see the previous section on what we know) whether they are ever diagnosed as such or not. Not all of those on the ASD scale will suffer heightened anxiety, but many will, and it is important for counsellors to be aware of this likelihood.

So what can counsellors do to minimize anxiety in the counselling rooms? An important first step would be to allow the client to bring in a support person. Because of social difficulties, those with ASD may not have close friends to invite, but many have someone who has been kind to them either in their personal lives, or someone in a professional capacity whom they have come to trust. Many have supportive family members. Some ASD clients may have partners or someone else whom they know well and who they consider to be a friend, despite the difficulties in social interaction. Counsellors can make an overt offer of the option for the client to bring a support person to sessions, especially the first session, in an effort to make the client feel more at ease, and more importantly, safe. People who do not

feel safe and whose brains are focused on fear and anxiety are unlikely to be taking much of benefit from a counselling session.

Counsellors can also minimize sensory stimulation, so that the brains of those with ASD are not overwhelmed by perceptual data. This can include dimming or turning off bright lighting, minimizing noise disturbance (even a loudly ticking clock can be distracting or irritating to someone with a heightened sense of hearing) and checking that the room temperature is comfortable. The only way to check what is right for any given client is to explore that with them and maybe their support person. What is annoying for some (e.g. noises heard through an open window) may be comforting for others (e.g. fresh air) depending on personal neural pathways.

Some sensory experiences can be comforting, for example, a silky cloth or other piece of favourite fabric. Some clients may bring their own object of comfort without being asked, but counsellors can also have a container of such things available on the table, readily available for clients to handle if they choose. Young children, in particular, often find something to play with repetitively in such a container and this can play a role in allaying their anxiety. As well as fabrics, stress balls, novelty toys, dice and so on can provide some distraction from anxiety.

Speaking softly, and/or using a low tone of voice may be helpful, as can checking on the preferred physical distance for the client in relation to the counsellor. This can be achieved by providing a number of seating options and the counsellor indicating where they themself will be sitting, so that the client can choose their own seat with this knowledge. Some clients may prefer to sit closer to the door than the counsellor, so that they have the feeling that an escape route is available if they choose to leave suddenly if overwhelmed by all the social interaction.

Session length should be kept relatively short, particularly during the early stages of the client–counsellor relationship, or if a counsellor observes some particularly anxious cues from the client. Clients with ASD can become very tired from the effort of social contact and trying to interpret the other person's social feedback and intentions, especially in a situation, such as counselling. Because of their differences and lack of social skills, many of those with ASD have experienced bullying by other people. Building a relationship where the client comes to trust the counsellor and feels safe with them should be paramount.

Counsellors need to be prepared for a lack of social feedback from ASD clients, something which can leave a session feeling 'empty', because whether counsellors know it or not, a huge amount of two way communication between counsellor and client takes place through body language, facial expression and eye gaze. When this is missing, counsellors are left without a rich source of information on which they usually gauge not only how well the session is going from the client's perspective, and the level of rapport achieved, but which also helps them make decisions on what sort of counselling techniques and practice they will engage in at any point during that session.

Rather than wonder what is going wrong, counsellors need to accept that this is a common feature of sessions with ASD clients and to seek feedback in other ways, depending on the special ASD characteristics of that client. If verbal communication is adequate the counsellor may need to ask open questions to elicit this feedback. If not, then a form where the client can regularly provide written evaluation of the content of the session through the use of scaling or open ended questions may be appropriate. This ensures a pathway through which counsellors can check that the special needs of these particular clients are being met through counselling, and a forum in which it is safe for the client to indicate what their needs are.

Counsellors should not worry about gaining or maintaining eye gaze if it is not given, or attribute it to other motives, such as lying or untrustworthiness. Eye gaze can be difficult or overwhelming for those with ASD. Likewise, fidgeting is not necessarily a sign of boredom or disengagement, but can be a cue that the client is experiencing heightened anxiety. Special care should be taken to interpret those feedback cues which are picked up by the counsellor, as they may not have the same coding as that present for feedback in clients not affected by ASD.

Depending on how ASD affects any particular client, such as the severity of the position on the ASD scale and the symptom cluster experienced by the client, counselling interaction may need to make less use of verbal interaction and more use of other non-verbal techniques. Some ASD clients know what they want to say but cannot readily bring the language required to express that to their tongues, probably because of the weak connectivity between different regions in the brain. If this is so, it is important for counsellors (and the client's

support people) to leave extra long spaces while the client processes their thoughts through the brain's language and motor centres. This might mean waiting several minutes for the answer to a question. It is also helpful to keep questions requiring a reply simple, rather than asking complex questions which require an answer of several parts. If three sorts of information are needed, ask three questions, one at a time, and leave space for each answer.

Clients may wish to write their side of the interaction down rather than speak it, and ASD clients often write long narratives between sessions to read out in counselling to eloquently express how they feel, or to convey what has happened. Because writing can be difficult for those ASD clients affected by dyspraxia (the tight grip required to hold a pen can cause fatigue and frustration) some clients might find using a computer keyboard to be helpful. Email counselling might be one way of communicating with ASD clients, either as an adjunct to, or instead of one on one counselling where both people are in the room together. The slow pace of reply when counselling by email also allows time for thorough cognitive processing for the client.

Drawing and art and creative therapies are other modes of interaction relevant to counselling, which can suit ASD clients, as can any counselling mode which relies not on client–counsellor interaction, but on participation in the therapy providing self-insight and self-awareness for the client, as well as providing a platform for them to brainstorm their own solutions. For clients with dyspraxia, counsellors should take care to provide thick, easy to grasp art materials such as crayons, felt tips or brushes, as these do not require such a tight grip. It is important that creative therapies are suited to the client's sensory sensitivities, for example, some clients may not like the feel of paint or play-dough, or the sand in a sand tray, irrespective of whether they are children or adults.

Particular issues which may arise in counselling for clients with ASD include a lack of awareness of why other people have behaved the way that they have, a lack of empathy or awareness for the way that other people feel in response to the actions of the ASD client, an inability to form close or intimate relationships with others (including family members, potential partners and potential friends), an inflexibility in dealing with situations (and poor emotional regulation in response when anxiety arises when the situation does not evolve as expected)

and bullying or abuse from others who prey on others who are socially different, less socially skilled and/or who lack social support networks to protect them.

Counsellors can work with clients to extend their social support networks, even if this means that many in the networks are other social service or health professionals, because good support networks are important protective factors for any person. Some social skills can be taught intellectually (as described in the previous section on mirror neurons) and clients can learn the different positions of eye gaze and mouth position on an intellectual level, where they cannot learn by imitation, for example, the position of the inner and outer corners of the eyebrows, or the corners of the mouth, or how wide the eyes are open, and which emotions these are likely to convey. This learning can form part of the counselling process.

Looking at social processes from a logical point of view can be helpful too, such as analysing a social situation through bullet points written on a piece of paper and identifying where the client could have acted differently, and what would logically have followed from that difference in action. Social audits are a similar process, where the stages of a problematic event are written in the order in which they occurred. The client then awards or deducts moral points to each action of the event to determine which actor needed to do something different, and at what point. The client then brainstorms with the counsellor, and possibly their support person, what other actions could have been taken by them. An example of a social audit might be: Ben got in my way. (Ben loses one point.) I asked him to move. (I get five points.) He wouldn't move. (Ben loses three points.) I hit him and kicked him and broke his nose. (I lose ten points.) I could have asked a teacher for help and then I would have got eight points instead of losing ten, and the teacher would have asked Ben to move out of my way. This technique relies on evaluation of actions and reactions, without requiring evaluation of emotional motivation. It plays to the strengths of the ASD client by requiring logical thought instead.

Relaxation techniques are valuable tools which counsellors can share with clients. Clients can utilize these either when experiencing anxiety or when experiencing frustration when a situation doesn't go the way in which they expect it to and they cannot process the difference fast enough to produce a flexible response, which makes

them more anxious. An inability to express frustration or anxiety, or to ask for help, through the immediate processing of language can add to emotional dysregulation, which can result in fierce meltdowns or tantrums. Relaxation techniques might be passive ones such as deep breathing, mindfulness, yoga or visualizations, or active ones, such as regular exercise to provide physical release of unexpressed emotion.

A failure of the mirror neuron system may also result in an inability to name or express the emotions being experienced by the ASD client, or even to recognize that these emotions are present. Finding names for these emotions and linking them to physical symptoms which the client experiences can help clients to communicate their emotional states. For instance, being short of breath, wanting to avoid a situation or having sweat on the palms of the hands may indicate anxiety. Wanting to break something which isn't doing what the client had hoped it might do might indicate frustration. Counsellors can work with clients to support this learning.

Finally, it is important that counsellors working with ASD clients are client centred in their practice. Counselling should only proceed at a pace comfortable for the client. A slower pace may be needed for those with lower intellect, or those with verbal communication processing difficulties, and it is easy to mistake the second of these for the first if the client is slow to respond to questions. Care should be taken not to make this assumption. (Many of those with ASD have a very high intellect.) Neural connections and thinking patterns in those affected by ASD are often rigid and inflexible because of the structure of their brain, as described above. For counselling to truly make a difference to these clients, the counsellor needs to use their own empathy to understand the thought processes of the client. As described in the section on learning and attention in Chapter 3, learning is most effective when it can be integrated with prior learning because it has congruence with this.

People do not begin complicated jigsaws again from scratch because they can't fit one new piece into the picture of the jigsaw. Instead, they are most likely to discard the difficult piece. And so it is with learning. We don't discard a lifetime's learning for one new idea that doesn't fit with everything else we have experienced. A counsellor must be able to visualize the perspective of the ASD client and work from there, fitting into their jigsaw in appropriate

ways, rather than trying to create an entirely new picture from the perspective of the counsellor. The jigsaw must be appropriate for the client and the structure of their brain.

If an obsessive interest is a feature of the client's ASD, this can be an opportunity for the counsellor to develop rapport, and for integrating learning and motivation through relating the content of the counselling session to that special interest. For instance, for a client mad about rugby, the counsellor can relate appropriate social interaction to the rules of play, or to the interaction between team members needed in order to collectively win the game. If they are interested in the Twilight movie series, emotional interaction and empathy can be noticed and intellectually observed and discussed through dissecting the characters of that story together.

It is also important for counsellors to recognize and utilize strengths in ASD clients. Rigid logical thinking can lead to success in mathematical subjects and careers. An inability to empathize with others can mean less susceptibility to being emotionally manipulated. An obsessive interest can lead to a career which the client is passionate about, or at the very least to them being an interesting and informed source of information about that topic. In addition, each ASD client brings their own talents and strengths of personality, as do all clients.

Obsessive Compulsive Disorder and Tourette's syndrome

What do we know?

Obsessive Compulsive Disorder, or OCD, is characterized by compulsions and obsessions which the affected person often knows are irrational but is powerless to control. Obsessions are thoughts that repeat and cycle over and over again and compulsions are repeated behaviours, ritualistically enacted. They can include checking, washing and ordering. Everyday processes often form the basis for obsessions and/or compulsions. These obsessions and compulsions can go on for hours, and a person may compulsively check that the carpet is straight, or that the oven is off (even though they only checked moments before)

or wash their hands over and over again (Graybiel and Rauch, 2000; Van Den Heuvel *et al.*, 2009). Hoarding things (Van den Heuvel *et al.*, 2009) and praying, counting or silently repeating words may also be compulsions for some people (Chamberlain *et al.*, 2005). Some people suffer mainly obsessions (a cognitive–affective dysfunction), some mainly compulsions (an executive behaviour dysfunction) and some suffer from a combination of the two (Graybiel and Rauch, 2000).

Often fear and anxiety at not performing the behaviour that the person believes will keep them safe, despite its irrational nature, is at the root of behavioural compulsions (Graybiel and Rauch, 2000). Fear and anxiety also inform obsessive thoughts. Patients with OCD often suffer from other anxiety disorders (Graybiel and Rauch, 2000). Affected people may be secretive or lack insight about their disorder (Jenike, 2004).

OCD often runs in families (Chamberlain *et al.*, 2005; Jenike, 2004). OCD that has an early age of onset affects more males than females, and is often co-morbid with (exists alongside) other motor tic dysfunctions, such as Tourette's. OCD that begins in adulthood, peaking in the 20s, is more likely to affect women and is more sensitive to treatment. OCD is also linked with trichotillomania (compulsive pulling of one's own hair) and body dysmorphic disorder (a false picture of one's own body and appearance; see also the sections on eating disorders in Chapter 5 and healthy eating in Chapter 6) (Chamberlain *et al.*, 2005; Graybiel and Rauch, 2000).

Functional imaging of the brain suggests that in those with OCD, there is abnormal metabolic activity in the orbitofrontal cortex, the anterior cingulate/caudal medial prefrontal cortex and the caudate nucleus (the anterior part of the striatum). Activity within this cortico-basal ganglia network, which is sometimes called the 'OCD circuit', is increased when a person with OCD is resting in comparison to a person without OCD who is resting. This circuit is also abnormally active when OCD symptoms are present, and returns to normal when OCD has been successfully treated. The OCD circuit is thought to have a role in directing the formation of habitual and routine behaviours (Chamberlain *et al.*, 2005; Graybiel and Rauch, 2000).

Different sets of basal ganglia loops have specialized functions, depending on which regions of the brain they are looping through. (The

basal ganglia are interconnected masses of grey matter in the interior regions of both hemispheres of the brain, connected by the striatum.) It is thought that these loops may jam and depending on which loop it is, this may account for symptom specificity, particularly between Tourette's syndrome and OCD. The loop which is dysfunctional in Tourette's syndrome passes through the motor cortex (associated with movement, and therefore compulsive tics and twitching, etc.) rather than the OCD circuit detailed previously (Graybiel and Rauch, 2000).

The anterior cingulate cortex is also closely connected to the motor cortex (Graybiel and Rauch, 2000), which may explain the high co-morbidity with Tourette's syndrome and the compulsion of repeated physical actions. OCD patients show significant memory dysfunction (Chamberlain *et al.*, 2005), perhaps as a result of the anterior cingulate cortex, which is associated with attention, directing itself towards obsessions and compulsions rather than the functions of learning and memory.

Many studies have found abnormalities in the basal ganglia related to OCD dysfunction. These included glucose levels and regional cerebral blood flow (Chamberlain *et al.*, 2005). There are pharmacological treatments which are successful in many cases of OCD, including those which moderate serotonin and dopamine (Graybiel and Rauch, 2000). In some OCD patients, the functional abnormalities can be reversed by careful exposure to the stimuli which trigger the obsessions and compulsions, and the prevention of rituals in response (Graybiel and Rauch, 2000; Jenike, 2004). (This would represent a controlled rewiring of synaptic brain pathways.) The efficacy of responses to both medication and exposure therapy are correlated with different levels of metabolic activity in the orbitofrontal cortex and the anterior cingulate cortex. Both are part of the basal ganglia circuit (Graybiel and Rauch, 2000). Abnormally lower metabolic function in the orbitofrontal cortex of those with OCD predicts a better response to medication than CBT or similar talking therapies, and abnormally higher metabolic function in the orbitofrontal cortex predicts the opposite (Etkin *et al.*, 2005).

OCD can be heritable (as is Tourette's syndrome) and there is now also evidence that it can be triggered by bacterial or viral infections (Graybiel and Rauch, 2000). Grey matter and white matter volumes appear to be lesser in some regions of the brain in those with OCD,

which may indicate a vulnerability to the condition (Van Den Heuvel *et al.*, 2009). OCD waxes and wanes over time and successful treatment is usually the remission of symptoms, rather than the extinction of the dysfunction (Jenike, 2004).

What does this mean for counselling?

There will be some cases where CBT type counselling is helpful in mitigating the symptoms of OCD, particularly when the client falls into the category of late onset OCD. It is presently rarely practical for counsellors to obtain brain scans to find whether clients are likely to respond to this type of therapy, or to respond better to medication, so the client must have the choice of which treatment method to trial first. Clients who have not responded to pharmacological treatments in the past may be good candidates for exposure therapy. Whether therapy will be more helpful than medication or not can be predicted to a certain extent by the age of onset, so counsellors should ask when the client considers this to have been. Later onset of symptoms is more likely to mean that CBT exposure therapy will be beneficial.

Non-pharmacological therapies include the slow and careful exposure of the client to the stimuli which trigger the OCD obsessions and/or compulsions, starting with very little exposure at all, and building up the amount of exposure to the stimuli over a period of time. While exposed to the stimuli the client must try to refrain, with the support of the counsellor, from engaging in their ritualistic response, until, after many sessions, the response is extinguished. It is important for counsellors to check the emotional safety of their clients before the session ends, as a client who has not been properly debriefed may leave the session highly anxious at being deprived of their habitual routines, which they believe keep them safe. Such treatment within counselling should only be engaged in after full discussion with the client, and the client's permission to proceed.

Counsellors need to recognize that the constant looping in the brain of obsessive thoughts and compulsive behaviour has created extremely strong neural pathways to support the continuation of these thoughts and behaviours, which can easily be re-triggered, even in the absence of the initial underlying OCD circuit dysfunction. Overcoming these

pathways will be 'hard work' and it will take a long time and a lot of practice for these pathways to be extinguished and for strong new pathways to form, linked to higher order thinking, where the brain can suppress the unwanted obsessive, compulsive pathways and choose the new ones where thinking and behaviour is a choice and is not related to obsessions and compulsions. Sometimes clients can be helped to develop and use visualizations to overcome behavioural compulsions, such as concentrating on imagining their family safe within an orb of white light, rather than focusing on touching their own nose fifty times to achieve the same purpose.

Because OCD is likely to reoccur within a client's lifespan, counsellors can work with affected clients to set up a resilience plan, including early recognition of familiar symptoms and pathways to early treatment. Natural support people can be included in the plan, helping with early symptom recognition, and support in accessing treatment. This is beneficial not only for the support aspect, but because sufferers often have a lack of insight into how OCD is affecting them.

Finally, counsellors can work with clients prone to OCD to develop strategies against the poor memory retention which typically affects those who are affected, for example, a system for writing out lists of tasks and the use of memory prompts.

Personality disorders

What do we know?

Personality disorders are sometimes known as BPD (Borderline Personality Disorders). This can be confused in the literature with Bipolar Disorder, because the same initials are often used for that acronym as well (but not in this book).

Personality disorders are characterized by an inability to regulate emotions, control impulses or have successful interpersonal relationships with others (Grosjean and Tsai, 2007; Juengling *et al.*, 2002; Minzenberg *et al.*, 2008; Ni *et al.*, 2006; Schmahl *et al.*, 2003b). Self-image is usually poor (Grosjean and Tsai, 2007). Personality disorders are often co-morbid (present alongside) other dysfunctions,

such as anxiety, depression, PTSD, substance abuse and dependency and psychosis (Ducci *et al.*, 2008; Grosjean and Tsai, 2007; Schmahl *et al.*, 2003b; Zetzsche *et al.*, 2006). About half of those with borderline personality disorders also fulfil the criteria for PTSD (Schmahl *et al.*, 2003a). Those with personality disorders may experience dissociation and temporarily fail to correctly ascertain the generally accepted reality of the world. The hallucinations experienced are generally 'non-bizarre' and may last from minutes to hours (Grosjean and Tsai, 2007; Irle *et al.*, 2004). Those affected also tend to have a low empathy for perceiving or comprehending the emotions and motivations of others (Grosjean and Tsai, 2007).

An exaggerated fear response is often present (Grosjean and Tsai, 2007; Juengling *et al.*, 2002; Minzenberg *et al.*, 2008; Schmahl *et al.*, 2003a). Suicide rates in those with personality disorders are significantly higher than in the general population, and this may be connected with both impulsivity and emotional intensity (Ni *et al.*, 2006).

Diagnostic criteria have been organized into four psychopathologies: thought disturbance, emotional disturbance, impulsivity and intense, unstable relationships (Grosjean and Tsai, 2007). Personality disorders are often differentiated into three categories with indistinct and overlapping boundaries and numerous subdivisions. (The three categories are paranoid/schizoid, antisocial and avoidant.) The term borderline personality disorder is sometimes used in the literature to cover all of these, and sometimes used to cover a subset of those in the antisocial category, along with antisocial, narcissistic and histrionic personality disorders, as it is in the DSM-IV (American Psychiatric Association, 2000). For the purposes of this book, the term is used in general reference to personality disorders.

Personality disorders are associated with early childhood trauma and stress (Ducci *et al.*, 2008; Grosjean and Tsai, 2007; Irle *et al.*, 2004; Juengling *et al.*, 2002; Schmahl *et al.*, 2003a; Schmahl *et al.*, 2003b), and poor parenting and attachment (Grosjean and Tsai, 2007; Schmahl *et al.*, 2003a). Early stressors include failing to perceive or respond adequately to the child's distress (Grosjean and Tsai, 2007), neglect, separation and abandonment (Grosjean and Tsai, 2007; Minzenberg *et al.*, 2008; Schmahl *et al.*, 2003a), chaotic and inconsistent environments (Grosjean and Tsai, 2007), sexual and/or physical abuse (Ducci *et al.*, 2008; Irle *et al.*, 2004; Juengling *et al.*,

2002; Schmahl *et al.*, 2003a) and harsh parenting styles (Grosjean and Tsai, 2007). The deficit in empathy noted by Grosjean and Tsai (2007) may be due to inadequate parental modelling and therefore a failure to activate the mirror neuron system during critical periods of childhood development (Grosjean and Tsai, 2007). (See also the section on mirror neurons in Chapter 4.)

Personality disorders are believed to arise from a combination of environmental stressors and genetic vulnerability (Ducci *et al.*, 2008; Grosjean and Tsai, 2007; Lesch and Gutknecht, 2005). Those with a family history of personality disorders or psychiatric dysfunction are more vulnerable (Grosjean and Tsai, 2007; Ni, 2006). There is a link between BPD and stress (Grosjean and Tsai, 2007; see also the section on stress in Chapter 4) which aligns with the high co-morbidity between personality disorders and PTSD (Schmahl *et al.*, 2003a). Neural dysfunction relating to stress includes elevations in cortisol, elevations in glutamate and decreased BDNF levels. People with BPD respond to stress by releasing higher levels of cortisol than normal (Schmahl *et al.*, 2003b).

The prefrontal cortex seems to be most affected, including areas such as the amygdala (associated with fear and anxiety) and the hippocampus (integral to learning and memory). Functional imaging studies have found dysfunction in the prefrontal cortex in those with BPD to be pronounced (Grosjean and Tsai, 2007; Irle *et al.*, 2004; Juengling, *et al.*, 2003). The prefrontal cortex is important in the regulation of emotion and impulse control (Irle *et al.*, 2004; Juengling *et al.*, 2003; Schmahl *et al.*, 2003a; Schmahl *et al.*, 2003b). Women with BPD had decreased blood flow to the prefrontal areas of the brain when exposed to abandonment scripts (narratives/stories about abandonment) in comparison with control groups (Schmahl *et al.*, 2003a).

Small functional imaging studies have found a decreased metabolism and function in the premotor and prefrontal areas of the brain in those affected by BPD, including the anterior cingulate cortex (Schmahl *et al.*, 2003a). The anterior cingulate regulates the ability to focus attention and is also involved in emotional regulation and the determination of sensitivity to pain (Juengling *et al.*, 2003). However, Juengling *et al.* (2003) found that glucose metabolism was significantly increased in the anterior cingulate and other areas of the prefrontal

cortex, but decreased in the left hippocampus and left cuneus (part of the occipital cortex) in those with BPD.

There are differences in brain structure between those with BPD and those without. Minzenberg *et al.* (2008) found that grey matter concentration is increased in the amygdala and decreased in the anterior cingulate in those with BPD, and that there is a corresponding difference in the numbers of neurons and their quality of structure. This could account for the increased fear response and the decreased attention and emotional regulation usually present in BPD dysfunction (Minzenberg *et al.*, 2008). Zetzsche *et al.* (2006) found an enlarged amygdala was not always present, but when it was, it was most common in BPD subjects who were also affected by anxiety and/or major depression. Both lower hippocampal and amygdala volumes have commonly been described in subjects with BPD (Grosjean and Tsai, 2007; Irle *et al.*, 2004; Schmahl *et al.*, 2003a; Schmahl *et al.*, 2003b). As with several other brain dysfunctions, it is not known whether smaller hippocampal volume predisposes a person towards BPD, whether it is a resultant cause, or both (Schmahl *et al.*, 2003b). However, raised glucocorticoid levels associated with stress are known to be toxic to hippocampal neurons (Grosjean and Tsai, 2007).

Memory, sustained attentional focus, facial recognition and the correct comprehension of others' expressions, the ability to attend to conflict resolution and the ability to be empathetic towards others are all compromised in people affected by BPD. The hippocampus, its size reduced in those affected by BPD, is known to be important in memory attention and cognition (Grosjean and Tsai, 2007). It is speculated by Grosjean and Tsai (2007) that stress and a poorly socialized home environment during periods of early development contribute to the lack of brain development in the necessary prefrontal cortex regions, and that this is exacerbated by dysfunction of the neurotransmitter system, which includes the glucocorticoids associated with excessive stress and *N*-methyl-D-aspartate (NMDA) which plays a role in cognition, memory, facial recognition and brain plasticity.

NMDA dysfunction may provide the interface between environmental neglect (including a lack of opportunity to mimic, learn and integrate appropriate social skills) and/or trauma in childhood, and poor brain plasticity of the hippocampus, amygdala, anterior cingulate and prefrontal cortex. LTP, which is a plasticity receptive condition of

the neural synaptic connections, is dependent on NMDA. NMDA is a receptor for glutamate, the primary excitatory neurotransmitter in the human brain. These receptors are differentially distributed in the brain according to developmental periods and are associated with synaptic pruning at critical periods. Overactivity of NMDA transmission may be implicated in the stress induced death or atrophy of hippocampal cells because of the high concentration of glutamate (Grosjean and Tsai, 2007). Dissociative states and psychotic symptoms such as magical thinking or reality lapses linked with BPD could also be a result of an NMDA system dysfunction (Grosjean and Tsai, 2007).

A genetic contribution to BPD may be the serotonin transporter gene, 5-HTT. Mood, thinking, motor functions, food intake, sleep rhythms and sexual activity are all modulated by the brainstem raphe nuclei serotonin (5HT) system, which releases serotonin to the rest of the brain. There is variability in the expression of the transporter gene. Differences in expression have been linked to anxiety, depression, aggression related personality traits, Bipolar Disorder, eating disorders, substance abuse, ADHD, ASD and OCD (Lesch and Gutknecht, 2005). Ni *et al.* (2006) connect 5-HTT with BPD and emotional dysregulation and impulsivity.

Ducci *et al.* (2008) found evidence to support variances in expression of the MAO-A gene in conferring vulnerability or resilience to antisocial personality disorder in populations of American Indian women who had experienced trauma in childhood and adolescence. This gene encodes the enzyme Monoamine Oxidise A, which breaks down neurotransmitters, such as serotonin, dopamine, norepinephrine and epinephrine (the last two being noradrenalin and adrenalin, respectively) in the brain.

The parietal cortex of those with BPD has significantly stronger leftward asymmetry when compared with those who are not affected. In the general population, the left parietal cortex is usually larger than the right. (The human brain has the evolutionary advantage of asymmetry; meaning that the two hemispheres of the brain work together, but on different tasks, or different parts of the same task, and are not functionally symmetrical.) Those with BPD who did not experience psychotic symptoms had a reduced right parietal cortex size (smaller than that of healthy control subjects) giving them a stronger leftward asymmetry, which may protect them against psychosis

(Irle *et al.*, 2004). Reduced leftward asymmetry of the parietal cortex is linked with the dysfunction of schizophrenia. A small subset of people with BPD who had more psychotic symptoms, and more schizoid personality traits, showed reduced size of the left parietal cortex (Irle *et al.*, 2004).

The corpus callosum, which links the two hemispheres of the brain, is reduced in people who suffer early trauma or neglect. This may also mean a compromised ability for the two hemispheres to work smoothly together (Grosjean and Tsai, 2007). Irle *et al.* (2004) speculate that a smaller right parietal cortex could be the result of a traumatic childhood deficient in healthy social relationships, leaving it under stimulated. The parietal cortex sits directly behind the frontal cortex, and both continue to mature into adulthood and so are likely to be responsive to environmental input with resultant brain plasticity (Irle *et al.*, 2004). BPD has shown itself to be amenable to long term psychotherapy involving the formation of new brain pathways for emotion, empathy and impulsivity, and symptoms can disappear over a period of several years, although it is likely that a vulnerability to stress will remain. Medication mediating NMDA can also be helpful in conjunction with talking therapy (Grosjean and Tsai, 2007).

What does this mean for counselling?

Personality disorders are positively responsive to long term talking therapies, where learning opportunities are provided to make and maintain new brain pathways for social interaction, empathy and emotional regulation. In order to take advantage of the brain's capacity for plasticity (and in particular, that of the prefrontal and parietal cortices, which continue to mature into adulthood) counselling for BPD should be provided within a stable, secure and clearly structured environment where healthy social interaction and emotional management can be modelled within the counsellor–client relationship. This not only lessens anxiety, but provides an alternative to chaotic, insecure environments that the client may have experienced in the past.

Long term continuity of counsellor is important, so that the client can begin to build pathways for trust and attachment, although it

is to be hoped that there will be other, preferred objects of primary attachment for the client, given that counsellors are only in clients' lives in a professional capacity. The counsellor must also set and model clear boundaries for the relationship, which will add to the structure and security present.

To get themselves into an optimum state for synaptic plasticity, clients should be encouraged to engage in regular cardio exercise, to address any sleep related issues so that they can sleep well and to partake of a healthy diet (see Chapter 6). These things support neurogenesis in the dentate gyrus of the hippocampus and the maintenance of neurons in general through raised levels of BDNF. Counsellors should work with clients to minimize or eliminate any environmental stressors, including social stressors, and a detailed plan of necessary steps to achieving this can be created. Stress is a clear causal factor in personality disorders, and while it is still present it is unlikely that there will be any improvement.

Counsellors should be aware of the high possibility of the co-morbidity of other dysfunctions with personality disorders. If other dysfunctions are present (such as OCD, anxiety, depression, PTSD, etc.) these may require additional and/or different ways of working, or different interventions. For instance, the counsellor might encourage the client to work with their GP or psychiatrist to check out possible complementary medications, and also consider whether any of the other potential interventions described in the relevant sections of this book (related to those dysfunctions) might be of benefit when used alongside strategies for BPD.

Once a successful, secure client–counsellor relationship has been established, client centred counselling can then take place and the client can identify their primary goals for change. This could take several forms, including wanting insight and understanding of the self through working on reframing past trauma, unravelling tangled thoughts and cognitions, learning to self-regulate emotions and to name feelings and identify their source, building and practising social skills for interacting with others, forming healthy attachments and/or managing impulsivity.

Because of confused cognition, it could be helpful for the counselling process to utilize white boards or written work on paper to identify beliefs in a non-verbal medium and locate blind spots, inconsistencies

and paradoxes within these beliefs. Written words and pictures provide a different sensory input medium for the re-processing of old information, allowing the brain to subvert well worn neural pathways for cognition related to thinking and talking, and to examine things in new ways.

New neural pathways – whether they are for thinking, feeling or behaviour – take time to become stable, default pathways, used preferentially above old, habitual ones. Practice and repetition are key to cementing new learning to the procedural memory so that it becomes embedded learning (see also the section on learning and attention in Chapter 3). Neural pathways involved in BPD have evolved over many years, including periods of critical early development, and will take many years to 'unlearn'. This suggests that time spent in counselling or other talking therapies will be lengthy in duration if there is to be real improvement. The capacity for learning and attention in those affected by BPD is already compromised, so that alone will take practice. New learning, including new thoughts and perspectives which comprise insight, must be reviewed again and again from session to session so that it can become habitual. For most BPD clients, the pace should be slow and repetitive or the new learning will be lost.

Specific tasks, such as practising empathy (the ability to understand and predict how others might feel by imagining the self in the shoes of another) can be part of the counselling session if congruent with the goals of the client. Empathy can be practised by using picture books or DVDs and having the client predict what the characters in the book or the movie are feeling and why, and through the identification of social cues (facial expression, body language, expression of emotion and actions) and environmental cues (the weather, the behaviour of other characters and situational constructs, such as a car crash or a lost dog). Clients can be encouraged to name the feelings they would have if they were in the same situation.

Other specific tasks might involve the strengthening of memory and attention (practising memory and concentration tasks) or facial identification. This sort of work is likely to build the capacity of the underdeveloped prefrontal regions of the brain as well as confer new skills to those with BPD. In order to make memory and attention tasks relevant, it should be possible to integrate them into the general counselling work, such as by having the client memorizing

their own goals or strategies to be recalled and written out again at the next session.

Because the pathology of personality disorders involves anxiety, hyper arousal of emotion and impulsive action, and because stress is a causal factor, those with BPD can benefit from learning and practising breathing and relaxation techniques, as well as mindfulness, because these help counteract those symptoms and triggers. Counsellors need not only to be able to describe the techniques, but to feel comfortable in facilitating clients to practise these until they are confident to try them alone. Disorganization of thought can lead to disorganization of action, so helping clients plan a routine to use breathing, muscle relaxation and mindfulness and to monitor their progress (on a calendar, or in a diary, etc.) is necessary in order for this to become a habit with long term benefits.

Intensely emotional personalities, such as those with BPD, often try to micromanage their own lives and those of others to provide the safety which was not present in their early lives. Counsellors can work with BPD clients to help them acknowledge this situation, celebrate it as a self-protective strategy, which has been intended to keep themselves safe, and identify which parts of their controlling behaviour are genuinely helpful and which parts are getting in the way of the life they would like to have. If there are some behaviours that they no longer find useful, the counsellor can support them to choose alternative behaviours, to make a plan for how to stop before acting, to use the new behaviour in preference to the old, and to maintain this behaviour consistently until it becomes a new pathway in the brain.

Psychosis/schizophrenia

What do we know?

Schizophrenia is regarded by many as the most disabling mental dysfunction (Ben-Shachar and Laifenfeld, 2004). As far back as the 1980s, the literature was suggesting a connection between psychosis (the perception of sensory input which doesn't exist in reality, e.g. hearing non-existent voices, seeing hallucinations, experiencing delusions,

including paranoid delusions, etc.) schizophrenia and a failure of brain plasticity (Haracz, 1985). Schizophrenia is characterized by psychosis, disorganized thought and speech, and cognitive impairment (Ben-Shachar and Laifenfeld, 2004; Hakak *et al.*, 2001). Episodic memory is often poor (Ben-Shachar and Laifenfeld, 2004). An impairment of brain plasticity is likely to compound itself as flawed interactions with the environment lead to the formation of dysfunctional brain pathways and circuitry, which can be construed as a 'miswiring' of the brain (Ben-Shachar and Laifenfeld, 2004).

Cellular difference in the brain has been observed in schizophrenics, including decreased size of neurons in the hippocampus and the dorsolateral prefrontal cortex (Ben-Shachar and Laifenfeld, 2004; Hakak *et al.*, 2001), increased density of neurons within a defined space, and distortions in the spatial orientation of neurons (Hakak *et al.*, 2001). Ben-Shachar and Laifenfeld (2004) and Stephan *et al.* (2006) note reduced size and synaptic connectivity of the dendritic spines of neuronal axons. The reduction in dendrite size and connectivity may be a reflection of reduced capacity for brain plasticity (Stephan *et al.*, 2006). EEG studies indicate a malfunctioning in hippocampal neuronal circuitry in those affected by schizophrenia and also in some of their unaffected relatives (Ben-Shachar and Laifenfeld, 2004).

Chronic schizophrenia is accompanied by changes in normal brain structure, such asymmetric reduction and ventricular enlargement (De Lisi, 1997). (Ventricles are cavities in the brain filled with cerebrospinal fluid.) Irle *et al.* (2004) record that size reductions in the left temporal and parietal cortices have been shown to be associated with psychotic symptoms, and that these were more pronounced in individuals with greater reductions in the size of these cortices. (The area of the left hemisphere of the brain occupied by the parietal cortex is usually larger than that on the right and this is called asymmetry.)

The cause of schizophrenia is thought to have a significant genetic contribution (Ben-Shachar and Laifenfeld, 2004; Hakak *et al.*, 2001). There may be a genetic underlying to the impairment of brain plasticity as well (Ben-Shachar and Laifenfeld, 2004). The expression levels of genes involved in myelination, development, synaptic plasticity, neurotransmission and signalling are altered in the dorsolateral prefrontal cortex of the postmortem brain tissue of those affected by schizophrenia (Hakak *et al.*, 2001).

The down-regulated genes connected with myelination are related to the formation of the myelin sheath by a type of glia cell called oligodendrocytes. Myelin is important for the transfer and speed of signals to axons (and therefore between neurons) and also for the general trophic health of axons. Oligodendrocytes promote neuronal maturation. Brain imaging studies have found subtle white matter (myelinated tissue) abnormalities in schizophrenic brains. Several differentially expressed genes in those affected by schizophrenia relate to the formation of the cytoskeleton, or scaffolding structure, of cells. Oligodendrocytes have been reported to regulate the formation of the cytoskeleton. Myelination of the prefrontal cortex happens in early adulthood, typically the age of onset for schizophrenia (Hakak *et al.*, 2001). It is also possible that environmental events and stimuli around this developmental period contribute to the development of schizophrenia (Ben-Shachar and Laifenfeld, 2004).

Genes which express GABA (y-aminobutyric acid) were found to be up-regulated in schizophrenic brains. Other neuropeptides with a broad range of functions were found to have altered expression levels as well. Genes allowing the neurotransmitter dopamine to bind to proteins in order to activate downstream signalling pathways are dysregulated in schizophrenics. Genes involved in neuronal development and plasticity are up-regulated compared to controls (Hakak *et al.*, 2001). In the study of Hakak *et al.* (2001) the medication or otherwise of the subjects did not appear to make any significant difference to gene expression.

There are also brain differences in neurotransmitter pathways and synaptic transmission in those affected by schizophrenia (Hakak *et al.*, 2001). Stephan *et al.* (2006) argue that dysconnectivity (a disorder of connectivity) between brain regions, observed in the brains of those affected by schizophrenia, could result from abnormal modulation of glutamate receptor *N*-methyl-D-aspartate (NMDA) dependent plasticity by other neurotransmitter systems. Ben-Shachar and Laifenfeld (2004) also link NMDA abnormalities to schizophrenia.

Functional imaging studies have shown reduced coupling between the frontal and temporal cortices, and electrophysiological testing/EEG, which measures brain waves, shows reduced functional connectivity between regions, including gamma band synchronicity. Artificially messing with the electrophysiological states of healthy

volunteers produces schizophrenia like symptoms of perceptual distortion (Stephan *et al.*, 2006). An NMDA blocker (ketamine; commonly used as an anaesthetic and sometimes as a recreational drug) is known to produce psychotic and dissociative symptoms in humans (Irle *et al.*, 2004). Low levels of NMDA have been observed in the prefrontal cortex of diagnosed schizophrenics (Ben-Shachar and Laifenfeld, 2004).

Functional coupling between neurons determines whether they survive synaptic pruning in critical development periods. An impairment in plasticity would affect the way regional connections are established in the developing brain. Genes identified with schizophrenia have been identified which also relate to glutamatergic synapses and NMDA receptor dependant signalling, affecting synaptic plasticity (Stephan *et al.*, 2006). It is unclear whether cellular dysconnectivity as a result of dysfunctional plasticity is causal in schizophrenia, or dysfunctional synaptic regulation by neurotransmitters (also a result of impaired plasticity), or both.

As discussed above, the neurotransmitters GABA and glutamate are associated with the condition of schizophrenia. Some studies have shown reduced glutamate concentrations in the prefrontal cortex of those affected and a reduction of glutamic acid in their cerebrospinal fluid (Ben-Shachar and Laifenfeld, 2004). Other candidates for neurotransmitter variations include changes in dopaminergic function (Ben-Shachar and Laifenfeld, 2004; Hakak *et al.*, 2001; Stephan *et al.*, 2006) and acetylcholine modulation (Stephan *et al.*, 2006). Dopamine is very important for reinforcement of new learning and for emotional learning, and acetylcholine is key to perceptual learning (Stephan *et al.*, 2006). Therapeutic drugs which act as dopamine receptors are effective antipsychotics, while dopamine blockers can produce psychotic symptoms similar to those observed in schizophrenia (Ben-Shachar and Laifenfeld, 2004).

Psychotic states, including the presence of delusion, hallucination and/or disordered speech or thought (known as 'positive symptoms' of schizophrenia) have been associated with hyper dopaminergic states (high concentrations of dopamine) in the mesolimbic regions (the dopamine pathway in the brain which begins in the ventral tegmental area in the midbrain and runs to the limbic system via the nucleus accumbens, the amygdala, the hippocampus and the medial

prefrontal cortex) and an absence of psychotic symptoms, but the presence of low, flat mood, the inability to experience emotion or pleasure, and/or a lack of motivation (known as the 'negative symptoms' of schizophrenia) with hypo dopaminergic states (low concentrations) in the mesocortical projections to the frontal cortex (Ben-Shachar and Laifenfeld, 2004). Dysfunction of the dopamine system in the mesolimbic areas of the brain (including metabolism, storage, release and uptake) has been found in first episode schizophrenic subjects. Dopamine has also been associated with the processes of brain plasticity, because it can both block and facilitate LTP in the amygdala and the hippocampus, including in the dentate gyrus of the hippocampus where neurogenesis takes place (Ben-Shachar and Laifenfeld, 2004).

Mitochondrial gene up-regulation has been observed in connection with schizophrenia (Ben-Shachar and Laifenfeld, 2004). (Mitochondria are parts of cells which are outside of the nucleus, but within the main body of the cell. They contain maternally transmitted DNA within them.) Mitochondria are less numerous, more likely to be deformed, and reduced in density in the brains of those with schizophrenia. Antipsychotic drugs tend to normalize mitochondrial density and volume (Ben-Shachar and Laifenfeld, 2004).

Converging evidence supports a dysfunction of mitochondria in schizophrenia, including mitochondrial hypoplasia (incomplete or arrested development of mitochondria) and a dysfunction of the oxidative phosphorylation system. (The addition of phosphate to organic compounds within the brain, mediated by oxygen.) In schizophrenia there is evidence of an altered oxidative stress state in the brain. Antipsychotic medication interacts with the mitochondrial oxidative phosphorylation system (Ben-Shachar and Laifenfeld, 2004). Monoamine oxidase, the enzyme responsible for the metabolism of dopamine, is located on the outer membrane of mitochondria (Ben-Shachar and Laifenfeld, 2004).

Mitochondria provide energy to neurons for all their essential processes by producing Adenosine Triphosphate (ATP) through oxidative phosphorylation. Mitochondrial dysfunction leads to alterations in ATP production (ATP is a nucleotide produced via cellular respiration in the mitochondria, and nucleotides form the building blocks of DNA) and cytoplasmic calcium concentrations

(cytoplasm describes the parts of the cell that exclude the outer membrane and the cell nucleus) as well as reactive oxygen species (chemically reactive oxygen molecules involved in cellular signalling processes, which can cause cell damage when they are present in high concentrations) and nitric oxide production. These effects have been shown to contribute to altered synaptic strength and plasticity. (Nitric oxide in the brain controls the supply of oxygen to mitochondria, stimulates the production of new mitochondria, and kills parasites, viruses and tumours by inactivating their mitochondria.) Neurons involved in nitric oxide signalling have a distorted distribution in schizophrenic brains (mainly an increase, in the hippocampus, neocortex, the myelinated white matter of the lateral temporal cortex and in the cerebellum; Ben-Shachar and Laifenfeld, 2004).

Mitochondria are also key in the processes of the generation of action potentials through intracellular calcium signalling (because they uptake and release calcium) which moderate neurotransmitter release, cytoskeletal dynamics and activity dependant regulation of gene expression (Ben-Shachar and Laifenfeld, 2004). (Action potentials are electrical charges sent from the neuron down the axon for the purposes of signalling other nearby neurons, and can be regulated by the amount of calcium in the cell body.)

Because mitochondria are a key factor in energy metabolism, this could contribute to findings of decreased energy metabolism in the frontal cortex (sometimes called hypofrontality) in schizophrenic subjects. This is thought to contribute to cognitive dysfunction. Other regions of the brain have also been found to have altered metabolic rates including the temporal cortex, the thalamus and the basal ganglia. Reduced levels of ATP have been observed in the prefrontal cortex and left temporal cortex (Ben-Shachar and Laifenfeld, 2004).

Mitochondrial dysfunction could either cause or arise from anomalies in brain plasticity. As the abnormalities in perception, emotion and cognition lead to further flawed interaction with the environment, abnormal synaptic connection and modelling would be reinforced (Ben-Shachar and Laifenfeld, 2004). Psychotic schizophrenic symptoms can wax and wane over the course of the lifetime. Pharmacological treatment is generally accepted as necessary for successful symptom management (Ben-Shachar and Laifenfeld, 2004; Port and Seybold, 1995).

What does this mean for counselling?

While people are experiencing a schizophrenic episode, and/or their condition is not successfully managed, there is little place for counselling because of thought and cognition distortion. Counselling deals with reality, even though there may be many perspectives on the 'truth'. Hallucinations, delusions (paranoid or otherwise) and internal voices (sometimes experienced as external) will make it too difficult for the client to separate the actuality of the counselling session from what is happening in their own head. (There is, however, a place for supportive key workers such as social workers to help those diagnosed to manage their lives in a reality which is different to what they are currently experiencing via their brains, and a place for psychiatry and pharmacological intervention. If clients present for counselling with untreated or potentially undiagnosed schizophrenia, it is crucial that they are supported to be connected to these types of support systems.) It is important to remember that many people do become well again and successfully control this condition with medication (including antipsychotics) at which point counselling can become appropriate.

There is a high genetic component in the development and progressive course of schizophrenia, which is unable to be affected by counselling. However, we now know that genes can be differently expressed in response to environmental triggers, which include physical and/or emotional stress levels, diet, sleep, hormonal function, the presence or absence of drugs (including medications and alcohol as well as recreational drugs), the presence or absence of inflammation, bacteria and/or viruses, and the physical environment – including day/night cycles, temperature, noise levels and pollutants among other things (see also the earlier section on gene transcription).

Optimum healthy behavioural choices lead to optimum environmental conditions which lead to optimum gene expression. During periods of wellness, it is possible that the counsellor sharing this information may influence and/or support the client to make such choices. These could include changing their diet, avoiding recreational drugs (including alcohol), changing work shifts to fit in with normal day/night cycles, getting into a good sleep routine, making lifestyle changes to eliminate or minimize stress (such as increasing pleasurable social contact, avoiding stressful people, or reducing work or study

hours), getting appropriate rest and relaxation (including using relaxation techniques such as breathing, mindfulness and/or yoga), while at the same time partaking of regular cardio exercise, seeing their GP to test for any hormonal imbalances and/or inflammation or viruses which might be present (and taking the necessary steps to address those if any are confirmed) and living in a healthy environment where possible. (It is of note that schizophrenia usually makes its first appearance in adolescence, a time of significant hormonal fluctuation.)

Healthy choices will also help to promote brain plasticity, the failure of which is believed to underlie and reinforce the structural and functional changes to the brain in the presence of schizophrenia, and to be connected to the transcription of the genes responsible. Healthy choices, however, are unlikely to be enough to successfully manage this most disabling of all brain impairments, and clients should be encouraged to maintain medications which are responsible for wellness.

There are often periods in the life of those affected by schizophrenia when symptoms are more intense than at others. Whether this is due to hormonal factors, stress or other environmental triggers, it is important that clients learn to recognize the early signs of onset, and to take quick, preventative action, such as minimizing any stress and connecting with their psychiatrist. Counsellors and clients can create plans for maintaining wellness, which might include asking people close to the client to look out for warning signs, such as increasing paranoia, delusional thinking or cognitive distortion. Healthy choices (as discussed above) can be a part of this plan.

One of the main focuses for counselling clients who have experienced schizophrenia is likely to be the piecing back together of lives which have been devastated by the course of this dysfunction. This can take many forms: focusing on the rebuilding, including the making of new life choices, and the construction of social support systems, or on repair or recovery of what has been lost or become disconnected, such as careers, relationships and/or self-image and identity. This is likely to need much discussion, and client centred goals should inform the counselling priorities for each session. Repetition of discussion within sessions (and continuous checking in, as sessions progress from one to the next) will help facilitate the laying down of consistent new neural pathways.

Trauma encountered in childhood or teenage years may need to be discussed if this is congruent with what the client wishes to talk about, and counselling techniques appropriate in the treatment of PTSD (see also that section earlier in this chapter) could be useful, as could reframing, and/or asking the client to look back and say some comforting words to their younger selves, or to notice the strengths of those younger selves. As with dissociation, it is my experience that some clients who have been affected by schizophrenia come to understand the voices they have heard (and in some cases continue to hear) as poorly integrated parts of their younger selves. (Others see the voices as spiritual, as disembodied abusers or as a representation of what others think of them.)

Cognitive distortion may still be present in neural pathways, which will only be repaired by conscious rewiring. As with personality disorders, using visual aids to map out and process beliefs and ideas so that they can be reprocessed and reordered through a different channel of sensory input could be helpful (white boards, paper, etc.). In this way, things that don't fit together (blind spots, paradoxes, mismatches, etc.) are more easily identified, because the pieces of the puzzle have to fit together in the same place to make one picture, so to speak. A clear plan for identifying moments of choice and making those choices consciously in order to achieve set goals for life (instead of following automatic brain pathways set down in habitual procedural memory) is something that could also be co-constructed in counselling.

Recording goals and action plans so that the client has a copy to counteract memory impairment or thought disorganization that might be experienced once they leave counselling, which would be a barrier to them following through with what they have chosen to do, might be helpful. The client may wish to record these themselves during the session. Some clients might also wish to practise cognitive logic tasks or tasks which would strengthen memory.

Finally, people who have experienced psychosis have seen the world in perceptual ways that most people can't. The offshoot can be heightened creativity and less rigid barriers to creative perception. Creative therapies that use music, writing, drama and/or art (such as painting, sculpture, collage, mask making, drawing, etc.) can help clients to express feelings, thoughts and past experiences which might not find equal expression in words, and can be appropriate for use in

counselling if there is a good match of these techniques with the client and they give permission for this.

Eating disorders

What do we know?

Note: This section is intended to discuss contexts where behaviours concerning eating are considered dysfunctional, and may not apply to every person who is considered underweight or overweight, for which there may be other reasons, unrelated to eating behaviours.

There are several types of eating disorder with obesity and overeating at one end of the scale, and self-starvation, known as anorexia nervosa, at the other. Humans differ from animals in that eating has ceased to become merely a survival tool and a source of physical and neural energy, and has become an activity surrounded by social and cultural rituals, including the added dimension of how food intake contributes to physical appearance and body image. These dimensions can override the biological signals relating to metabolic need or to the palatability of food (Alonso-Alonso and Pascual-Leone, 2007). The majority of those affected by restrictive type eating disorders are female although males can also be affected (Sodersten, Bergh and Zandian, 2006). Obesity has become increasingly common in societies without a restricted supply of food and affects both men and women (Berridge *et al.*, 2010).

The brain has a highly developed system for biological reward for eating food, increasing the likelihood of continued survival. This system includes subcortical forebrain structures such as the ventral pallidum, the nucleus accumbens and the amygdala, as well as regions in the neocortex such as the orbitofrontal, the anterior cingulate and the anterior insulate cortices. Opioids (a neuropeptide) and endocannabinoids (naturally occurring brain chemicals similar to the active component of marijuana) increase feelings of reward in response to certain food cues, some of them culturally learned (Berridge *et al.*, 2010). Changes in dopamine receptor binding in obese subjects,

providing extra pleasurable reward for food intake, are likely to be a consequence rather than a cause of obesity (Berridge *et al.*, 2010).

The prefrontal cortex appears to be a critical area involved in the cognitive control of food intake. This may be because as part of the higher executive system of the brain, it can play a role in impulse control and suppression (Alonso-Alonso and Pascual-Leone, 2007). Damage to the right frontal lobe or disconnection of the frontal lobe from the rest of the brain can cause a passion for eating, a preference for fine food and unsurprisingly, weight gain. Conversely, hyperactivity of the right prefrontal cortex can lead to anorexia like symptoms, which in patients with right prefrontal epilepsy has been shown to cease after anticonvulsant therapy. As well as overeating, reduced function of the right prefrontal cortex is associated with inactivity, while its excessive activation is associated with greater physical activity as well as loss of appetite (Alonso-Alonso and Pascual-Leone, 2007).

Poor activation of the right prefrontal cortex is linked to impaired decision making and consequential planning, which may contribute to the inability of many obese clients to consider the consequences of what and how they eat in the present, and also the inability to commit to weight loss plans or action to regain an optimal physical state. Obese people have been found to have a lower treatment adherence and compliance in other areas of bodily health, such as compliance with breast screening programmes, despite a higher incidence of breast cancer among obese women (Alonso-Alonso and Pascual-Leone, 2007). A right prefrontal cortex dysregulation could also impact on the failure to use this executive planning centre of the brain to evaluate the consequences of choosing healthy versus unhealthy food (Alonso-Alonso and Pascual-Leone, 2007).

The prefrontal cortex is also an important region in the neural circuits involved in body mapping, or self-recognition, preferentially in the right hemisphere of the brain, alongside the prefrontal parietal and prefrontal temporo-limbic regions. Atrophy in the right frontal lobe of patients with dementia can cause changes in both body image and food preferences (Alonso-Alonso and Pascual-Leone, 2007). People who manage successful weight loss tend to have a higher activation of the right dorsolateral prefrontal cortex and to be more aware of how their bodies look. Lack of embarrassment about increased body

size has also been associated with decreased activity of the prefrontal cortex (Alonso-Alonso and Pascual-Leone, 2007).

It is possible that disruption of the right prefrontal cortex is sufficient to cause a switch in internal energy balance (energy homeostasis) allowing a general increase in body weight in affluent societies (Alonso-Alonso and Pascual-Leone, 2007). It is unknown exactly what might cause right prefrontal cortex dysfunction leading to atrophy and decreased activity in this region, but genetic factors and environmental stress through the hypothalamic–pituitary–adrenal axis (HPA axis) are likely candidates (Alonso-Alonso and Pascual-Leone, 2007; see also the section on stress in Chapter 4).

Leptin replacement in adults who are genetically deficient in this hormone has been shown to increase the grey matter density in areas of the brain including the prefrontal cortex (Alonso-Alonso and Pascual-Leone, 2007). Leptin appears to control satiety (feelings of fullness after eating) by signalling peptide synthesizing neurons in the medial-basal hypothalamus from the peripheral fat stores in the body (Connan *et al.*, 2003; Sodersten *et al.*, 2006). Peptides are also released from the duodenum (the upper part of the small intestine) to produce a feeling of being full. When fat stores are depleted, as they are during periods of starvation, leptin levels in the blood go down (Sodersten *et al.*, 2006). Insulin carried in the glucose metabolism in the brain may also specifically target the prefrontal cortex, enhancing its metabolic rate (Alonso-Alonso and Pascual-Leone, 2007).

Other neural systems shown to play a role in the modulation of appetite include neuropeptides, such as corticotrophin-releasing hormone, opioids, Neuropeptide Y (discussed below), peptide YY, vasopressin, oxytocin, cholecystokinin and gherlin (leptin is also a neuropeptide) and monoamines (compounds which combine to form neurotransmitters), such as 5-HT (serotonin), dopamine and norepinephrine (Hashimoto *et al.*, 2005). Heinrichs and Koob (2004), link corticotrophin-releasing factor/CRF to the metabolic process of energy homeostasis, which regulates the need for food intake. Appetite is diminished by the administration of corticotrophin-releasing factor receptor antagonists, or by environmental conditions which elevate corticotrophin-releasing factor levels, such as stress or appetite suppressing drugs (Heinrichs and Koob, 2004). There is some confusion around whether corticotrophin-releasing factors/CRF

promote comfort eating or suppress appetite in response to stress, or provoke different responses in different subjects. Appetite suppression tends to be supported by the majority of studies (Berridge *et al.*, 2010).

Connan *et al.* (2003) concur with the model of Heinrichs and Koob (2004), believing that genetic factors and early life experience (including in the womb) interact to produce a susceptibility to a submissive type of stress response, and to HPA axis dysregulation (see also the section on stress in Chapter 4) so that there is consistently raised corticotrophin-releasing factor/CRF activity, which leads to a persistent loss of metabolic homeostasis and lack of appetite. The HPA axis is modified by early maternal behaviour and nurturing behaviours. The monoamine 5-HT is also influenced by the attachment experience.

By adolescence, rats deprived of maternal nurturing weigh less than other rats, suggesting that early functional alterations in the HPA axis and 5-HT may impact on appetite regulation in later life. Lack of maternal behaviour may also contribute to the reduced feeding of an infant, which can affect neural pathways for appetite and expectancy of nutrition (Connan *et al.*, 2003). Connan *et al.* (2003) also suggest a connection between anorexia nervosa and impaired empathy and the ability to self-reflect, which requires the development of mirror neurons, and the ability to copy others and to have one's own actions reflected back to themselves. Reduced maternal nurturing at an early age could be the reason for this impairment.

Sodersten *et al.* (2006) provide evidence that anorexia nervosa can be caused by the voluntary withholding of food by healthy subjects and that this causes the body to go into a state where food is no longer desired, and actively avoided, because the brain's system of neural reward begins to reward and sustain the state of starvation. Once there is a reduction in food intake an increase in physical activity begins, which may be related to the stimulation of dopamine transmission in the striatum (Sodersten *et al.*, 2006) or to the hyper-activation of the prefrontal cortex (Alonso-Alonso and Pascual-Leone, 2007). Lowering of body temperature and slowing of heart rate occur as anorexia develops (Sodersten *et al.*, 2006).

Depression, anxiety and obsessive compulsive behaviours often accompany the dysfunction of anorexia, but these co-morbidities (simultaneously occurring conditions) appear to Sodersten *et al.* (2006) to be a result rather than a cause of anorexia. Sodersten *et al.*

(2006) consequently view anorexia (and bulimia nervosa, which is characterized by binge eating and purging through self-induced vomiting) as separate, discrete dysfunctions of the brain, brought about by the unavailability of food to the digestive system, rather than a symptom of other dysfunctions, such as anxiety.

Sodersten *et al.* (2006) experimented with the differences between men and women after voluntarily withholding food and found that men ate more after a short period of starvation whereas women ate less than they had previously, suggesting that women have more trouble compensating for periods of starvation, making dieting particularly dangerous for women. Neuropeptide Y is increased during periods of starvation as levels of leptin decreased. Recent evidence suggests that Neuropeptide Y can facilitate hoarding, including the collecting and storing of food common in anorexic nervosa sufferers (Sodersten *et al.*, 2006).

Sodersten *et al.* (2006) have developed a treatment involving teaching patients to eat and feel full again, which involves a system of biofeedback so that patients can monitor themselves. Co-morbid conditions, such as depression, anxiety and compulsion, and physiological symptoms such as the levels of leptin and Neuropeptide Y are reversed when the patients eat more food and gain more body weight. This response to changing environmental conditions in the availability of food to its biological systems demonstrates the plasticity of the brain and its neural connections (Sodersten *et al.*, 2006).

Nakazato *et al.* (2008) found that recovered anorexic patients still had symptoms of anxiety and OCD, but this remained correlated with a low body weight. This could be related to the data put forward by Connan *et al.* (2003) in which those people deprived of warmth and affection typically have a lower body mass index. Strober *et al.* (2007) conducted a large scale study which they believe shows a heritable correlation between family members who have anxiety and/or OCD and those who have anorexia nervosa, as differentiated by control subjects who have never had an eating disorder and their family members. Bulik *et al.* (2007) and Connan *et al.* (2003) also point to studies showing genetic heritability to contribute to the risk of developing anorexia nervosa. Bulik *et al.* (2007) and Strober *et al.* (2007) regard anxiety as a precursor to anorexia. These findings are in conflict with the findings of Sodersten *et al.* (2006).

Many studies have found a link between BDNF and eating disorders (Gray *et al.*, 2006; Hashimoto *et al.*, 2005; Lipsky and Marini, 2007; Nakazato *et al.*, 2008; Ribases *et al.*, 2003; Ribases *et al.*, 2005). The synthesis and release of BDNF is targeted by glutamate receptors (Lipsky and Marini, 2007). BDNF levels are associated with levels of food intake. BDNF disruption is associated with obesity (Gray *et al.*, 2006). Low blood serum concentrates of BDNF have been found in those with anorexia nervosa and bulimia nervosa. Low body mass index, or body weight, was positively correlated with low levels of BDNF serum (Nakazato *et al.*, 2008). Monteleone *et al.* (2005) found that blood serum levels of BDNF were significantly reduced in women with anorexia nervosa and in underweight women with bulimia nervosa, but not in overweight women with binge eating disorders similar to bulimia. This also suggests that levels of BDNF are correlated with body weight. The authors conclude that BDNF reduction may represent an adaptive change to low calorie intake.

Expression of BDNF in the hypothalamus is nutritionally regulated and appears to be further regulated by the hormone leptin, discussed earlier in this section. Because BDNF modulates synaptic plasticity, it is probable that it is involved in the plasticity of the hypothalamic feeding circuits but also possible that it affects the energy balance of the hypothalamus (Gray *et al.*, 2006; Lipsky and Marini, 2007; Ribases *et al.*, 2003). A variation in the BDNF gene, and its precursor protein, Valmet66, is thought to change the regulated secretion of the mature BDNF protein and increase vulnerability to anorexia nervosa (Hashimoto *et al.*, 2005; Lipsky and Marini, 2007; Ribases *et al.*, 2003; Ribases *et al.*, 2005).

Lipsky and Marini (2007) associate this gene with harm avoidance tendencies and dysfunctions, such as depression and anxiety, which are often co-morbid with anorexia. Reduced serotonin function is also thought to be responsible for the correlation with anorexia and dysfunctions, such as anxiety (Connan *et al.*, 2003). Connan *et al.* (2003) cite evidence of normalizing serotonin function in those recovering from anorexia nervosa who regain their body weight, but also contrary evidence of persistence of serotonin dysfunction in some subjects.

Weight gain during puberty due to a rise of circulating leptin levels, in order to reset the hypothalamus to maintain weight at a new

level, may provide a window of opportunity for anorexia nervosa in terms of vulnerability in the systems regulating weight and appetite. The rise in oestrogen around this time may also contribute to altered 5-HT and corticotrophin-releasing functions/CRF (Connan *et al.*, 2003). Women recovering from anorexia nervosa have a lower volume of neural grey matter and smaller hippocampal and amygdala volumes than healthy subjects. This may be due to insult to the brain during development caused by low calorie intake. It may also constitute a pre-existing vulnerability to anorexia nervosa (Connan *et al.*, 2003).

What does this mean for counselling?

There are few clues in the available neuroscience pertaining to eating disorders which will be helpful to counsellors. But at the least, counsellors can make clients aware of the risks of beginning stringent low calorie diets, particularly for those with a familial history of anxiety and those who have experienced anxiety and/or depression themselves, or who were parented in a style of non-secure attachment. Women are especially vulnerable. Clients who plan to diet often discuss this in the context of discussing thoughts and feelings about themselves, and these can be linked to low self-esteem, dissatisfaction at life or self-hatred. It is clear that after a relatively short period of time withholding food becomes a pattern of learned neural pathways, which trigger dysfunctions in the brain, such as depression, anxiety and/or OCD. If a client has been feeling bad about themselves, dieting is hardly likely to bring improvement.

It appears that the main cure for anorexia nervosa is eating and regaining body weight, which is only helpful if a client is motivated to do so, and which is unlikely for those in its grip. The role of a counsellor here must be to help clients to locate their motivation. Future visualizations are helpful where the client builds a possible future, one detail at a time, while in a relaxed state. The counsellor asks them to choose a country, then a landscape, then a dwelling and then to add detail to the dwelling before decorating each room with things they could care about and finally adding people and/or animals (if required) before adding themselves and visualizing what they might be doing there.

The ability to see random possible futures helps to build or rebuild neural pathways for motivation. Just asking clients about the future that they see ahead for themselves from the point where they are at can seem a hopeless task to them because they are often caught up in the symptoms of anxiety, depression and compulsion, and the future they envisage from their current place in life is unlikely to be one worth finding the motivation to get well for.

It is essential for counsellors to make specialist referrals and not work alone outside the limits of their competence when they become aware that a client has an eating disorder. If the most important thing for that client is to regain body weight, an eating disorder clinic or hospital where eating is compulsory may be the most appropriate place for the client to become well. Depending on the age of the client (parents can compel children and adolescents) clients are entitled to refuse this help. If they do so, the counsellor must ethically point out to them the risks of doing so: suicide, death by starvation and other associated health risks, such as heart failure. In comparison with being taught how to eat again, talking therapies alone have a low long term success rate for those who experience anorexia nervosa (Sodersten *et al.*, 2006). If the client insists on working with the counsellor without other support, it would be advisable to get specialist supervision pertaining to this, because anorexia nervosa has a significantly higher fatality rate than other psychiatric disorders (Bulik *et al.*, 2007; Sodersten *et al.*, 2006).

This doesn't mean that the counsellor has nothing to contribute. Building up a relationship of trust (while remembering that sometimes it is necessary to break confidentiality to ensure the safety of clients; something which should always be made clear in an initial counselling contract) can help clients who may have experienced anxious, insecure or indifferent attachment styles with their parents or caregivers to start to build neural pathways for a more healthy, warm style of connection. Naturally, the counsellor must not become a long term fixture in the client's life, so they need to support the client to build up other relationships of trust with people who can become a part of their natural and more permanent support networks. This can be done with the use of geneograms and maps of the communities of people in their life to help identify suitable people.

It may take some time for counsellors to become aware of anorexia or bulimia in a client's life. It is a dysfunction which thrives on secrecy and can become a part of a client's identity. Once unmasked, it is helpful for counsellors to externalize the eating disorder, saying 'you may be affected by anorexia', rather that 'I think you're anorexic', so that clients are able to see the eating disorder as separate from their life and choose whether or not to work against it. They are also able to blame it for the negative side effects it produces (being cold, feeling tired, poor concentration) without blaming themselves. This allows for greater personal resilience in the fight against the eating disorder.

For those who find overeating or obesity to be a problem in their lives, counsellors can support them to develop a programme towards wellness, and to gather a network of people around them who can encourage them to stick with it. CBT may be helpful here, to address underlying causes for the eating (thoughts and feelings) and to help clients practise a different way of behaving in response. Once again, the brain can create new pathways and let old ones grow over, but this takes repetitive practice.

The extra dopamine reward system developed by obese clients will create cravings that seem like compulsions, and although these will subside with new learning, they will make it hard to put into practice. Positive feedback should be given for effort as well as achievement. Counsellors and clients can work together to create new reward systems for the clients. These could include trips away, buying themselves non food rewards, a hot bath or soak, a massage or even pleasurable exercise.

Finally, counsellors should support all those who are affected by eating disorders to find ways to eliminate stress from their lives, or if this is not possible, to minimize it. Clients can benefit from being taught techniques for coping better with stress (such as talking to support people, keeping a diary, sorting things through with their boss, moderate exercise, etc.) and relaxation techniques (such as deep breathing, mindfulness, yoga and meditation). (See the sections on stress in Chapter 4 and exercise, mindfulness, yoga and meditation in Chapter 6.)

Chapter 6

What Can We Recommend?

Being able to recommend strategies that the client can use and monitor themselves is a way of placing power, and the ability to manage their own lives, back into the client's hands.

These strategies can support or provide long term solutions for the client's mental well being.

Exercise

What do we know?

Exercise not only makes the body fitter, stronger and more resilient, it literally changes the structure of the brain and makes the brain fitter, stronger and more resilient. The beneficial outcomes of exercise act directly on the brain and not just on physical health. Most importantly, physical exercise increases levels of brain derived neurotrophic factor (BDNF) and its receptor, which is vital for maintaining optimum conditions in the brain for plasticity of synapses and the ability to make changes to the formation of existing neural pathways (Cotman *et al.*, 2007; Gomez-Pinilla *et al.*, 2002; Trejo *et al.*, 2001). Exercise not only increases BDNF in the sensory-motor systems in the brain, which control movement, but also in the hippocampus, which is associated with higher cognitive function, including the 'executive' functions of attention, learning, memory, planning, emotional regulation and impulse control. Exercise also increases BDNF in the lumbar spinal

cord, the cerebellum and the cortex. These effects are noticeable within days of exercising (Cotman and Berchtold, 2002).

Physical exercise aids recovery after injury or trauma to the brain, and induces neurogenesis (the birth of new neurons) in the central nervous system (Gomez-Pinilla *et al.*, 2002; Trejo *et al.*, 2001). Exercise not only stimulates the growth of new neurons – specifically in the dentate gyrus, which is the progenitor cell layer of the hippocampus – the increase in levels of BDNF promotes the survival of those new neurons (Cotman and Berchtold, 2002). BDNF delivered directly to an injured spinal cord can promote regenerative growth and probably performs a similar role in the brain (Gomez-Pinilla *et al.*, 2002). It also has the ability to improve learning and performance because of its contribution to plasticity (Cotman and Berchtold, 2002). Learning is characterized by the formation of new neural pathways. Learning increases BDNF gene expression and BDNF, in turn, facilitates learning (Cotman and Berchtold, 2002).

Exercise also structurally changes the brain by increasing the length and complexity of the dendrites, or axonal branches (Cotman *et al.*, 2007). Exercise reduces the threshold of theta wave (a measurable electrical brainwave frequency) stimulation needed in a long term potential (LTP) laboratory setting, for which causes new neural pathways to be laid down. This indicates that exercise is producing optimal conditions for plasticity to occur (Cotman *et al.*, 2007).

Exercise has been shown to regulate the expression of many genes, including those associated with immune function, metabolism, anti-ageing, protein processing and the process of gene transcription itself (Cotman and Berchtold, 2002). Exercise increases the production of CREB which is necessary for the process of gene transcription. CREB is also regulated by BDNF, strengthening further the relationship between gene transcription and exercise. CREB is required for memory processes and building neuronal resilience to injury (Gomez-Pinilla *et al.*, 2002). Exercise mobilizes gene transcription profiles that will positively affect brain plasticity (Cotman and Berchtold, 2002).

Exercise regulates a number of complementary chemical processes in the brain that work together with BDNF. Exercise also increases levels of GAP-43 (Growth Associated Protein 43) which has a role in axonal growth and neurotransmitter releases, as well as learning and memory. Synapsin 1 is another growth associated protein which

also affects axonal growth and neurotransmitter release, as well as plasticity, through the maintenance of synaptic contacts. BDNF regulates the synthesis of Synapsin 1 (Cotman and Berchtold, 2002). Exercise increases levels of other growth factors, and also the levels of synaptic proteins and glutamate receptors. IGF-1 gene expression (Insulin Growth Factor 1) is increased through exercise. IGF-1 is necessary to neurogenesis and is linked with improved memory. IGF-1 works in concert with BDNF to promote plasticity in the brain (Cotman *et al.*, 2007).

Physical exercise can protect against general cognitive decline in elderly populations (Gomez-Pinilla *et al.*, 2002). Exercise is protective against brain dysfunction usually associated with ageing, such as Alzheimer's disease and dementia (Cotman and Berchtold, 2002). It also enhances learning and memory, and other 'executive' learning functions in older people (Cotman *et al.*, 2007).

Exercise is beneficial in the alleviation of depression in both the young and the old (Chaouloff, 1989; Cotman *et al.*, 2007). The benefits are similar to those achieved with antidepressants. It also helps combat depression in people with neuro-degenerative diseases. In the range below excessive exercise, more exercise is associated with greater antidepressant effects (Cotman *et al.*, 2007). There is promising evidence that exercise is protective against depression developing. Low levels of BDNF are also associated with susceptibility to depression and other mood disorders (Cotman *et al.*, 2007).

Inflammation, which interferes with growth factor signalling, is reduced by exercise. Diabetes, hypertension (high blood pressure) and cardiovascular disease are symptoms of poor physical health, but are also correlated with brain dysfunction and neuro-degeneration. Hypertension and glucose intolerance are risk factors for cognitive decline (Cotman *et al.*, 2007). Exercise not only aids efficient brain metabolism, or energy processing, it also supports vascular function. It improves blood flow in the brain (Cotman *et al.*, 2007).

To support exercise induced changes in the brain, such as enhanced plasticity and neurogenesis, the brain responds to its own increased need for nutrients and energy through greater expression of enzymes involved in glucose use and metabolism both in the hippocampus and other brain regions (Cotman *et al.*, 2007). Exercise leads to widespread growth of blood vessels in the hippocampus, the cortex and the

cerebellum, and these blood vessels provide the increased nutrient and energy supply. Increased blood flow ensures that the enhanced brain function stimulated by exercise can be supported and maintained. Twelve weeks of exercise increases blood flow in the dentate gyrus (the layer of the hippocampus where new neurons are made) and this is correlated with improved learning outcomes on hippocampus related tasks (Cotman *et al.*, 2007).

Prolonged exposure to stress (see also the section on stress in Chapter 4) releases corticosteroid stress hormones, which causes the degeneration of neurons, particularly in the hippocampus. Under acute or chronic stress, dendrites of neurons start to thin and suffer spine reduction, which affects synaptic plasticity (Cotman and Berchtold, 2002). In animals, voluntary exercise before a stressful event leads to improved behavioural measures for coping with stress and prevents the genetic down-regulation of hippocampal BDNF (Cotman and Berchtold, 2002).

Female rats that were low on estrogen were less active and were unable to receive the benefits of exercise induced BDNF availability, which may have implications for women with low estrogen levels (Cotman and Berchtold, 2002). The highest neural benefits from exercise are typically associated with moderate, long term exercise (Cotman *et al.*, 2007).

What does this mean for counselling?

Counsellors cannot go out and exercise on their clients' behalf but we can recommend exercise as a hugely beneficial tool for clients to use, whether it be to raise positive mood, to fight against depression or other mood disorders, such as anxiety, to reduce the effects of stress, to strengthen resilience in the brain and increase its structural health, to improve general learning and memory, or to improve physical health, reducing risk factors for future cognitive decline in the process.

Beginning exercise may be difficult for clients with low mood, or even for those with other constraints, such as a lack of time or young children. Then the role of the counsellor would be to help the client to develop a plan to overcome these obstacles, possibly incorporating support from people within their natural social and familial networks

or from other social services professionals. Counsellors can also help a client to self-identify what sort of exercise they would find most suitable. Someone who is naturally sociable or who has a goal of making new friends might prefer a team sport or to exercise with a neighbour. A client with Aspergers will probably prefer an individual sport with as much seclusion from other people as possible to reduce social anxiety.

Naturally, this is only suitable when clients would like to exercise but can't manage to make it happen. The counsellor's role is to provide the information on how and why exercise can help so powerfully, and the client's role is to make the choice as to whether exercise will be part of their plan for healing or making change. Exercise is particularly valuable as an alternative self-therapy for clients who have been recommended psychiatric medication for mood disorders and who do not choose to take up the option of medication.

Exercise enhances a client's counselling experience by preparing the brain for new learning and through promoting and maintaining cognitive change by providing optimum conditions for brain plasticity and conscious rewiring of neural pathways by the client.

Some clients report previous addiction to exercise, particularly those who have been affected by eating disorders, although this is not restricted to those clients. Exercise can deliver pleasurable chemical endorphins to the brain's reward system, so counsellors need to enquire if this has been a problem in the past and give information or help develop a support plan to stop it from becoming a problem in the future if the client discloses previous experience of this. It would be helpful to discuss this risk with all clients who intend to start a programme of exercise so that clients are able to self-monitor for this and take action to reduce their exercise to safe levels before the behaviour gets addictive. Some forms of exercise may be less addictive than others, for instance, brisk walking may be less addictive than running.

Many, although not all, opportunities to exercise are often free or low cost meaning that exercise – an incredibly effective strategy for maintaining the plasticity of the brain – is available to all.

Sleep

What do we know?

Good sleep is essential for good brain function. People perform significantly more poorly on neuro psychological tasks after sleep deprivation indicating that sleep particularly serves the frontal cortex region of the brain, which is associated with attention, learning, memory, planning, response inhibition and the manipulation of information for the solving of problems (Hobson and Pace-Schott, 2002). Frontal cortex deficits are characteristic of sleep disorders, such as sleep apnoea (Hobson and Pace-Schott, 2002). The frontal cortex is the last part of the brain to wake and is probably the first part to fall asleep. It is thought that it is the part of the brain most dependant on sleep (Hobson and Pace-Schott, 2002).

New brain imaging technology has allowed greater insight into what the brain does when we sleep. It indicates greater slow wave activity in the brain during the first non rapid eye movement (NREM) sleep of the night (Hobson and Pace-Schott, 2002). It is possible that the slower brain waves provide the natural equivalent of artificially induced LTP, which is necessary to achieve long lasting plastic change in the brain. The mechanisms for achieving LTP in the brain are not yet properly understood. Different brain wave frequencies during sleep, as opposed to wakefulness, have been shown to produce long lasting changes in neural responsiveness, and as such are a candidate for the mechanism for LTP (Hobson and Pace-Schott, 2002).

Blood flow and global cerebral energy decreases during NREM, but during rapid eye movement (REM) sleep, blood flow and cerebral energy are equal to, or greater than, waking blood flow and energy metabolism, indicating that the brain is very active during REM sleep (Hobson and Pace-Schott, 2002). Restoration of normal REM sleep glucose metabolism levels to the levels measurable during waking is an indicator of recovery from depression (Hobson and Pace-Schott, 2002).

During REM, activation of the limbic and paralimbic regions is increased. These are associated with emotion, instinct and memory, and the integration of autonomic functions with conscious awareness. The limbic and paralimbic regions encompass the amygdala, the hippocampus, the hypothalamus, the basal forebrain, and the parahippocampal, entorhinal, insular, caudal medial orbitofrontal

and anterior cingulate cortices. Selective activation of the limbic and paralimbic regions during REM sleep supports the theory that this type of sleep integrates instinct and emotions with learning, memory and cognition, through the mechanism of dreaming (Hobson and Pace-Schott, 2002).

We also dream during waking (day dreaming) and during NREM sleep, but to a much lesser extent than during REM sleep. Waking suppresses hallucination/dreaming in favour of thought, and conversely, REM sleep suppresses thought (cognition) in favour of hallucination. Most dreaming is characterized by a lack of analytical thought and most people have little voluntary control of their dreams. There is a shift in brain state during REM and the process of dreaming because different regions of the brain are switched on or off compared to their wakeful state (Hobson and Pace-Schott, 2002).

Although it is not yet proven, there is a growing body of evidence that sleep contributes to brain plasticity. One theory is that the hippocampus and the neo-cortex interact during sleep to consolidate information required during the previous period of wakefulness. It is also suggested that REM sleep enhances cortical plasticity in procedural memory and in the prefrontal cortex, but not in episodic memory (Hobson and Pace-Schott, 2002).

In the hippocampus of rats, sleep produces a replay of neuronal firing patterns from the previous period of wakefulness during both REM and NREM sleep (Hobson and Pace-Schott, 2002). Repeated use of neuronal pathways consolidates and strengthens those pathways (Doidge, 2007; Hobson and Pace-Schott, 2002; Kandel, 2000). After a particular pathway/memory trace has been developed in the rat brain, its brain rhythms appear to switch from conditions favouring LTP, enabling neural plasticity and adaptation, to conditions favouring LTD which stabilize and temporarily close the brain to change, and in doing so consolidate the most recent change. Birds have also shown a similar pattern of neuronal activity, where the firing that maps the patterns of their songs from the previous waking is replayed while sleeping (Hobson and Pace-Schott, 2002).

Both NREM and REM states probably work together to consolidate our learning into memory. While awake, the brain is geared to capture sensory information from the environment. When this process is switched off during sleep, the brain can consolidate the learning that

occurred during waking as lasting change in the brain in the form of neuronal connections and the formation of neural pathways (Hobson and Pace-Schott, 2002).

When we sleep and how long for is regulated by a part of our brain known as the 'circadian clock' (Hobson and Pace-Schott, 2002; Reppert and Weaver, 2001; van der Horst *et al.*, 1999). Circadian rhythms are the external expression of an internal mechanism in the brain (located in suprachiasmatic nuclei of the anterior hypothalamus) that measures daily time. Several genes, widely expressed throughout the body, have been linked the circadian clock. Two of them are regulated by planetary dark-light cycles. Circadian clocks are also cued by other environmental cues but the dark–light cycle is the strongest cue (Reppert and Weaver, 2001; van der Horst *et al.*, 1999). The part of the brain that contains the circadian clock is located directly above the optic chiasm, perfectly positioned to take advantage of sensory information about the presence or absence of light. The intracellular molecular clock consists of interacting positive and negative gene transcription feedback loops. As the genes are expressed in alternate ways according to the sensory input, the clock responds by changing waking and sleeping times and the length of the sleep period (Reppert and Weaver, 2001).

In people without the use of their vision, circadian rhythms are set by the sensory input of light on the skin. Melatonin, the pineal hormone, can influence circadian rhythms as well (Reppert and Weaver, 2001). Signalling pathways activated by glutamate receptors are affected in different ways depending on the circadian time at which the stimulus is applied. The effect of the alterations in circadian gene expression is an alteration in the protein levels and the types of proteins produced by cells (Reppert and Weaver, 2001). The seasons of the year, which have different amounts of daylight in twenty-four hour periods, and whether it is day or night, therefore affect the way in which the brain functions.

The most likely way in which the circadian clock regulates behaviour is by the proteins from the clock controlled genes, which differ from the genes that make up the actual clock. The clock controlled genes are also rhythmically regulated by the core feedback loops, but their proteins are not essential to the function of the clock (Reppert and Weaver, 2001).

Disorders of the circadian clock system include jetlag (i.e. suddenly changing time zones and dark–light cycles, which usually change subtly from day to day) and sleep disorders. These types of sleep disorders often occur when people act against the priming of their circadian clock, for example, by doing shift work or partying late into the night and sleeping all day. Disorders of the circadian clock may contribute to neuropsychiatric disorders (Reppert and Weaver, 2001).

Homeostatic sleep drive is when the brain tries to balance up the need for sleep, in other words, the longer you've been awake, the sleepier you feel. People who need less sleep are more resistant to homeostatic sleep pressure (Hobson and Pace-Schott, 2002).

What does this mean for counselling?

It is probable that many undiagnosed cases of sleep apnoea are being treated as ADD or ADHD given that the symptoms mimic each other: a dysfunction of the executive prefrontal cortex functions, including attention, learning, memory and impulse control. When a client presents with these symptoms, it would be wise for a counsellor to check in with that client regarding the quality of their sleep, how they feel upon waking and whether there might be any barriers to their breathing normally during sleep, that is, snoring, a blocked nose or sinus conditions, recurrent allergies causing hay fever or a runny nose, problems with the bones of their jaw, or obesity or excess weight around the chest/lung or throat area. Sleep disruption and/or poor quality of sleep is common in many instances of mental health dysfunction.

If relevant after gathering this information, a referral to a sleep clinic to further explore the possibility of sleep apnoea could save unnecessary medication interventions and the frustration experienced when conventional treatments for ADD or ADHD fail to have any effect on the client's symptoms. Anyone who is having either temporary or long term difficulty with attention, learning, memory, problem solving, regulating emotion or impulse control should first explore whether a lack of sleep or poor quality of sleep could be the cause. It is possible to develop sleep apnoea at any time of life, to be exhausted and not get enough sleep to satisfy the sleep homeostasis

system because of commitments or activities undertaken or to make poor sleep choices, such as choosing to read a novel to the end before turning out the light, on a regular basis.

Clients may also be distracted or prevented from good quality sleep by trying to sleep in noisy environments, such as noise from neighbours or industry, living close to a busy airport, having the television on or music playing while sleeping or sleeping with a partner who snores. If any of these are possible contributors to lack of quality sleep, or lack of sleep at all, then clients and counsellors can explore possible solutions, such as the use of cheap foam ear plugs available from shops selling industrial work gear.

Counsellors should also check on how much sleep a person has typically needed at other stages of their life, because attempting to regularly get more sleep than a person's sleep homeostasis mechanism requires could cause insomnia, which leads to people's balance of REM and NREM sleep being disrupted as they try to get good quality sleep just as their morning alarm goes off.

Because of what is known about circadian clocks and circadian rhythms, counsellors can share information to enable clients to make choices about sleep and possibly shed light on the types of sleep disorders that arise by behaving contrarily to circadian rhythms. Because dysfunction of the circadian clock can contribute to poor mental health, those who experience depression while engaging in shift work, those who have low mood when leading a life style of excessive partying (although there may be other contributing factors, such as drugs and alcohol, and the way that these affect the brain and body) and those clients who regularly experience what is commonly known as seasonal affective disorder (SAD) will have more information on which to base their choices regarding length and time of sleep.

Regularly exposing the retina of the eye and the skin to safe levels of sunlight is also important for some clients to achieve healthy mood including those affected by SAD. For some this can be as simple as spending more time outside, sitting indoors where there is a pool of sunshine or getting into the habit of getting up earlier in the morning, provided the sun has already risen. Others may find that a move to a climate with more availability of sunlight is helpful (jetlag can be alleviated by Melatonin in the form of a pill, which can be purchased from many health food shops) and some find relief with specialist

light boxes to expose the retina regularly to extra visual light. Sun beds should be used sparingly and under professional supervision to ensure safe levels of UV rays. It would not be helpful to cure low mood and replace it with the development of skin cancer.

Once good sleep is achieved, clients and counsellors can review together what impact this is having on daily life, including the higher order executive functions of the brain and mood. Knowing that different people need different amounts of sleep is also helpful information for individuals, and counsellors can encourage clients to tune into their own individual needs in terms of sleep and what amount of sleep is optimal for their mental health.

Omega 3

What do we know?

Omega 3 may prove effective as a mood stabilizer by reducing 'over signalling' or excessive neurotransmission in the brain (Stoll *et al.*, 1999). The precise mechanism for this is not yet clear, but the incorporation of polyunsaturated Omega 3 fatty acids into the lipid (fatty) bi-layer of the cell membrane alters the physical and chemical properties of the membrane. Omega 3 has shown promising results in trials with depressive and bipolar clients to date, with significant improvement in mood in the majority of participants (Innis, 2007; Nemets, Stahl and Belmaker, 2002; Stoll *et al.*, 1999). There have also been good results in the reduction of post partum depression after child birth with trials of Omega 3 (Freeman *et al.*, 2006).

Polyunsaturated fatty acids such are essential components of the central nervous system and they have a role in both learning and memory (Kitajka *et al.*, 2004). Omega 3 fatty acids provide the brain with DHA, which supports growth and function of the nervous tissue. Reduced DHA is associated with impaired cognitive performance (Innis, 2007). DHA is important in the development of the human brain and it is essential that children have adequate levels (Innis, 2007).

Polyunsaturated fatty acids have an effect on gene expression, even in later life (Kitajka *et al.*, 2004). Omega 3 may also have a role in the

myelination of axons (Kitajka *et al.*, 2004; McNamara and Carlson, 2006). Both Omega 3 and Omega 6 are essential to our function but can only be absorbed through diet rather than sourced internally (Kitajka *et al.*, 2004). Fatty acids, and the complex lipids formed from them, are important components of cellular membranes and contribute to the structural and functional integrity of our cells. They have essential roles in the growth and function of the brain (Kitajka *et al.*, 2004).

Syntaxin 3 (a plasma protein) is essential for neural outgrowth of dendrites. Syntaxin 3 requires Omega 3 and 6 to 'oil' the plasma membrane fusion machinery so that it can bind to other proteins in order to be activated (Darios and Davletov, 2006).

Kiecolt-Glaser *et al.* (2007), found that high ratios of Omega 6 to Omega 3 can enhance the risk of depression and inflammatory disease. It is therefore important that the intake of Omega 3 be higher than the intake of Omega 6. Omega 3 at recommended dosage has no harmful side effects (Nemets *et al.*, 2002; Stoll *et al.*, 1999).

What does this mean for counselling?

Omega 3 is another tool that a counsellor can recommend so that people can take charge of their own well being. Some clients may find themselves able to regulate or improve low mood, depression (including post partum depression, sometimes known as post natal depression), BAD and anxiety.

Omega 3 has not been shown to have any harmful side effects and is easily available through supermarkets and health food shops. It is relatively inexpensive, and can also be obtained in New Zealand by doctor's prescription. It can be used on its own or in conjunction with other strategies, such as exercise, healthy diet and taking part in counselling.

Once again, Omega 3 is an option for those who have been recommended to take psychiatric medication for mood disorders and who choose not to. My experience is that there are a significant number of clients to whom this applies.

Recommending Omega 3 does not preclude a counsellor from supporting the client to explore the idea of trialling any prescribed

psychiatric medications and clients may be able to take both the medication(s) and Omega 3 if they discuss this with their prescribing clinician to ensure that the two do not conflict.

Deep breathing, meditative practice and mindfulness

What do we know?

Just like the field of neuroscience itself, neuroscientific study of meditation is in its very early stages (Lutz, Dunne and Davidson, 2007). Many people report meditation as being calming and bringing well being through an inner peace (Lutz *et al.*, 2007). Successful meditative experience is mediated by switching off mechanisms for external attention (Aftanas and Golocheikine, 2001). Some people report a blissful experience, commonly known as 'nirvana'. Brain activity can be measured by EEG and what is measured is defined as having varying frequencies or wavelengths, such as alpha, beta, delta and theta (with the names for each frequency taken from the Greek alphabet). The study by Aftanas and Golocheikine (2001) found that those who reported the meditation they had practised while being measured by EEG as blissful were predominantly producing specific frequencies in the theta range (often associated with dream sleep) and the lower alpha range, supporting the fact that those who engage in meditation which produces a blissful state are consciously moderating and changing the function of their brains from normal waking patterns to patterns more usually associated with deep resting. Deep breathing is integral to meditation as a condition of a meditative state (Lutz *et al.*, 2007), and is practised by all those who meditate, and this would seem to replicate the slower, deeper breathing of sleep.

Aftanas and Golocheikine (2001) also found that those who were able to reach this state had been practising for longer (at least a year, often longer) and that those who had only been practising meditation for a shorter term had their results affected by feelings of frustration at not being able to reach the desired deeper state.

This fits in with other findings presented in this book, such as that repeated practice is necessary to build new neural pathways for new skills (Doidge, 2007; Lutz *et al.*, 2007; Wolfe, 1998) and that as neural pathways become stronger, the neurons become more specifically targeted and more efficient and require fewer neurons to function (Doidge, 2007). In other words, the pathways become like 'second nature' and are effortlessly accessed. This study of meditative practice also supports the finding that new learning requires conscious attention and focus and this was what those in the initial stages of learning were practising (Doidge, 2007; Lutz *et al.*, 2007). The timeframe for becoming adept at this skill suggests that being able to change the length of your own brain waves at will requires very complex pathways in comparison to many other forms of learning.

In contrast to meditation, mindfulness is a practice which involves focusing on the sensory input from stimuli in the present moment, rather than using it to evaluate the future by reflecting on past input from similar stimuli or ignoring present stimuli to make future predictions based on past experience. Mindfulness focuses on the resulting effects of those currently occurring stimuli, such as the noticing of emotional states (Cresswell *et al.,* 2007). Mindfulness has been shown to reduce pathological mental health symptoms as well as adverse physical symptoms across a wide range of conditions and diseases. Studies show that mindfulness practice reduces negative affect, stress, mood disturbance and disease specific health symptoms across a wide range of populations (Cresswell *et al.*, 2007).

It is thought that the process of labelling affective stimuli, for example, emotions and states, may disrupt or inhibit automatic affective responses, that is, well worn neural pathways already created, reducing their intensity and/or duration and allowing the subject to choose different responses (or the option of not responding at all) and not allowing or not completing the established neural pathways by simply pausing at a given point on the neural pathway (Cresswell *et al.*, 2007). Studies suggest that mindfulness is associated with enhanced neural affect/feeling state regulation pathways, meaning that the practice of mindfulness is not only inhibiting old pathways but is building new ones of its own for engaging in mindfulness (Cresswell *et al.*, 2007).

Functional neuroimaging shows that mindfulness, or the labelling of affective stimuli, activates the right ventrolateral prefrontal cortex and attenuates, or weakens, responses in the amygdala. This implies that this pattern of brain activation is driven by top-down prefrontal cortex inhibition of limbic (emotional) responses, probably occurring through neural connections in the medial prefrontal cortex (Cresswell *et al.*, 2007).

Labelling seems to encourage treating affective states/emotions as 'objects' of attention, promoting detachment from negative states and allowing the brain to use pathways in different areas to do the neural processing. There is a strong positive association between dispositional mindfulness and activation of the medial prefrontal cortex, which has been found to be active in self-relevant tasks, such as monitoring your own emotional state (Kandel, 1999; LeDoux, 1996; Schore, 2000).

What does this mean for counselling?

It is probable that both mindfulness and meditation are helpful in rewiring neural pathways, but in different ways. Mindfulness is probably helpful because it can be used to disrupt and inhibit existing neural pathways triggered by incoming stimuli which activate automatic learned neural responses to the chemical signals activated by those stimuli, which are experienced as emotions or states. It can also be used to choose the focus of attention and which stimuli will be processed. For instance, a person may choose to process and focus on the feel of the sunshine on their skin, rather than to ruminate on their problems.

For those clients affected by rumination (including at night, and at times when they are at rest) or racing thoughts, mindfulness is a strategy that counsellors can offer to provide relief. There are many clients affected in this way, who keep themselves constantly busy so as to avoid the thoughts that come when they are still. Some of them then find their problems compounded, from lack of sleep because they lie awake thinking or avoid bedtime until very late, and because they are so constantly busy that exhaustion, both mental and physical, can set in. Mindfulness offers a way to tame those thoughts and to reclaim the safety of stillness.

Mindfulness also offers a way for clients to take advantage of brain plasticity by pausing at early stages of well worn neural pathways and allowing different neural pathways to be created and utilized as the previous ones fall into disuse.

In contrast meditation, rather than consciously focusing on incoming sensory stimulation, seeks a state where external stimuli are excluded from attention. Practised meditators do not notice physical discomfort, such as an itch or a cramp, and those who are very adept are able to meditate while lying on beds of nails, a famous phenomena that my friends and I used to marvel over at primary school, somewhat eclipsed nowadays by the many marvels that television and the internet have given us access to. Just as in states of sleep attention is focused inwards and the neural mechanisms for paying attention to external stimuli are switched off.

Deep breathing is a valuable focusing tool for both mindfulness and meditation; indeed, it is essential to reaching satisfactory meditative states. Breathing provides the brain with extra oxygen so that it functions optimally and is reported to be helpful in the management of both pain and anxiety. It may be redundant to state that the brain cannot function without oxygen and that low levels of oxygen impair cognitive performance. Counsellors can practise with clients as they learn to breathe deeply. Many clients have never done this before. It is usually recommended that clients breathe in for three seconds (counted one and two and three and…) hold the breath for three seconds, and expel the breath for three seconds. Effective breathing can be checked by whether the stomach rises as well as the lungs and clients can self-monitor this. The client's position should be comfortable without being hunched.

Clients who do not sleep well (see the section on sleep in this chapter), are affected by blocked or runny noses for whatever reason, who have bad posture, or who talk fast without pause often find deep breathing useful, as do the more obvious candidates who will find deep breathing a useful strategy for calming anxiety or for managing physical pain when medication is inappropriate or ineffective. Meditation is also a strategy for managing anxiety or physical pain. Clients with nasal blockage should learn to breathe deeply through their mouth until such time as the nasal blockage can be addressed.

It is important that counsellors tell clients who are considering meditation that it is normal for a long period of conscious, repeated practice to be necessary to acquire the skill at a competent level. For some clients this may lead to them focusing on different strategies, such as mindfulness, which is more easily achievable. For others, it may mean that they don't give up when nirvana-like results elude them even after a long period of practice, and that they are more likely to achieve their goal eventually because their expectations are realistic. Those who do achieve competency in meditation will have changed the structure of their brain significantly and will experience greater mental control and increased emotional well being.

The ability to redirect attention internally is likely to mean a greater control of the limbic system, including the amygdala, which is alert to potential threat, as well as the ability to change the frequency of their brain waves when actually engaged in the process of meditation. For this reason, meditation is rightly considered an effective tool in managing anxiety. Meditation may also change the chemical response in the neural reward pathways of our brains, leading to the reported feelings of well being even after the person meditating emerges from the meditative state.

Healthy eating

What do we know?

Both Kandel (1998) and Lipton (2005) believe that every interaction with the environment that we have, and all incoming stimuli, affect our brain and subtly change it. They list the food that we put into our bodies as one of these environmental interactions. Food is not only input from the external environment, but because it provides the minerals, vitamins and nutrients (extracted by our digestive system) which literally fuel our brains, it has an impact on our capacity for effective cognitive function.

Some minerals such as calcium (obtained most readily through the consumption of dairy products) are essential for brain function (Ben-Shachar and Laifenfeld, 2004; Lamprecht and La Doux, 2004),

as are substances like glucose (obtained from natural sugars in food, including carbohydrates), which provide energy for the brain and which are essential for its function, enabling the synthesis of neurotransmitters and communication between neurons. Low glucose levels affect thinking, learning and memory (Magistretti *et al.*, 1999). Physical health is intricately entwined with brain health, or mental health, as well as mental health dysfunction (Cotman *et al.*, 2007; Schloesser *et al.*, 2008; Zarate and Manji, 2006).

The availability of certain nutrients can affect gene transcription and decide the up or down-regulation of genes (Doidge, 2007; Lipton, 2005). Food provides our intake of Omega 3 and 6 (see the section on Omega 3 above) which contains DHA, an important lipid for coating cellular membranes for optimal function (Kitajka *et al.*, 2004). Omega 3 is also involved with myelination (Kitajka *et al.*, 2004; McNamara and Carlson, 2006), which seems to be a neuroprotective factor and which also facilitates neuronal signalling (see the section on myelination in Chapter 2).

Much has been written elsewhere about nutrition and healthy eating and this is easily accessed by interested people. What is relevant to this paper is that eating well is important for brain health and can affect not only the structure of our brain, but both mood and cognitive performance (Geary, 2006). It is reasonable to expect that this will also be expressed through behavioural response to mood and cognition.

What does this mean for counselling?

Most people are aware that eating healthily is important, but not that what they eat can have an impact on how their genes are expressed or on the structure of their brains and the health of their brain's function.

Many people are dissociated from the idea that food is fuel for our brain and bodies and have come to think of it as entertainment (in watching the performance of its preparation, as an adjunct to socializing and in terms of sensory pleasure). Food seems to be regarded variously as a status symbol (better money buys more desirable food) and the enemy, as we are simultaneously encouraged and enticed to consume more of it while being presented with slim body images as the ultimate in body beauty. It is no wonder that people are confused

by food because many celebrities in the public view, with the money to consume as much of it as they want, often only pretend to be partaking of it while simultaneously semi-starving themselves. For those who do not perceive what is happening behind the facade, it is distressing to find that eating food, in the manner in which we are encouraged to, is often not possible in conjunction with obtaining or maintaining the slim body image considered to be ideal, depending on individual metabolism.

Many clients present with eating disorders, ranging from imagined food intolerances to diets that cut whole food groups from a person's diet, to controlled eating (where people eat only a strict, counted amount of food, and tend to be rigid about what they will eat), to bulimia (eating the food and then vomiting it up again before the body can digest and process it, often characterized by binge eating) and anorexia, where very little food is eaten at all, progressing towards a state of eating nothing. Those with anorexia can die from self-inflicted starvation (see also the section on eating disorders in Chapter 5). Prolonged existence on a very low calorie diet can produce feelings of euphoria which become addictive. Controlling food intake can also provide a sense of external control over the world lacking in the client's life (many anorexic clients have underlying anxiety).

Strong neural pathways for feelings of disgust and rejection of food are built. Eating disorders change the structure of the brain, often in long term ways. And when one part of the brain changes, because of the dense interconnections between neurons, it changes other parts in a domino effect.

Body dysmorphia is also associated with eating disorders (see the section on a map of ourselves in Chapter 3). People have their perceptions of their bodies loaded onto internal brain maps, and these maps seem to be able to overwrite analysis of visual input when their bodies change shape and become significantly lighter or heavier, so people with body dysmorphia still believe that they are at their original weight. Hence the person who is affected by anorexia and is all skin and bone sees a fat person when they look in the mirror. Body dysmorphia can go the other way as well, with some obese people not perceiving their bodies as being overly large. This is likely to be the result of a failure of brain plasticity and the ability to adapt and change neural pathways.

Obesity through comfort eating (see the sections on neurons that fire together wire together in Chapter 2 and eating disorders in Chapter 5) made possible by the greater availability of food in this day and age (with the cheapest usually being of the poorest nutritional quality) is literally a growing problem for society, which is not only expensive for society in terms of health costs, but which impacts on people's internal sense of worth. Sometimes this is because of negative feedback from others, sometimes it is because of opportunities missed because of body size, and sometimes it is due to negative internal feedback either because of perceived physical unattractiveness or because of the person's inability to control their urge to eat. Some people who present with mood disorders are affected by these things and poor quality nutritional food intake, whether from undereating or overeating, can also lead to mood disorders. It is reasonable to assume that counsellors will see many clients with eating disorders, even if those eating disorders are not disclosed or do not become the focus of any attention in counselling.

So what can counsellors do? First, eating an appropriate amount (i.e. not too much or too little) of food with good nutritional value is such a basic condition of healthy brain function that it should form part of the initial assessment process undertaken with every client when the client and counsellor are working together to determine what the client's goals are.

My feeling is that time is often wasted attempting to counsel clients whose most basic need is better food (or better sleep) and that supporting them to identify when this is the case is probably the most helpful thing we can do for them. Often clients who experience low mood and fail to make any change to this state through counselling and/or medication fall into this category. I have worked with women, who when tested for anaemia and given iron supplements, found that their lives were changed immeasurably by the extra energy and motivation they were able to access and that they were no longer affected by depression or low mood.

Second, counsellors can share how appropriate or inappropriate food intake can impact on a client's life. This does not mean that counsellors should be food Nazis, just that if a counsellor finds a client receptive to this information, they can support the client to find ways to identify how to achieve a balanced diet of sensible amounts.

This might include referral to a nutritionist or health professional, supplying links to further nutritional information, identifying support people within the client's natural networks, or in the case of the more severe eating disorders, referring to specialists who work with those dysfunctions if that work has not been part of the counsellor's own training to date.

It could also include supporting clients to develop new neural pathways relating to their intake of food and/or their response to it.

As with everything, some clients may have no interest in making healthy food intake part of their strategy, and information surrounding it should be presented as options for the client to explore, rather than as advice, or the client being expected to make the counsellor's goals their own. Counsellors don't make change in other people's lives; people do that themselves, and change is only achieved when it is the goal of the client. This of course does not preclude counsellors from challenging clients to pay attention to unrecognized blind spots or to notice incongruent goals and explore them.

Chapter 7
Conclusion

Scope

It must be acknowledged that there are many recent neuroscientific findings which are not covered within this book, such as the discovery that the design and function of a cell is much like a silicon chip (Lipton, 2005) and that scientists have discovered a way to functionally connect the two (Colicos and Syed, 2006). What I have covered here has been carefully constrained into the areas of neuroscientific discovery that have implications for the practice of talking therapies such as counselling, not that counselling is necessarily limited to verbal interactions between counsellor and client, because it can involve non-verbal therapies such as creative or play therapies as well! I would encourage those who have had their interest in neuroscience whetted to continue to pursue that knowledge, because there is much that I have not touched on here, and a greater level of complexity to the explanations I have given.

The focus of this book has been to make accessible to counsellors the new findings in the field of neuroscience so that they can integrate this knowledge into their practice. Along the way, in my own pursuit of personal knowledge, I hope that I have provided a case study of how this knowledge can be applied. It is not ethical to counsel one's own children, and I have not attempted to do this, but I have used what I have learned to ensure that my son's needs are met by other professionals and to give all of us greater insight.

A key learning from the material presented here is the plasticity of a functional brain and connected to this are the ways in which plasticity can be facilitated, giving people greater control over working towards optimal brain function.

A new era for psychiatry?

Peled (2004) proposes a new way of diagnosing and classifying psychiatric dysfunction, based on brain plasticity, rather than on groups of similar symptoms herded into category types observed by psychiatrists and other health professionals. As I researched this book I was very struck by how all the artificial distinctions I had made for explanation's sake were interconnected; some examples include Bipolar Disorder with stress, stress with sleep, sleep with depression, depression with anxiety and brain health and plasticity with nearly everything. In my opinion, the DSM-IV (American Psychiatric Association, 2000) has the rigidity of artificial boundaries separating one thing from another when often there is significant overlap or combination of symptom types. It makes more sense to base classification on underlying dysfunction rather than just on observable symptoms, which may emerge in different ways in different clients although the basic causal factors are the same.

Peled (2004) suggests that a new system be based on different types of failure of brain plasticity and puts forward three types, the dysfunction of any of which may affect another type or progressively worsen its own condition, with the dysfunction causing further deterioration and dysfunction. The plasticity disorder types he proposes are: the failure of plasticity related to experience based learning, the failure of plasticity of the limbic system which deals with emotion and mood and also with motivational reward, and the failure of connective plasticity between different regions of the brain, allowing integration and coherence and rational perception.

Functional imaging and other ways of measuring brain activity have taken us into a new era of understanding the brain, albeit that we are still very much at the beginning stages of exploring these new capabilities, and that our knowledge is still very limited. It is important that the helping professions connected with neuroscience are able to change and adapt to incorporate our new understandings and this may mean developing new tools with which to do so, including the way in which we approach mental health.

A new era for counselling

Counsellors have a role to play in this, not in developing or putting forward the new systems and/or tools, but in keeping abreast of new knowledge and new ways of looking at mental health so that we are able to best support our clients towards positive outcomes, and the knowledge that we share with them to enable them to make informed decisions is up to date and relevant.

Our primary role may be to provide talking therapy to our clients but every one of our clients is centred by the workings of their own brain. What they do affects the function of their brain and even which genes are expressed. Conversely, how their brain is functioning affects every corner of their lives. Counsellors cannot afford to ignore the way in which the brain works, whether in the context of thinking, feeling or behaviour.

Counsellors deal on a daily basis with people who are building, or who wish to build, new neural pathways: through gaining insight, by being able to feel differently about things or by doing things differently to gain a different outcome. To understand the brain as fully as possible is to facilitate this process.

Kandel (1998) predicts that advances in brain imaging technology will mean that the effectiveness or otherwise of any therapies related to brain cognition will be measurable. Any field related to human therapy which does not avail itself of the new emerging neuroscientific knowledge will find itself redundant and replaced by other fields which do. Psychologists are quickly incorporating neuroscience into psychology and developing the integrated discipline of cognitive neuroscience, sometimes known as social neuroscience. I believe that counselling is also perfectly positioned to make use of the new learnings and that these will add to, and complement, what we already do, making counselling even more useful and relevant to our clients and leaving it a much stronger discipline.

Psychiatry and counselling working alongside each other

Psychiatric medications will no doubt improve and become more specifically targeted and more effective. They will provide relief for many clients with dysfunctional mental health. But there will be some clients who choose not to change the delicate balance of their brain with medication and others for whom medication remains ineffective. No matter which category a client falls into, counselling will be a way in which clients can be supported to make the changes they need to (see Chapter 6) to provide a basis for good mental health through brain plasticity. Neuroscience underpins psychiatry and counselling as potentially complementary, congruent approaches to supporting the optimal functioning of our client's brains, and in the process, a good quality of life for those clients.

Future directions

Neuroscience will continue to build its knowledge base into the future, like a snowball rolling down hill, gathering more and more snow and growing faster and faster as its size increases. My hope is that counselling theory will mesh itself with that snowball of knowledge, cross linking the practice of counselling with the discipline of neuroscience. I also hope that others will take the opportunity provided by this knowledge, including what is discussed here, to develop more and more relevant counselling techniques, including those which will benefit clients with dysfunctions of the brain. Many clients come to counselling with trauma or brain dysfunctions (diagnosed and undiagnosed) and it would be wonderful if counselling as a professional body was able to offer them a service which truly made a difference to those things, and therefore their lives and the lives of those around them.

It is also hoped that the counselling strategies discussed here, and any others that arise, are explored and tested further in a way which is not just anecdotal, because it is important for techniques used within the counselling profession to be evidence based and to be able to demonstrate consistent efficacy of outcome; either in terms

of appropriateness of service for the client or in terms of facilitating positive change in client's lives.

Sharing knowledge

Using the metaphor of pathways for the patterns of neural connections is helpful for explaining the neuroscientific knowledge contained in this book to clients. As per the section on attention and learning, the use of metaphor is a way in which we can connect knowledge which we have no context for with knowledge that we have already consolidated in our brains. All of us know what a pathway is and understand the concept of how pathways become clearly defined and easy to traverse with constant use and how unused pathways can become overgrown and difficult to find. We understand the unsureness that comes from using a new pathway and the wondering if we are on the right track or whether it would be better to go a different way.

As I reviewed what I had written here, I realized just how often I had used metaphor to describe this neuroscientific knowledge to those who will read it, and I thought about how often I use metaphor in my counselling practice to explain new ideas and concepts to clients. I have never believed that there is any point in counsellors having miraculous techniques to use on clients who will go away grateful; instead I think that after modelling those techniques, the counsellor's role is to explain them to clients so that they can use them throughout their own life to continue to make positive change. Metaphor is a natural strategy for counsellors to use because it helps them to support clients to gain insight by viewing the world in different ways so that personal experiences can be reframed, opening up the possibility of new ways of looking at the client's story and the potential for new choices to be made.

Several times during the sections entitled 'What does this mean for counselling?' I have suggested that the counsellor shares knowledge with the client. I am a great believer in equality and equal access to knowledge and that counsellors should share power with their clients, rather than ensconcing themselves in the role of the expert who holds the knowledge and therefore the power. Having said this, it is up to

the counsellor to gauge the amount and complexity of the knowledge that is right for the client. If the client's eyes glaze over and their body language says that they are disengaging, the counsellor needs a simpler explanation, or possibly to couch their explanation in metaphor(s) that will be familiar to the client, so that what the counsellor is saying becomes relevant to the client.

Hopefully, counsellors will be working in a way where they are co-constructing the narrative of the session together with their client, rather than being in the position of being 'the expert'. The counsellor will have the things that they have learned on their professional journey to add to the narrative, but the client will always be the expert in their own life because they have been getting to know themselves for a very long time while the counsellor has probably met them quite recently. The two sets of knowledge should be woven together rather than the counsellor imposing their knowledge as preferential.

Some clients may have no interest in sharing the knowledge the counsellor has to offer and this is fine too. My own experience is that most clients are hungry to know more because it gives the underlying explanations for the things that they struggle with, and gives them hope to make the change that they have come to counselling for.

So what about my project?

I started this journey not only because I'm a counsellor who is fascinated by neuroscience and how it relates to the profession I practise on a daily basis, but because I hoped to gain some new learning that would assist me to support my talented oldest son to lead life as he would like to lead it and not to find his life a sadder, more restrictive place because of his mental health issues.

So what have I learned? I have gained information about addiction and how it replaces neural pathways that exist for natural biological reward that I could pass on to him, although I simplified the language and used the metaphors that I learned to make it easier to fit new knowledge into the existing complexities of our brain pathways. He does not often actively seek cannabis for his own use any more. I learned that everything affects everything else and all four of his

nagging wisdom teeth, which were continually causing infection, have been removed. This immediately increased his mental resilience as he no longer suffered pain and headaches and he could sleep better. Consequently, his concentration and emotional regulation have improved as well and his anxiety is lowered.

The research I did for the section on sleep was particularly valuable and I learned that long term lack of sleep can mimic all the ADHD symptoms of a poorly functioning right orbitofrontal cortex. Prescribed melatonin supplements have helped to address this and to give him more energy, both physical and mental. The dentist gave him jaw exercises to do and these have corrected his jaw which was clicked out of place. (A misaligned jaw can contribute to sleep apnoea and incorrect sleep cycles.) This happened at the same time as the removal of his wisdom teeth, when we noticed a great improvement.

The GP referred him to the sleep clinic in the hope that the poor sleep that he has always had would be addressed and we have recently received the results. An upper airway 'resistance' means that his effort to breathe while he sleeps wakes him briefly an average of twenty-five times every hour (that's nearly once every two minutes). He will be fitted with a C-Pap mask designed to deliver extra air when he needs it and it is hoped that this will make a difference. Being better informed has helped me to be assertive in advocating for my son's needs with mental health services and sometimes I have been the one to explain information to them, such as the existence of the melatonin supplement and the importance of sleep for executive function.

As I finish this book, he feels his life is much improved. He's on a professional cooking course and has been attending regularly, managing to interact with others, although he is still very shy. He has a part time job as a chef in a good restaurant. When things go wrong, he is now more likely to cope than crumble. Life is brighter, he's less sad, more hopeful for the future, and has more good days than bad. It's a long process back to full integration into normal life but we hope that eventually he will have the energy and motivation to exercise and will therefore be able to increase the plasticity of his brain and be more flexible in his thinking, his behaviour and his problem solving ability. Quality sleep itself should also allow better functioning of executive processes, such as impulse control, emotional regulation, forward planning and so on, and the development of new neural connections

over time. I feel unbelievably proud of his progress since I began this journey two years ago.

The knowledge that the brain is plastic and that there is always the potential for change has been a fantastic thing to hold on to. Hope cannot be overrated.

Everything affects everything

One of the most important lessons has been that everything we do and experience changes our brain, which in turn changes what we do and how we perceive things. Our brain affects our mood and how we feel and what we think. This again affects our actions and the environments we place ourselves in, and the focus of what we choose to process in our brains. Everything affects everything, in a very cyclic chain of cause and effect (Doidge, 2007; Kandel *et al.*, 1995; Lipton, 2005). Kandel (2000) believes that because of this interlinking, the distinctions we have made between internal and external causes for psychiatric disorder are no longer valid. External social factors modify the brain and the state of our brain modifies the way in which we behave.

As people, whether we are counsellors or clients, we can control some parts of that interlinked cycle of cause and effect through internal choice. Other parts we cannot control, such as another person's actions towards us, natural disasters which affect our lives or the economic conditions of our time and place.

I am often reminded of the serenity prayer:

> *Give me the courage to change the things I can,*
> *The serenity to accept the things I can't change,*
> *And the wisdom to know the difference.*
>
> (most commonly attributed to Reinhold Niebuhr, 1943, and published in various similar versions)

Some of us have more capacity to make change than others. Some people with low brain plasticity, and therefore low capacity for change,

can be supported to reach a stage where their brains can make better use of natural plasticity, whether that be through better sleep, exercise, healthy eating, taking Omega 3, the absence of former addictions, avoiding stress, using mindfulness or learning to use breathing to calm themselves and relax.

Once in the optimal state to access brain plasticity and to build new neural pathways, it is time to apply the serenity prayer. Counsellors and clients can both do this on their own behalf, and better still, they can do this together so that clients can reach their goals, access their own potential and lead the lives they want to lead. There are many things in this world that can't be changed, but excitingly, we now know that change in brain function *is* possible through plasticity.

Afterword

This work began its life as a Masters in Counselling thesis. I graduated with First Class Honours from the University of Auckland, New Zealand, in May 2013 after two years of part time study, completed while also working full time as a counsellor. I was pleased that I chose to write about a subject that fascinated me, as that made reviewing the literature more of a pleasure than a chore and I was able to retain my motivation. One of the things that made my chosen topic hard going was the fact that many of the texts that I was reviewing assumed more than just a lay person's knowledge of neuroscience and they often quoted very in-depth chemical and mathematical processes. To address this, I slowed the progress of my study by reading some books on basic neuroscience. This gave me enough working knowledge to sift through the readings, but I am very aware that it would take years that I don't have to understand everything to the fullest level. Some readings were little more than advanced mathematical and chemical equations! However, I have been careful to make sure that I fully understood what I have included in this book.

The further difficulty with the neuroscience readings was that even when the authors were explaining the neuroscience, sometimes they missed vital pieces of the picture that they were attempting to illustrate, perhaps assuming what they already knew as authors to be common knowledge. I found this frustrating, because I would reread sections several times only to find that not enough information was given to make things clear to me. This meant I had to use other readings and cross reference them to gain understanding of the first, as well as accessing dictionaries of neuroscientific terms.

Narrowing down the material that I read was problematic. The field is so wide and there is just so much out there. Often the title is

no indication of the research contained within. I would enter areas of neuroscience that I am interested in into the data bases that I use and literally hundreds, sometimes thousands, of available texts came up. Some that I printed off turned out to be just what I was looking for and others turned out to be irrelevant, but I couldn't find this out without wading through most of the text. This was also the case with books that I borrowed from libraries.

Because I was heavily committed in other areas of my life, I found it frustrating when some of the time that I put into my study turned out not to further my study into the topic I had chosen. I wanted to concentrate on the known neuroscience that would make a difference to counsellors as they work with their clients, relating to plasticity, neurons that commonly fire together becoming 'wired' together, emotions and their chemical processes (and how those relate to behaviour), and memory and learning, etc. I also wanted to look at abnormal brain function, because I believe it is often the case that when people come to counselling and are unable to make progress to change their behaviours, trauma to the brain, abnormal brain chemistry or lack of development in certain neurological areas can be the cause.

I think it is important for counsellors to be able to recognize the possibility that some people are constrained by more barriers than others because of the internal workings of their brains. And it is also important to be able to differentiate between what is the result of behavioural choice and what is the result of abnormal brain function.

Another issue for me was figuring out where the line was between what I could use in terms of observed data and the theories drawn from that data. I had thought that I would be researching data that would provide only clear facts about how the brain works but this has turned out not to be the case. The observable functions of the brain are often on a minute scale. The interactions between the different parts and the different parallel processes are incredibly complex. And because of this, I was really struck by how much we really don't know about the way that the brain works. Study in this area is very much in its infancy (particularly study which involves the human brain) and probably comparable to what doctors of physical health knew about the way the body works two or three hundred years ago.

We have experts in the field of mental health and what they know is much more than a lay person knows. But what they know is also very much only a beginning in terms of all there is to know about the brain and how it works.

Much of the research I read is theory drawn from observed data. This means that within a decade the theory may be outdated, or at the very least, modified to incorporate what is yet to be learned. But all I could do is to go on the very best theories that are available now. In some places in this book I had to acknowledge that there is more than one possible theory currently being applied and that experts disagree.

Research that I read was naturally based on the narrow focus of what each researcher was interested in and there are so many functions of the brain, and subsets of those processes, that initially it was very difficult to see a whole picture because I was lost in a sea of details. Some detail was at the very micro level and it was hard to relate that to what I was learning about macro level processes. It emphasized for me again how complex the brain is, and even more so when I realized that the brain drives the entire physical body.

I read a valuable book (which didn't progress my own study that much, but which was valuable scaffolding for what I am trying to absorb) on integrative neuroscience, which was an overview of main theories and how all they might fit all fit together. It ranged from macro to micro level in terms of processes in the brain and connected these together, which made more sense of things for me. In addition, it gave insight into all the ways of researching and studying the brain, often cited in the texts I was reading (Gordon, 2000).

I started to do a bit of reading into social neuroscience, which is the study of how neuroscience relates to human behaviour, and the way in which people interact with each other and choose behaviours. It arose from the field of psychology as psychologists have always been interested in scientifically quantifiable research into behaviour and some of them have begun to realize that it is now possible to study not just external outer behaviour, but the function and internal motivation for that behaviour from within. The fields of psychology and neuroscience are moving closer together and it is important that the field of counselling (which is more person centred, taking into account the client's own goals, and ability to make change) is not left behind.

I believe it will be essential in the future for counsellors to have enough knowledge of neuroscience to remain relevant and in touch with the knowledge base that will be emerging to knit several disciplines together, not to subsume each other, but to have common threads of connection, providing an integrated set of areas of expertise rather than disciplines operating in opposition or isolation.

When I first began this project my children were growing up and not needing the close attention that I had once given them, and the time seemed right. And then, soon after I was already engaged in my work, I found myself with full custody of my first grandson, just over 1 year old. The time was not really right anymore, but I had left the starting blocks and did not want to pull out of the race. In fact, I was determined to complete it anyway.

I had a personal reason for wanting to do this project: my oldest son, and the father of my grandson, was experiencing mental health problems. Doing this work was both a way to distract myself from some very upsetting things in my life and a potential pathway to being able to discover something which could change his circumstances, and therefore mine, and those of the rest of my family. I was hoping that along the way I might come across some knowledge which would be relevant and helpful to his situation and that we could put it to good use.

My life had become an endless round of appointments with mental health professionals and other social service workers, and none of them seemed to have anything to offer which was making a difference to us. I had started to read a bit about what seemed to be affecting my son and quickly found myself in a position of being more informed about what ailed him than any of the people that we went to see, including the psychiatrists. I found the system really frustrating, as there was no value put on my knowledge or the self-knowledge of my son and my family (all the knowledge was presumed to reside with the medical professionals) and to offer any input at all was considered to be presumptuous, and to contribute anything angered the professionals in the mental health service, even though they themselves seemed to have nothing to offer. Even defining the problem was problematic.

I began to wonder why I wasn't using my counselling training and behaving in the way that I did when I was working with clients, which

was constructed around my own world view (client centred, holistic, strengths based, etc.) in my own life.

Naturally, mothers do not counsel sons, but I hoped to find some information which could help during the course of my studies and I began to consciously apply my world view and the actions which would naturally arise from that to my own situation; noticing my son's strengths out loud, focusing on small improvements and small positives and trying to avoid endless focus on and discussion of the problems. I began to speak to him and others as if he would be well again, instead of assuming that he and we (and I) would be perpetually trapped by what was happening at the current time. I say trapped because he was totally dependant on me; to do the daily tasks that required contact with people (because he was severely affected by anxiety) and to calm him and mop up the fall out from his emotional dysregulation and lack of consequential vision, including with the police and courts as well as with family.

I also became aware of how our professions really operate in silos, unaware of the knowledge that each other has access to. Psychiatrists, psychologists, counsellors, social workers, nurses, NGO workers and field workers all knew things, but had no points of reference to what the others knew, and many discounted knowledge from outside their own field as irrelevant. And within this were the professionals who had trained so long ago that they were not even abreast of the knowledge held within their own fields (which really brought home to me how important continuous professional development is across the entire course of a career – there is always something new to learn!) None of this fitted with my holistic world view and I really, really wanted to connect the dots that were important to me because of my experience with my son. I am never content to be told that something 'is', I need to know how it all fits together.

To be honest, I have always had an interest in the area of neuroscience. I worked for Parents as First Teachers (PAFT) for 12 years before becoming a counsellor. PAFT is a New Zealand governmental contract designed to support parents and caregivers of children aged 0–3 years and a large component of it was to teach early brain development knowledge to those parents and caregivers. So when I did my counselling training, even though I loved what I was learning – strengths based practices, solution focused strategies,

narrative therapy, co-constructionism, being client centred, etc. – I was always asking the trainers how this fitted in with neuroscientific knowledge, only to given blank looks and to be told that they didn't know. Often it was implied that it wasn't important. I wondered how they could be content without connecting the world in a holistic way.

So I undertook the project of writing this book. To do this, I had to type with our baby on my knee when he was fractious with teething, get up two hours early every day to do readings (always readings and more readings; I once spent an aeroplane flight highlighting key passages in readings on eating disorders) and give up entire weekends (I still met the needs of my children and family, but leisure and relaxation time was non existent), while others such as my teenage daughter babysat in the living room (thank you so much, darling).

My process was to do the readings, highlight them, write the main points onto blank paper and then connect the readings together in my book as I typed it out. Then it was back to the source materials as I reviewed each chapter. Had I missed anything? Had I made the connections which needed to be made? It required a lot of lateral thinking to understand how the pieces of the jigsaw fitted together. As I have already noted, many of the readings themselves were highly complex: full of technical and scientific jargon and abbreviations, as well as being highly specialized in terms of chemical, mathematical and biological knowledge. I had to teach myself as I went along.

And that's where I think that what I have produced has value; comparative to the source material, my work is in plain language and reasonably accessible to all who have an interest. A glossary of terms is provided to clarify the material even further. Putting that glossary together was really helpful for my own understanding as well. I hope that other counsellors are able to use this book to incorporate neuroscientific knowledge into their existing practice, and I believe it fills a gap in terms of our knowledge base as counsellors. I like to think that other counsellors will use the learnings I have presented as a basis from which to add to the strategies and techniques that the counselling profession might use to complement this knowledge.

Over the course of this project my son has come a wonderfully long way in a positive direction. A combination of being strengths based myself (which was hard while those around me were reluctant to come on board with this approach), environmental changes, having his

infected wisdom teeth removed and his formerly clicky jaw corrected, having a supportive tutor on his course, being in a place where rent was affordable, giving up smoking cigarettes, minimizing cannabis use, getting better sleep, and a kick start from some top of the line anxiety pills (Bruspirone, which are no longer needed) has made a huge difference in my son's life.

I hope that what I have learned may be used by other counsellors to benefit people in the sort of situation that my son, my family and I found ourselves in and that it will be used by counsellors in a strengths based, co-constructionist sharing relationship between client and counsellor. The knowledge that the brain has the characteristic of plasticity and the capability for change has helped us immensely, because it provided the hope that carried us through to better times. And now, I think, I am ready to let this go for a while and concentrate on other things.

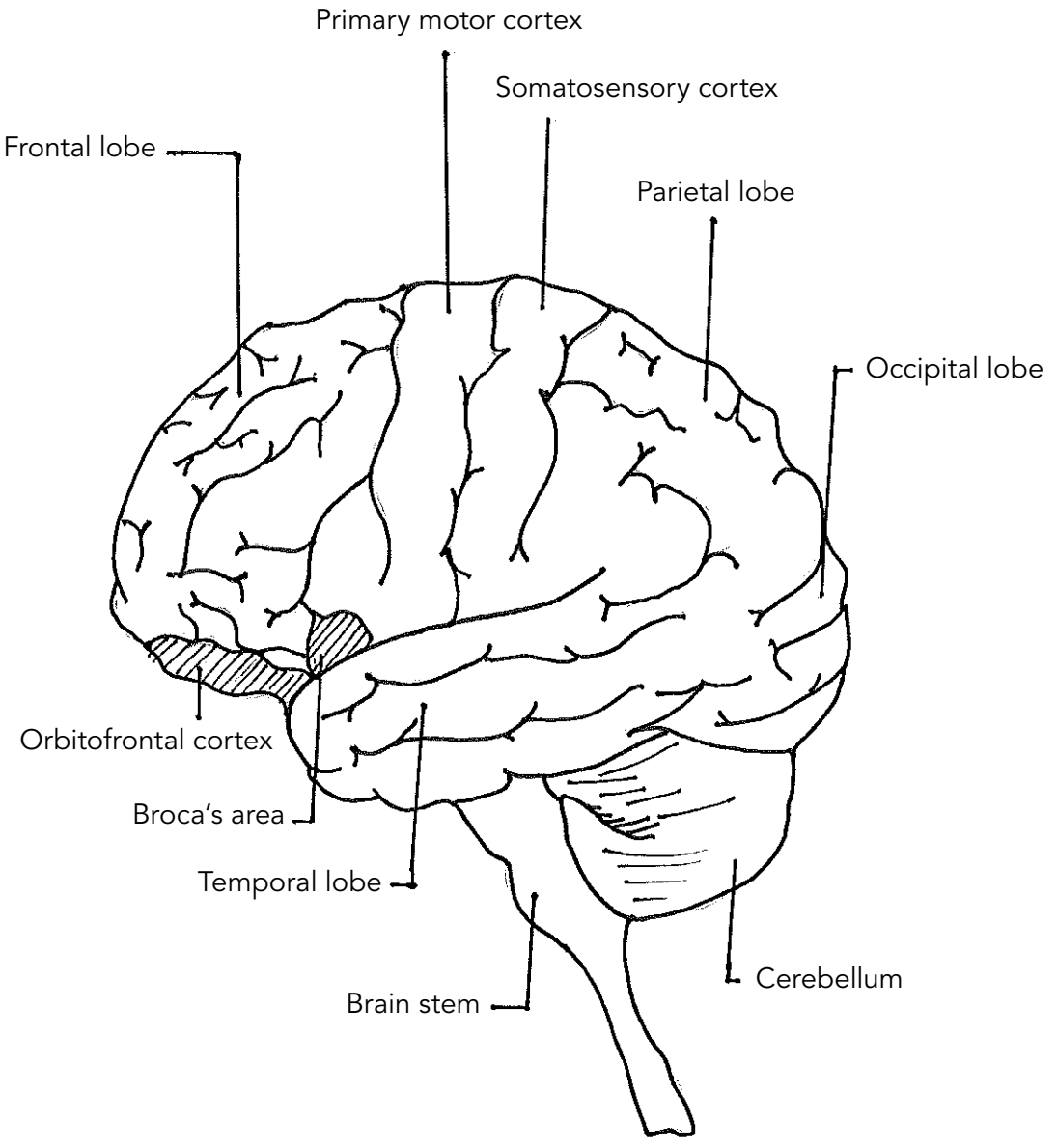

Figure 1: Regions of the brain.

Note: Lobe is a synonym for cortex.

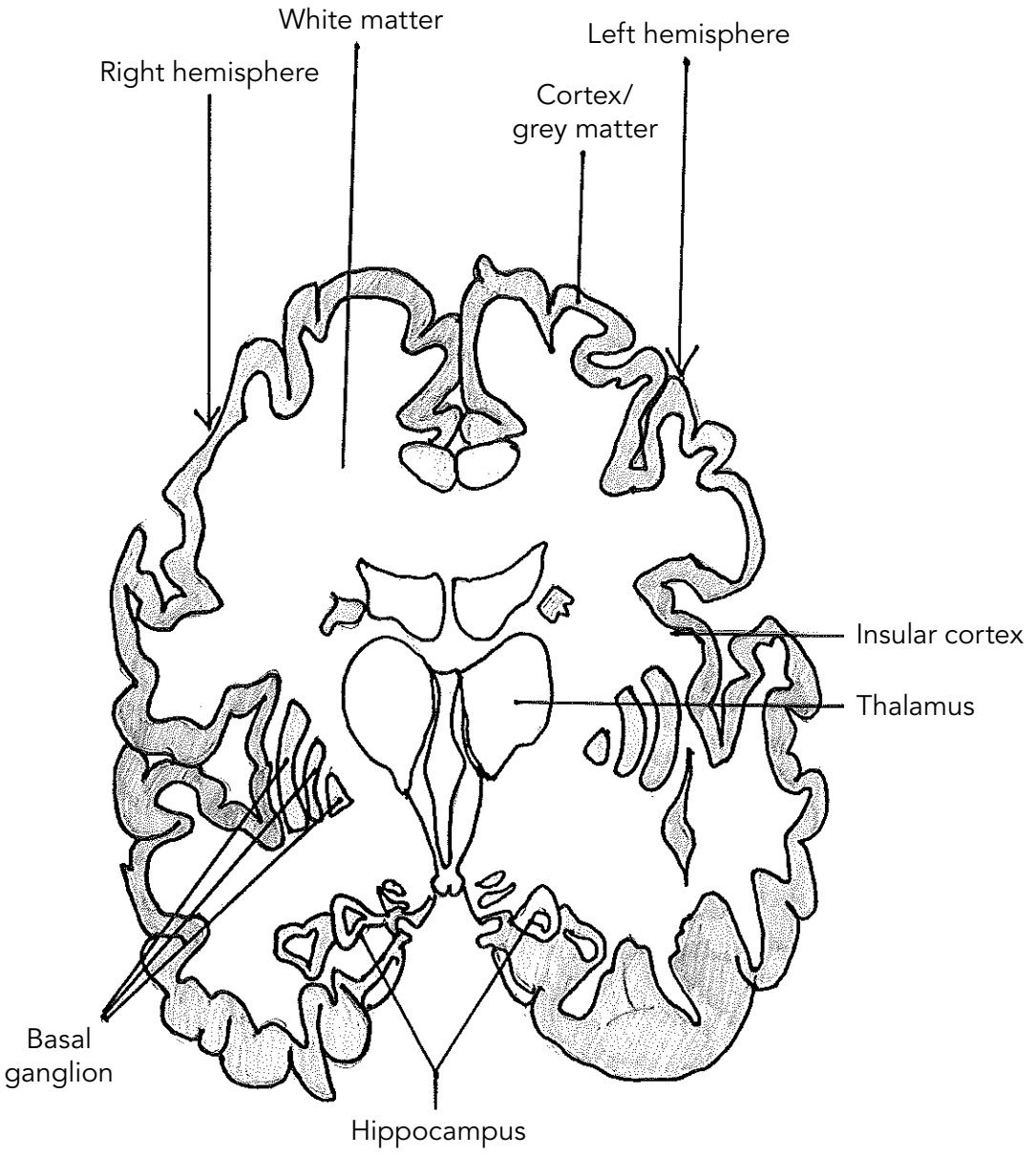

Figure 2: Overview of the hemispheres of the brain.

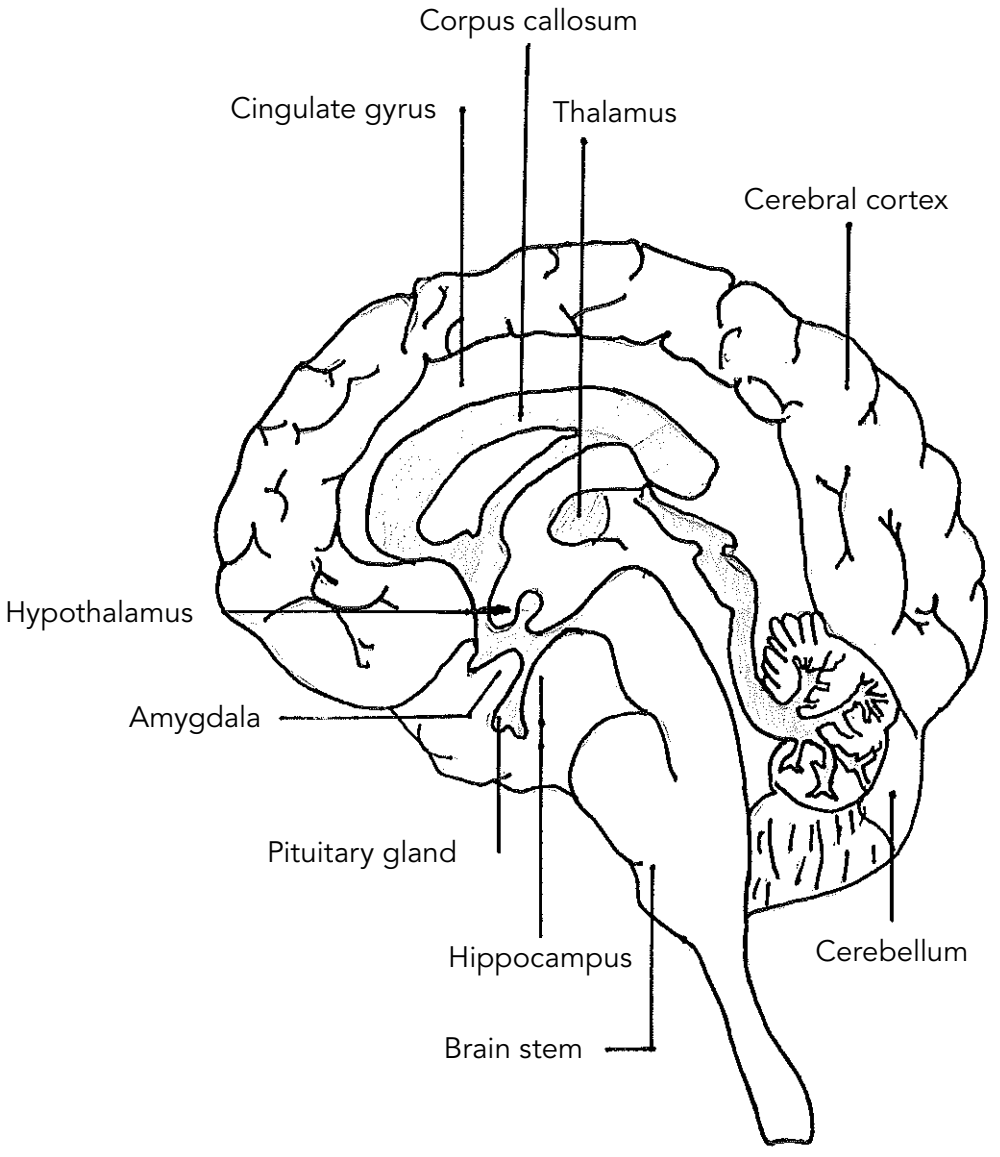

Figure 3: The limbic system of the brain.

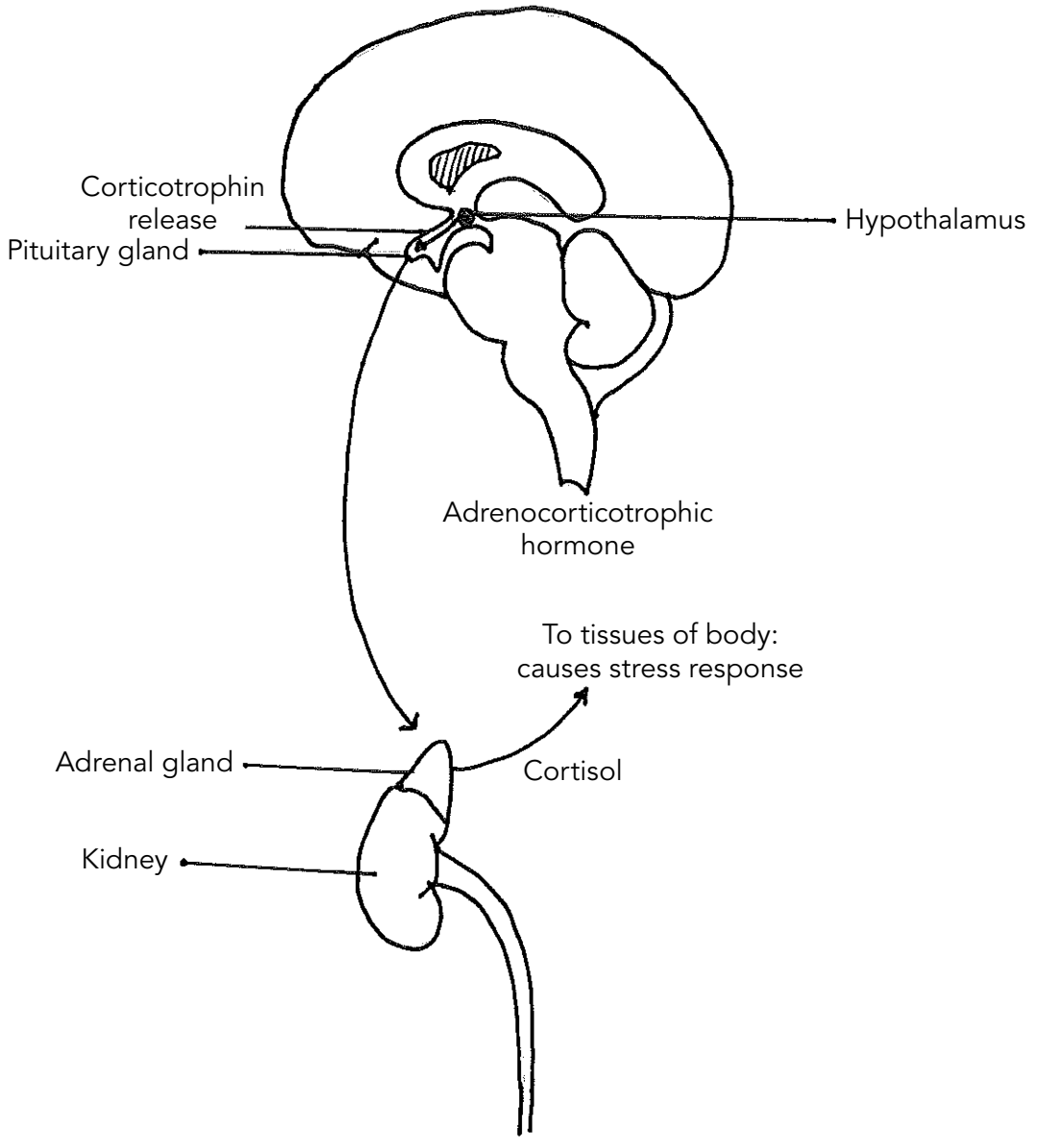

Figure 4: Hypothalamus–pituitary–adrenal (HPA) axis.

Glossary

5-HTT A serotonin transporter gene.

Acetylcholine A neurotransmitter, important in learning, memory, concentration and perception.

Action potentials Electrical signals from the neuron sent down the axon, which must reach a certain threshold to fire.

Affect When used as a noun, describes a person's emotional state and presentation.

Afference The brain's reception of sensory signals from the body.

Alpha A type of electrical wave created by neuronal activity in the brain, able to be measured by EEG.

Amino acids The building blocks of proteins. Divided into non-essential (made within the human body) and essential (not made in the human body) amino acids, which need to be up taken through diet.

Amygdala A collection of nuclei that lie deep inside the cerebral cortex of the brain connected to the hypothalamus, the thalamus and the hippocampus. Specifically concerned with emotion, especially anxiety and fear.

Anterior/rostral The part of any region of the brain lying closest towards the front of the brain.

Anterior cingulate cortex A region of the brain near the hippocampus thought to be responsible for directing attentional focus.

Antioxidant A chemical compound or substance that inhibits oxidation or reactions promoted by oxygen. Can prevent damage caused by excessive oxidation.

Asymmetry Refers to the differing functions of the left and right hemispheres of the brain, which can work together simultaneously on different tasks or different facets of the same task, conferring an evolutionary advantage. Sometimes one hemisphere can be more dominant than the other.

ATP/Adenosine Triphosphate A nucleotide/DNA building block which transports chemical energy within cells. Contains three phosphate groups. Associated with gene transcription and cellular signalling among other things.

Atrophy Loss of function due to disuse. (Can apply to neurons, glia or their component parts and also to other parts of the body, such as muscles.)

Aural/auditory Related to hearing and the processing of sound stimuli.

Autonomic function The part of the brain that controls automatic functions, such as breathing and circulation. The nervous system.

Axon The output fibre of a neuron, which ends in terminals designed to connect it with other neurons.

Basal Meaning the base of a region.

Basal forebrain Located under the striatum, in the bottom region of the front of the brain. Made up of basal ganglia, the basal nucleus (and other important collections of nuclei), the amygdala, a part of Broca's area and the ventral palladium. Produces acetylcholine, which is highly important in encouraging brain plasticity and learning.

Basal ganglia Interconnected masses of grey matter located in the interior regions of the cerebral hemispheres and upper part of the brain stem. Connected by the striatum.

Basolateral nucleus A collection of neurons in the amygdala. Its projection into the nucleus accumbens is regarded as the limbic-motor interface of the brain. It also has a reciprocal connection with the hippocampus and is connected to the medial prefrontal cortex;

a circuit implicated in fear extinction. Assigns emotion to incoming stimuli.

BDNF/brain derived neurotropic function A trophic factor in the brain responsible for optimal brain plasticity.

Beta A type of electrical wave created by neuronal activity in the brain, able to be measured by EEG.

Brain stem The part of the brain at the base, which connects the brain to the spinal cord, relaying messages between the brain and the body. The brain stem controls basic bodily functions, such as breathing and heart rate.

Broca's area A region of the left prefrontal cortex associated mainly with speech and language.

cAMP/cyclic adenosine 3', 5'-monophosphate A nucleotide involved in protein binding and in transmission of signal between neurons. Known as a 'second messenger' type of transmitter.

Candidate mechanisms Possible catalysts/pathways for processes whose biological workings are as yet unknown.

Canonical neurons Neurons which respond during the actual execution of actions.

Cardiovascular Relates to the circulatory system of the heart and blood vessels.

Cascades See neural cascades.

Caudal/posterior The part of any brain region closest towards the back of the head.

Caudal medial prefrontal cortex Mid prefrontal cortex.

Caudate nucleus Part of the basal ganglia. There is one in each hemisphere of the brain; partly responsible for movement and coordination.

Central nervous system A system comprising the brain, the spinal cord and the peripheral nerves of the body.

Cerebellum The part of the brain at the back of the head, between the cerebrum and the brain stem.

Cerebellar vermis A narrow, wormlike structure separating the two hemispheres of the cerebellum.

Cerebral Of the brain.

Cerebral cortex Comprised of a thin mantel of grey matter, covering the surface of each cerebral hemisphere. It's made up of layers of neurons and the pathways which connect them.

Cerebral hemispheres The two halves of the cerebrum, the largest part of the brain.

Cerebrum One of the main areas of the brain, divided into two hemispheres, and further divided into four regions: the frontal, the parietal, the temporal and the occipital cortices. The largest region of the brain.

Chiasm A crossing of two tracts of nerves (as in the optic chiasm) or ligaments.

Cholecystokinin A neuropeptide.

Chronic Persistent; constant or frequently reoccurring and of long duration.

Circadian clock A genetic body clock located in the brain, which regulates physical cycles including sleep/wake cycles.

Cognition Thought processing.

Cognitive Of or relating to thought processing.

Co-morbidity Conditions that occur together simultaneously.

Conditioning A specific learned response to a specific stimulus.

Corpus callosum A neural structure comprised of axons which joins the left and right hemispheres of the brain. The largest neural structure of this type.

Cortex/cortices Regions of the brain including the anterior cingulate, the temporal medial, prefrontal, the orbitofrontal, the entorhinal, the perirhinal, the parahippocampal and the dorsolateral prefrontal cortices (cortices is the plural). Lobe is a synonym for cortex.

Cortical/cortico Of, or relating to, the cerebral cortex.

Corticosteroids Adrenal steroids, triggered by stress.

Cortisol The primary stress hormone. Necessary for metabolizing starches. The major natural glucocorticoid.

Coupling Functional connectivity between cortices.

CREB/cAMP response element binding A gene transcription factor.

CRH (corticotrophin-releasing hormone)/CRF (corticotropin-releasing factor) A hormone produced in the hypothalamus, which stimulates production of corticotrophin, made in the pituitary gland, which releases corticosteroids, triggered by stress.

Critical periods Specific periods of in childhood and adolescence where the brain is optimally primed for specific development.

Cuneus A small part of the occipital cortex, associated with visual processing but also linked to inhibitory control.

Cytoplasm Everything contained within the membrane of the cell, excluding the nucleus. Contains cellular mitochondria.

Cytoskeleton The scaffolding structure of cells.

Declarative memory Memory for past events. Sometimes called episodic memory.

Demyelination Loss of the myelin coating of the cell.

Dendrite The receptor elements of neurons. The two major sites of dendrites are spines and shafts.

Dentate gyrus Region of the hippocampus where new neurons are formed, even into adulthood.

Depolarized A change in a cell membrane's electrical charge, making it more positive (or less negative), which may result in an action potential (an electrical signal to another neuron).

DHA/docosahexaenoic acid An essential fatty acid found in Omega 3. Supports nervous system tissue growth and function.

Differentiation Where cells are specialized for different and specific functions.

Discrete Separate, distinct and/or individual.

Dopamine A neurotransmitter, related to feelings of pleasure when released, and also to the retention and reinforcement of learning.

Dopaminergic Describes the dopamine system.

Dorsal/superior The part of any brain region closest towards the top of the head.

Dorsolateral prefrontal cortex Another region of the prefrontal cortex. A late maturing region of the brain linked with impulse control, organization, working memory and motor planning. Linked to many other regions of the prefrontal cortex, including the orbitofrontal cortex and the hippocampus.

Down-regulation Decreased expression of a gene.

Dysfunction Abnormal or impaired functioning.

Dyslexia Difficulty in processing and using written symbols.

Dyspraxia An impairment of the ability to perform coordinated movement.

Dysregulated A failure to regulate or moderate a process appropriately.

EEG/electroencephalography Measurement of neural electrical activity in the brain through means of electrodes placed on the scalp for a period of time.

Endocannabinoids Naturally occurring chemicals in the brain with a chemical composition similar to that of marijuana.

Endocrine The system that releases glandular secretions (hormones) into the blood stream.

Entorhinal cortex A region of the brain important for long term memory storage.

Enzyme A protein which catalyses chemical reactions without being altered or destroyed itself in the process.

Estrogen A female hormone.

Epinephrine/adrenalin A neurotransmitter secreted from the adrenal gland.

Episodic memory Memory for past events, sometimes called declarative memory.

ERK/extra cellular signal related protein kinase Protein kinases outside the cell involved in the process of neuronal signalling. Part of the MAP kinase cascade, involved in the regulation of cell division.

ERP/event-related potential Flow of electrical signal between neurons related to input data.

Excitatory An excitatory post synaptic potential describes positive electrical ions flowing into a cell and negative ions flowing out (known as depolarization) making it more likely that the cell will fire an action potential (send a signal to the next neuron). Many excitatory signals are needed to raise the threshold to where an action potential is fired.

Executive functioning Higher order brain function; involved in planning, attention, learning and remembering, impulse control, emotional regulation and empathy.

Extracellular Outside of a cell membrane.

fMRI/functional magnetic resonance imaging A non-invasive brain imaging technique using magnetism, radio waves and a computer, which checks which brain areas are active at specific times and can provide information about cellular activity.

Focal lesion An infection, tumour or injury that develops in a specific, restricted area of tissue.

Forebrain In the adult brain, this comprises the two cerebral hemispheres, the thalamus and the hypothalamus. It is the main control for sensory and associative processing, voluntary movement and the function of large bodily organs.

Fronto A prefix indicating front of head.

GABA/y-aminobutyric acid A powerful amino acid. Amino acids are the building blocks of proteins.

Gamma A type of electrical frequency created by neuronal activity in the brain, able to be measured by EEG.

Ganglion A cluster of functionally related neuron cell bodies.

GAP-43/Growth Associated Protein 43 A protein associated with axonal growth, learning, memory and neurotransmitter function.

Gene transcription/expression The switching on or off of genes within cells according to environmental triggers and stimuli.

Genetic template Heritable genetic coding which is passed down from one generation to the next.

Gherlin A neuropeptide.

Gland An organ that produces secretions for use elsewhere in the body or brain. Glands in the brain include the adrenal gland and the pituitary gland.

Glial cells A type of cell in the brain; more numerous than neurons, but not responsible for the transmission of information. Instead, glial cells play a supporting role in providing the myelin sheath around neural cells, and scavenge the debris of dead cells in the brain.

Glucocorticoid A class of hormone which primarily affects the metabolism of carbohydrates, and to a lesser extent, fats and proteins. Made in the outer region of the adrenal gland. Chemically classed as a steroid. Cortisol (known as the stress hormone) is the major natural glucocorticoid.

Glutamate A common amino acid, allowing transmission of signals between neurons.

Glutamatergic Describes the glutamate system.

Glycolysis A biochemical reaction involved in cellular energy production through fatty acid oxidation.

Granule neurons Neurons in the dentate gyrus which give birth to new neurons in the process known as neurogenesis.

Grey matter The cortex of the brain, which contains neurons. Grey matter is darker than white matter, which contains myelinated neural fibres. A synonym for basal ganglia.

GSK-3/Glycogen Synthase Kinase-3 A protein enzyme found in the brain.

Gyrus/gyri Any of the prominent elevated folds of the surfaces of the cerebral hemispheres (gyri is the plural).

Habituation A decrease in response to a stimulus after it is repeatedly experienced.

Hippocampus A structure deep in the temporal hemisphere of the brain. Involved in memory systems, attention and learning, and other higher order functions of the brain.

Histone Any of a group of small, basic proteins usually found in association with DNA.

Histone deacetylase A class of enzyme that removes acetyl groups from histones. Acetyl groups allow gene transcription to occur and deacetylation inhibits gene transcription.

Homeostasis A healthy, balanced state maintained by constant adjustment to biochemical and physical function in the body and/or brain.

Hormone A chemical substance produced and released by cells or glands which act as chemical messengers to affect and/or regulate the function of other cells.

HPA axis/hypothalamus–pituitary–adrenal axis A strong linkage between the hypothalamus and the nearby pituitary glands and the adrenal glands, which are located elsewhere in the body, just above the kidneys. Involved in the management of stress and emotional regulation.

Hyper A prefix meaning heightened.

Hypo A prefix meaning lowered.

Hypofrontality A state of low metabolism in the frontal cortex and forebrain associated, but not exclusively, with addiction.

Hypothalamus A structure regulating autonomic, endocrine and visceral function in the brain. Located in the brain below the thalamus, it regulates body temperature, sleep, appetite and sexual development. Through its control over the pituitary gland located closely nearby it

also regulates the thyroid, pancreatic and adrenal glands and also the gonads.

IGF-1/Insulin Growth Factor 1 A protein with high sequencing similarity to insulin. Linked to neurogenesis and memory.

Inferior/ventral The underneath section of any region of the brain; the bottom area of any part of the brain.

Inhibitory An inhibitory post synaptic potential is where negative electrical ions flow into a cell, and positive ions flow out (known as polarization). This makes it less likely that the cell will send a signal to other neurons.

Insula/insular cortex A portion of the cerebral cortex folded between the temporal and the frontal lobes. Associated with consciousness and self-awareness, emotion, perception, motor control and cognitive function.

Insulin A hormone made by the pancreas, which controls cellular use of glucose for energy and therefore blood sugar levels.

Insult Harm or damage.

Interneurons Neurons which perform a specific relay function between sensory and motor neurons.

Intracellular Within a cell membrane.

Kinase A class of enzyme which transfers phosphate during phosphorylation.

Lateral Areas of any region of the brain which are away from the middle or the midline of the brain.

Leptin A hormone associated with appetite regulation.

Lesion Structural damage to a specific part of the brain.

Limbic system A part of the brain surrounding and including the hippocampus, amygdala and part of the brain stem, which is involved in emotional regulation and also learning and memory. Consists of the limbic lobe and parts of the frontal, parietal and temporal cortices.

Lipids Fats.

Lobe A synonym for cortex (cortices is the plural).

LTD/long term depression A state of stable, non-plastic synaptic connection, allowing semi-permanency of synaptic connection between neurons.

LTP/long term potential A state of change in neural connectivity, where synapses are 'soft' and vulnerable to change, promoting plasticity as electrical messages pass between neurons. Able to be provoked under certain frequencies in laboratory conditions.

MAO-A/monoamine oxidise A An enzyme which breaks down neurotransmitters, such as dopamine, serotonin, norepinephrine and epinephrine.

MAP/mitogen-activated protein kinase A protein kinase activated by a catalyst for cell division.

Massed practice A skill repeatedly practised over a long duration.

Medial The mid part of any region of the brain. Towards the midline of the brain.

Medial frontal cortex The mid part of the prefrontal cortex.

Medial temporal lobe/cortex The mid part of the temporal lobe, which is located adjacent to the occipital lobe and contains the auditory cortex.

Membrane The outer 'skin' surrounding a cell, containing the rest of the cell's components.

Meso Prefix meaning 'mid' or middle.

Metabolism The entire range of biochemical processes in a living organism, including the build up and breakdown of substances. Commonly used to refer to the transformation of food into energy.

Midbrain The short, upper part of the brainstem, which contains major nerve pathways.

Mirror neuron A special class of neuron that fires when observing actions as well as when performing them. Associated with imitative learning, empathy and understanding the social intention of others.

Mitochondria Parts of the cell outside the nucleus, but inside the membrane, which synthesize energy for the cell. The DNA contained within them is always inherited through maternal lineage.

Mitogen A substance which induces cell transformation through the division of cells.

Mitosis The most common process of cellular division.

Molecular The study of basic biochemical building blocks of life; how DNA forms RNA polymerase, which forms proteins, etc.

Molecules The smallest particles of a substance which retain the chemical and physical properties of that substance.

Monoamines A single type of amine (an organic compound containing nitrogen); often compounds which function as neurotransmitters (chemicals which carry messages between neurons).

Motor Concerned with the function of movement.

Mu A type of electrical frequency created by neuronal activity in the brain, able to be measured by EEG.

Myelin A mixture of protein and fats surrounding nerve cells, increasing signal speed between those nerve cells.

NAA/*N*-acetyl-aspartate A neurochemical compound.

Neo-cortex A more recent evolutionary part of the brain that controls sight and hearing and may be involved in memory storage. Includes the parts of the brain associated with higher order functioning, such as the prefrontal cortex and the orbitofrontal cortex.

Neural Relating to neurons.

Neural cascades Interacting gene transcription and behaviour signalling pathways in the brain.

Neurochemicals Chemicals that circulate in the brain ('neurochemical' being the processes that relate to their interaction).

Neurogenesis The renewal of all types of nerve cells in the brain through the formation of new cells.

Neuro modulator Brain chemicals which moderate emotional and physical states.

Neuron A nerve cell which carries information in the brain and which relays information between the brain and other parts of the body.

Neuronal fibres Axons can range in length from one millimetre to one metre and these are known as nerve fibres. Sometimes these axons can become bundled together in shared pathways stretching from one region of the brain to another.

Neuropeptides Peptides, or molecules of amino acids, which constitute proteins found in the brain.

Neuropeptide Y A neuropeptide which modulates emotional response and resilience to stress.

Neuroplasticity Changes in the brain, which can be synaptic (synaptic plasticity) but which might also be chemical or related to the architecture of the brain or to the density of neurons.

Neurotransmitter A chemical which carries messages between neurons.

Neurotrophic factors/trophic factors Proteins within the brain which promote neural survival and general brain health.

Norepinephrine/noradrenalin A neurotransmitter which is a secretion of the adrenal gland.

NMDA/*N*-methyl-D-aspartate A glutamate receptor.

NREM/non rapid eye movement A phase of sleep associated with slower brain waves and low brain activity.

NT-3/neurotrophin-3 A neurotrophic factor.

Nucleotides Small molecules, which, when joined form the nucleic acids of RNA (the gene transcription factor) and DNA. In addition, nucleotides participate in cellular signalling and metabolism.

Nucleus/nuclei The central part of a cell, containing genetic material.

Nucleus accumbens A collection of neurons which forms the main part of the ventral striatum, which is part of the basal ganglia.

Associated with reward, pleasure, laughter, addiction, aggression, fear and the placebo effect.

Occipital cortex One of the major regions of cerebral cortex of the brain.

Oligodendrocytes Glial cells involved in the formation of the myelin sheath which coats axons.

Opioid A neuropeptide.

Optic Relating to vision and visual perception.

Orbitofrontal cortex The region of the brain connected with executive or higher cognitive processes, such as impulse control, emotional regulation, the ability to plan and to envisage consequences, putting information into context, and learning and attention. Also associated with sensory integration. Located in the frontal lobes of the brain and connected to many other regions of the prefrontal cortex.

Oestrogen A female hormone.

Oxidative phosphorylation The addition of phosphate to an organic compound, in this case mediated by oxygen.

Oxytocin A hormone made in the brain believed to have a role in attachment and bonding.

Para A prefix meaning alongside, or near to.

Parahippocampal cortex/parahippocampal gyrus A region of the brain which is part of the paralimbic cortex, which is next to the limbic lobe.

Paralimbic cortex A three layered cortex comprising the pyriform cortex, the entorhinal cortex and the parahippocampal cortex. Sometimes called the mesocortex. Lies close to the limbic structures and is sometimes called the paralimbic system, in conjunction with the limbic system, as the boundaries between the two are often blurred, particularly in relation to the amygdala.

Parietal cortex One of the major regions of cerebral cortex of the brain.

Pathology The study of the nature of disease/dysfunction, and the structural and functional changes these conditions produce.

Peptides Molecules made up of two or more amino acids, which come together to form proteins.

Peptide YY A neuropeptide.

Peripheral In neuroscience terms, meaning outside of the brain.

PET/positron emission topography A brain imaging technique which uses short lived radio-active substances to produce three dimensional images of brain function.

Pharmacology Medication which chemically intervenes in the brain.

Phosphorylation A biochemical process involving the addition of phosphate by kinase enzymes.

Physiological Consistent with normal physical bodily function.

Pineal gland Located in the brain; produces the hormone melatonin, which helps regulate sleep/wake cycles.

PKC/protein kinase C A protein synthesized in the brain.

Polarized A negative state of electrical charge in a cell; inhibiting the rise of action potentials (electrical signals between neurons).

Posterior/caudal The part of a region of the brain lying closest towards the back of the brain.

Post-synaptic Situated behind, or occurring after, a synaptic connection.

Precursor A substance from which another, usually more active or mature, substance is formed.

Prefrontal cortex The anterior/frontal lobes of the brain.

Prefrontal cortical hypofrontality A low state of metabolism in the prefrontal cortex, leading to dysfunction.

Premotor cortex An area of the motor cortex lying within the frontal lobes and very close to the primary motor cortex. Not fully

understood, but associated with planning movement, using abstract rules to perform tasks, understanding others and empathy.

Pre-synaptic Situated in front of, or occurring before, a synaptic connection.

Primary motor cortex The main part of the motor cortex where impulses from nerve centres to the muscles, facilitating movement, originate.

Procedural memory Automatic memory for skills and actions.

Progenitor cells Cells which can differentiate into specific types of cells. Similar to stem cells, which can differentiate into any type of cell.

Protein Formed from combinations of peptides, which are formed from combinations of amino acids. Proteins provide the instructions for many of the internal processes in the brain. Used to make neurotransmitters.

Psychosis Psychiatric dysfunction that involves sensory perception without corresponding external stimuli; causing hallucination and/or delusion, and a lack of communication with reality as it is generally perceived.

Pulvinar nucleus A collection of neurons in the thalamus.

Purkinje cells A special class of neuron found in the cerebellar cortex. They are some of the largest, most complex neurons in the human brain, with a profusion of dendrites and dendritic spines and long axons. These cells are the sole output for motor coordination in the cerebellar cortex. Also implicated in learning, attention and cognition, they receive more synaptic input than any other part of the brain.

Putamen The large, dark part of the grey matter/basal ganglia; forms the striatum together with the caudate nucleus and is co-existent with the insula. Projects to the premotor cortex.

Pyramid cells A type of neuron found in the cerebral cortex, the hippocampus and the amygdala, which performs an excitatory function. Named for their triangular shape.

Raphe nuclei A cluster of nuclei (plural of nucleus) found in the brainstem, which release serotonin to the rest of the brain.

Reafference The integration of sensory data so that the signals from varying sources match each other in timing.

Receptors Specialized neurons, or parts of neurons, which receive and relay coded sensory information, or which uptake chemical messages from neurotransmitters.

REM/rapid eye movement A phase of sleep where the brain is very active; associated with dreaming.

Rostral/anterior The part of any region of the brain closest towards the front of the head.

RNA polymerase An enzyme that transcribes a gene and determines how it is expressed.

SAD/Seasonal Affective Disorder A condition marked by low mood that corresponds to seasonal lack of daylight and sunlight.

Salience Noticeable, relevant; perceived to be important.

Satiety A feeling of having consumed all that is desired. Usually applied to gratification of appetite.

Second messenger A protein within a cell which responds to some other protein or cellular event by causing a change in cellular function; such as genetic transcription, the activation of other proteins or the influencing of enzymes.

Semantic memory Memory for facts, language, names, etc. Often regarded as a subdivision of episodic/declarative memory.

Senses/sensory Concerned with the uptake and processing of data from external stimuli. Includes vision, hearing, touch, smell and taste.

Sensitization A preferential focus to a repeatedly experienced stimulus, often linked to a conditioned response.

Serotonergic Describes the serotonin system.

Serotonin/5-HT A neurotransmitter involved in the transmission of neural impulses. Involved in mood regulation, the feeling of pleasure,

pain perception, gastro-intestinal function (including hunger and satiety), as well as other physical functions.

Spatial Related to the perception of the position of objects and features in the environment.

Spine In neuroscientific terms, a tiny part extending from the dendritic shaft; specialized to receive synaptic input.

Stimulus/stimuli External event that provides sensory input (stimuli is the plural).

Stria terminalis A band of fibres running from the amygdala, along the surface of the thalamus and the caudate nucleus, to the hypothalamus.

Striatum Part of the basal ganglia; connections between the nuclei of the basal ganglia.

Subcortical The portion of the brain beneath the cerebral cortex.

Sulcus/sulci The crevices between the each fold (gyrus) on the surfaces of the cerebral hemispheres (sulci is the plural).

Superior/dorsal The part of any region of the brain closest towards the top of the head.

Superior colliculi A bump on the lateral part of the midbrain (paired with the inferior colliculi) which receives visual signals from the retina of the eye as well as other regions of the brain. Helps with spatial orientation and the brain's response to sensory stimuli.

Suprachiasmatic nuclei A collection of nuclei in the hypothalamus.

Supramarginal gyrus Part of the parietal cortex.

Sympathetic adrenomedullary system Part of the autonomic nervous system; automatically maintains homeostasis and mobilizes flight or fight responses.

Synapse The site of communication between two neurons, across which either electrical or chemical messages are transmitted.

Synapsin 1 A protein associated with axonal growth, learning, memory, neurotransmitter function and the maintenance of synaptic connections.

Synaptic plasticity Changes in the synaptic connections which link one neuron to another, i.e. which neurons are connected to which other neurons.

Synthesis Production of a molecular compound.

Syntaxin 3 A plasma protein essential for dendrite growth.

Temporal cortex One of the major regions of the cerebral cortex of the brain.

Temporal sulcus Part of the temporal cortex.

Thalamus A structure in the brain which processes most of the information reaching the cerebral cortex in the brain from the central nervous system.

Theta A type of electrical wave created by neuronal activity in the brain, able to be measured by EEG.

Thyroid A large endocrine gland, located in the base of the neck, which helps regulate heart rate and blood pressure, body temperature, metabolism and the rate of biochemical reactions, and growth.

Traits Personality characteristics.

Trophic Providing a nurturing function.

Up-regulation Increased expression of a gene.

Valmet66 A precursor protein to BDNF/brain derived neurotrophic function.

Vascular Relating to blood vessels.

Vasopressin A hormone released by the pituitary gland.

Ventral/inferior A part of any region of the brain lying closest towards the bottom, i.e. closer to the neck than the top of the head.

Ventral pallidum Lies within the basal ganglia, and part of the limbic loop; a pathway involved in motivation, behaviour, emotion and addiction.

Ventricles Four discrete cavities in the brain separating some regions from others while providing a communicating link. Filled with cerebrospinal fluid produced by structures on the walls and roofs of the cavities.

Viscera Large organs in the body, such as heart, lungs and stomach.

Volumetric Indicates size or volume.

Ventral tegmental area A group of neurons in the centre of the brain which comprise the dopaminergic cell bodies of the dopamine system.

White matter The part of the brain that contains myelinated neural fibres. Called white matter because myelin is white in colour. Generally underlies grey matter, although four deep cerebellar nuclei made up of grey matter are contained within white matter.

y-aminobutyric acid See **GABA**.

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