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History of Plant Pathology

Prof. S. G. Borkar

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Preface

Historical facet provides the chronological events in a given discipline/subject till to date and are useful to understand the development and further scope of development in that subject, if it is related to science. The science of plant pathology is made interesting and of in depth understanding when one studies the history or historical events in the development of this subject.

One will wonder, if know, that the defeat of Germany in World War II was due to the epidemic of plant disease known as late blight of potato or Irish famine. The change in drinking habit of Britishers from coffee to tea was due to epidemic of coffee rust in coffee plantation areas which makes the tea to rule the world till date as morning drink over coffee. There are several historical examples of such kinds which caused human starvations, change in food habits, malnourishment and migration around the globe due to plant disease problems.

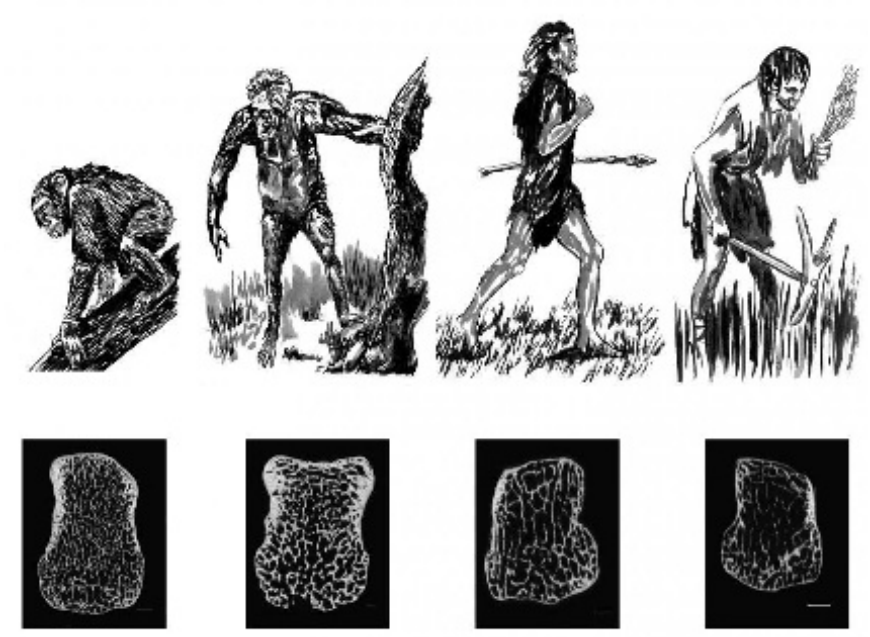
History of plant pathology, thus, makes this subject interesting not only for the students of plant pathology but also to those who wanted to know about this subject. The book entitle "History of Plant Pathology" by Prof. S. G. Borkar includes the development in this branch of science from the ancient era till 20th century and depict the historical events of this science in individual era around the world and its impact on human civilizations.

People say history repeats itself, but the history of plant pathology should not repeat as it causes starvation, agony, subsistence losses not only to the farmers but also to general public around the world. The chronological events of 20th century in the history of plant pathology rather should add new control remedies and management practices of plant diseases.

Prof. S. G. Borkar

1

Introduction



The evolution of human species is considered to be 2.8 million years old (Ghosh, 2015 AD). The three key species viz; *Homo erectus*, *Homo neanderthalensis*, and *Homo sapiens* represent landmarks in the human evolutionary chain, particularly with respect to diet, feeding behaviour, and social development. *Homo erectus* is the first descendant of the (by discovery) *Homo* species, *Homo habilis*. Fossilised remains of the species date back to 1.8–1.0 million years ago which has been found in Africa, Georgia (Europe), China and Indonesia.

The *Homo erectus* employed the use of many stone-based tools to help them in their daily activities. The style of some tools used by them lead archeologists to believe that they were used for cutting, specifically for meat.

This suggests that there was a higher consumption of meat, (which was also supported by the jaw and canine structure of the skulls) which was significantly altered as compared to *Australopithecus*. *Homo erectus* has foraged and gathered non-animal food is not easily determined. It was similar to the activity of *Australopithecus* and chimpanzees, i.e. generally just opportunistic foraging of vegetables, fruits, roots and shoots. Ragir Sonia (2000 AD) suggests that the *Homo erectus* hunted and killed their prey, then took it back to their families to share. Shipman (2000 AD), suggests that the meat was scavenged (obtained from fossil evidence in animal bones that bore a greater resemblance to tooth marks than tool marks). The scavenger theory proves to be more reliable with its archeological evidence. The use of tools, particularly those designed to cut or puncture, emphasises the growing importance of meat in early human society.

Neanderthals, as the species is known, inhabited Europe and parts of western Asia around 3,50,000–1,30,000 years ago. They became extinct in Europe nearly 24,000 years ago. They were built more robustly than *Homo sapiens* and some possessed a large cranial capacity as well. The diet of the Neanderthals is the source of today's popular Paleolithic diet. The emphasis lies heavily on the consumption of fatty meats and vegetables. Many anthropologists suggest that the Neanderthals were hunters, not scavengers, as scavenging would not have fulfilled their meat quota (Richards Michael et al., 2000 AD). Neanderthals were renowned for their hunting and may have hunted their prey by leaping on them or wrestling them to the ground (Wikipedia, 2007 AD). Similarly, amongst their tools, large amounts of weapons have been found in the Neanderthal arsenal, suggesting that hunting was an important way of life. Thus, it was concluded from the above analaysis that the Neanderthals has the growing importance of meat and hunting amongst the early humans which lead to *Homo sapiens*.

Homo sapiens to which the present human beings belongs originated in Africa about 2,00,000 years ago. From then onwards we have grown to cover the entire globe, to a population of about 7.3 billion (as of 2015 AD). For about 1,90,000 years, *Homo sapiens* followed a hunter-gatherer lifestyle, similar to that of *Homo neanderthalensis*. Their diet consisted of the meat, some wild grains, fruits, vegetables and nuts.

In the beginning the *Homo sapiens* were hunters and their food consisted only of meat, leaves, fruits, and seeds, which they picked wherever they could find them. Plant diseases affected leaves and shoots and caused mildew and blight, fruits and seeds suffered form rot, thereby forcing humans to keep looking for healthy fruits or food plants of some kind to satisfy their hunger.

More than 12,000 years ago, man became dependent on plants for their food against starvation. There is evidence suggesting that he was practicing some form of crop/plant culture as early as 7000 BC.

The growing of plant crops around 10,000 years ago marked a significant change in primate nutrition of the mankind and allowed the domestication and flourishing of wild grains. Cultivation of these grain crops allowed formation of human clusters and development of culture.

This development of agriculture resulted in greater class stratification and specialisation of duties, and the delegation of farming and food-harvesting duties to the lower and lower-middle class of farmers. This is the first instance in primate evolutionary history where food is actively grown and commodified instead of being consumed as subsistence for survival. The alteration of feeding patterns, types of food consumed, and increased dependency on single crops has led mostly to social, cultural and political changes in *Homo sapiens* (especially until the 19thcentury).

As humans started settling down and became farmers, they began growing one or other few kinds of food plants in small plots of land and became dependent on these plants for their survival throughout the year. Probably every year, (or in some years) part of the crop was lost to diseases. In such years, food supplies were insufficient and hunger was common. In years when wet weather favoured the development of plant diseases, (large amount of crop or the entire crop was destroyed) resulted in famines, causing immense suffering and probably the deaths of many humans and animals from starvation. It is not surprising, therefore, that plant diseases are mentioned in some of the oldest books available (Homer, c. 1000 BC; Old Testament, c. 750 BC) and were feared as much as human diseases and wars.

The science of phytopathology, like all natural sciences, had its beginning in the dawn of man's civilisation (Whetzel, 1918 AD). All wild plants have diseases and from that time man began to domesticated by cultivating those wild species that suited his needs. He must have necessarily observed and considered the diseases that robbed him of part or all of the fruits of his labour. Not until he acquired the art of writing, he could record his observations and opinions with respect to the maladies which affected his crops. Even long thereafter, records of plant diseases were inscripted but in fragments, woven here and there into his historic or religious writings. Later on, he began to seek order in the multitudinous facts of nature, his observations on plant diseases were segregated more or less along with the related facts and data into his writings of botany and agriculture. Only in relatively modern times there have been attempts to organise the facts and phenomena of diseases in plants into a separate science of plant pathology.

The history of phytopathology divides itself into eras, and these again into periods. Each era is characterised by a general and dominating point of view regarding the nature, cause, and control of diseases in plants. The beginning of each is marked by an epochal change. These changes from one era to the next come as the result of not only accumulation and organisation

of phytopathologic facts and theories, but by revolutionary discoveries in the fundamental sciences on which plant pathology is based. These eras in our science are often almost coincident with great historic epochs. The change from one era to the next with considerable accuracy designates the year or decade at least, which marks the passing of one and the beginning of another. These eras are designated as follows:

1. The Ancient Era (ancient to 5th century)
2. The Dark or Middle or Medieval Era (5th–16th century)
3. The Pre-modern or Autogenetic Era (17th century to 1853 AD)
4. The Modern or Pathogenetic Era (1853 AD–1906 AD)
5. The Present Era (1906 AD onwards).

2

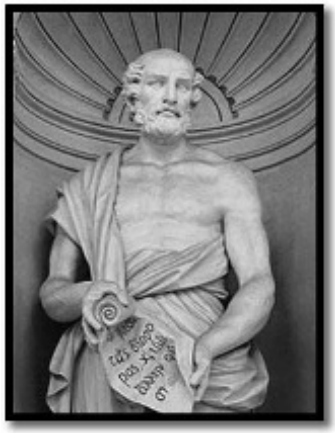
Plant pathology in ancient era

In ancient era, efforts to control plant diseases were hampered by the lack of information on the causes of diseases and also due to the belief that diseases were manifestations of the wrath of God. Nevertheless, some ancient writers, e.g. Homer (c. 1000 BC), mentions the therapeutic properties of sulphur on plant diseases, and Democritus (c. 470 BC) recommended controlling plant

blights by sprinkling plants with the olive crush left after extraction of the olive oil. Most ancient reports, however, dealt with festivals and sacrifices to thank, please, or appease a God and to keep God from sending away the dreaded rusts, mildews, blasts, or other crop scourges. Very little information on controlling plant diseases was written anywhere for almost 2000 years. During the two millennia of fatalism, a few important observations were made on the causes and control of plant diseases, but they were not believed by their contemporaries and were completely ignored by the generations that followed. It was not until about 1200 AD that a higher plant, the mistletoe, was proposed as a parasite that obtains its food nutrients from the host plant, which makes it sick. It was also noted that the host plant can be cured by pruning out the part carrying– the mistletoe. Nobody, however, followed up on this important observation.

The climate and soil of countries around the eastern Mediterranean Sea, from where many of the first records of antiquity came to us, allowed the growth and cultivation of many plants. The most important crop plants for the survival of people and of domesticated animals were seed-producing cereals, especially wheat, barley, rye and oats; and legumes, especially beans, faba beans, chickpeas and lentils. Fruit trees such as apple, citrus, olives, peaches and figs, as well as grapes, melons and squash, were also cultivated. All these plant crops suffered losses annually due to drought, insects, diseases and weeds. Because most families grew their own crops and were dependent on their produce for survival until the next crop was produced the following year, losses of any amount of crops, regardless of cause, created serious hunger and survival problems for them. Occurrences of mildews, blasts and blights on cereals and legumes were mentioned in numerous passages of books of the Old Testament (about 750 BC) of the Bible. Blasts, and more importantly the smut diseases, destroyed some or all kernels in a head by replacing them with fungal spores. Blights and rusts weakened the plants and used up the nutrients and water that would fill the kernels, leaving the kernels shriveled and empty.

Mention of plant diseases is found again in the writings of the Greek philosopher Democritus, who, around 470 BC, noted plant blights and described a way to control them. Aristotle (384BC–-322 BC) reported that the life arose spontaneously from the decomposing organic matter and also the period swept by the thought. It was not, however, until another Greek philosopher, Theophrastus (c. 300 BC) made plants and to a much smaller extent, plant diseases the object of a systematic Study.



Theophrastus

Theophrastus was a pupil of Aristotle and later became his successor. Among others, Theophrastus wrote two books on plants. One called *The Nature of Plants*, which included chapters on the morphology and anatomy of plants and descriptions of wild and cultivated woody plants, perennial herbaceous plants, wild and cultivated vegetable plants, the cereals, the legumes and medicinal plants and their saps. The other book called *Reasons of Vegetable Growth* included chapters on plant propagation from seeds and by grafting; the environmental changes and their effect on plants; cultural practices and their effect on plants; the origin and propagation of cereals; unnatural influences, including diseases and death of plants; and about the odour and the taste of plants. For these works, Theophrastus has been considered the *Father of Botany*. The contributions of Theophrastus to the knowledge about plant diseases are quite limited and influenced by the beliefs of his times. He observed that plant diseases were much more common and severe in lowlands than on hillsides and that some diseases (e.g. rusts) were much more common and severe on cereals than on legumes. In many of the early references, plant diseases were considered to be a curse and a punishment to the people by God for wrongs and sins they had committed. This implied that plant diseases could be avoided if the people abstained from sin. Nobody, of course, thought that farmers in the lowlands sinned more than those on the hillsides. Yet Theophrastus and his contemporaries, being unable to explain plant diseases, believed that God controlled the weather that brought about the disease. They believed that plant diseases were a manifestation of the wrath of God and therefore, avoidance or control of the diseases depended on people doing things that would please the same superpower.

In the 4thcentury BC, the Romans suffered so much from hunger. Due to the cause of repeated destruction of cereal crops by rusts and other diseases, they created a separate God, whom they named Robigus. To please Robigus, the Romans offered prayers and sacrifices with the belief that he would protect them from the dreaded rusts. The Romans even established a special holiday for Robigus, the Robigalia, during which they sacrificed red dogs, foxes and cows in an attempt to please and pacify Robigus so he would not send the rusts to destroy their crops.

2.1 Status of plant pathology in ancient literature

Probably, the development of agriculture started in an ancient era. Chinese practicing crop rotation as early as 3000 BC and in the 1st century BC they were supposed to keep the field fallow for a year (Singh, 2001 AD). Seed health was mentioned in China by Fan Sheng-Chih in the 1stcentury BC. The plant diseases were initially attributed to various reasons viz; divine power, curse of God, religious belief, occultation, superstition, effect of moon and

stars, bad wind, etc. Details of plant diseases along with their management were mentioned in the following major early/ancient literature:

1. Much earlier (about 4700 BC and later) than the time of Theophrastus in the west, Kautilya in India composed the four Vedas (religious verses). These were passed on through word of mouth, in absence of a scripts from generation to generation for centuries. RigVeda (4700 BC) is considered to be the oldest composition of religious hymns. It was followed by Yajur Veda (hymns and rituals), Sam Veda (Rig Veda recomposed for singing) and Arthara Veda (3000 BC and later) which contained charms and spells for warding off evils and diseases. This composition specifically mentioned blight as a disease and its control.
2. 3400 BC: Report of the prevalence of microbes by ancient Greek civilisation.
3. In the pre-Aryan India, the Indus Valley Civilisation or the Harappa Culture (c. 2300 BC) is credited with a fairly well development of grain silos; but there is no positive evidence that the pre-Aryans knew of plant diseases as such. Nevertheless, India's awareness of plant diseases can be traced back to more than 3000years.
4. In the Aryan period, the plant diseases were known. There are specific references to plant pests (insects, worms, etc.) in the Vedas. One cannot be certain if the ancient Aryans distinguished between the damage due to pests and that due to diseases.
5. 1500–500 BC: Symptoms of plant diseases and plant protection were mentioned in Hindu Mythology like Rig Veda, Atharva Veda, Reghuvamsha of Kalidas, Jataka of Buddhism, etc.
6. There are references in the Atharva Veda (1500–500 BC) of worms, insects and other pests, seen and unseen.
7. There was a hymn praying for protection against the lighting injury and an unidentified tree, Sirakthian was used (as a fungicides) against the disease causing blight or white stems in barley and gingelli (Chullavagga, 500 BC-500 AD, Arthasastra of Kautilya, 321–186 BC).
8. In 1100 BC in China and in 600 BC in Assyria: Existence of ergot disease (Holy fire) is mentioned.
9. Homer, c. 1000 BC; Old Testament, c. 750 BC: there is mention of mildew and blasting. Sulphur was reported to control plant diseases.
10. In 700 BC: Roman King Numa Pompilius: Initiated the celebration of annual festival of "Robigalia" to ward-off rust disease.

11. In 470 BC: Greek Philosopher Democritus mentioned blights and suggested control measures and also recommended using olive crush left after extraction of olive oil for controlling blights on plants.
12. Susruta, the great Indian pioneer in medicine and surgery who wrote Susruta-Samhita (c. 400 BC) and Charak, were aware of diseased conditions in plants and often compared them with diseases in man.
13. A phanerogamic semiparasite *Loranthus longiflorus* as a cause of disease was mentioned by Susruta (c. 400 BC).
14. The oldest text on Indian agriculture *Krishi Parashar* was probably written by Parashar (c. 400 BC), before the Arthashastra of Kautilya. His book is considered the first extensive coverage of ancient agriculture of the Aryans. Plant protection is mentioned in one verse with respect to powdery mildew, rust, insects and larger animals as enemies of crops and invokes the Wind God to move them away from his field. Parashar had declared that origin of plentiful yield was the seed, implying seed health.
15. Atharva Veda gave references about chanting of mantras to protect crops and grains from insects such as grasshoppers and animals such as rats. Parashara (400 BC) specifically gives the following mantra for controlling grain destroyer: "Salutations to the feet of the revered preceptor. Let success prevail! The ever victorious feet of Rama (i.e. Rama himself) the Lord of Lords, the Emperor of Emperors, the revered one, commands from his heavenly abode situated on the peak of the Himalayas, the slope of which are white like the conch, the jasmine flower, the Moon-Hanuman, the son of Wind, moving fast like wind, destroyer of invaders, standing on the seashore amidst hundreds and thousands of monkeys with his tail raised and claws harsh and strong, 'Let there be well being.' Winds are blowing with great force in a section of a farm belonging to so and so hailing from such and such family/group. If the destroyers of crops such as gandhi, shankhi, pandarmundi, dhuli, shringari, kumari, madaka, etc., and goats, wild boars, pigs, deer, buffaloes, parrots, sparrows, winged insects, etc., do not leave that farm by your order, you shall strike them hard with your strong tail like thunderbolt."
16. In 370 BC–286 BC: Theophrastus (The Greek Philosopher)first botanist to study in detail and write about the diseases of trees, legumes and cereals; recorded plant morphology, propagation, effect of climate on plant growth, etc., and also plant diseases in his books entitled *Enquiry into plants*, *Reasons of vegetable growth* and *The Nature of Plants*. He is also known as the "Father of Botany". His approach was observational and speculative rather than experimental.

Theophrastus was disciple of Plato (428BC–348 BC) and Aristotle. Although most of the work of Theophrastus is lost, two of his books, *Historia Plantarum* and *De Causis Plantarum*, still find a place as reference. In his writings, Theophrastus elaborately mentioned plant diseases (rusts, mildews, blight, etc.) but expressed the opinion that these diseases were due to bad nutrition and bad air.

17. Although, Susruta did not deal with plant diseases, he wrote "The proper season, good soil, water and vigorous seeds produce a healthy plant".

    Phanerogamic parasites: Dodder (*Cuscuta reflexa* Roxb.), as a parasite has been mentioned in *Bhavaprkashanighantu* (Pandey and Chunekar, 1999 AD).

18. In the Indian subcontinent, more than three centuries before the Christian era, during the Mauryan Empire (c. 320 BC), agriculture was fairly developed and was the main concern of the government.

    Kautilya, (also known as Chanakya) and Vishnugupt, had written (c. 320 BC–296 BC) the classic treatise Arthashastra (Science of Source of Livelihood) which incorporated many observations and recommendations for healthy crop culture and listed treatment of seeds in addition to recommendations for punishment against sale of spurious seeds. Interestingly, Kautilya had recommended that seeds should be left in open for 7 or more nights and days. Some other recommendations of Kautilya that can be viewed in the light of modern science included (i) sugarcane seed cutting end should be treated with curd and honey and pasted with cow dung and (ii) cucurbits should be cultivated on riverbeds during summer, a practice that has sustained for more than 2000 years and is still followed. These composers and writers belonged to the north-west region of the Indian subcontinent. Others (Varo, 116BC–27 BC; Maro, 70BC–-19 BC) during that time did mention specific treatments of seed.

19. In the south, the Tamil poet Tholkappier (200 BC) considered plants as living beings, mentioned monocot and dicot plants, and wrote about benefits of rice–legume rotation.

20. Indian ancient books which mentioned the diseases/enemies of plants are

    (a) Artha Shashthra of Kautilya: 321BC–186 BC

    (b) Sushruta Samhitha: 200AD–500 AD

    (c) Vishnu Purana and Brihit Samhita: 500 AD

(d) Agnipurana: 500AD–700 AD

(e) Vishnudharmottara: 500AD–700 AD

21. Lord Pliny (100 AD) described plant diseases and suggested some remedies. He believed that diseases originate from the plants or from the environment. Mention of plant diseases are in Bible, Shakespeare's poems and plays, several Christian and in other Indian ancient literature.

22. The learned men during Vedic period were aware that the diseases are caused by microbes, blight, powdery mildew, rust and tumours on trees, fungi (mushrooms) and algae are also mentioned in the Vedas. During the Vedic period agriculture in India was fairly developed. Ploughs and other agricultural implements were in use.

23. In Rig Veda, not only the classification of plant diseases has been given but the germ theory of disease was also advocated. (Nene, 2003 AD). Also a number of verses are devoted as prayer to the Sun God for purifying and protecting everything and for destroying the tiny, invisible creatures that poison the food and cause diseases, vigorous and clean seed was recommended for planting. Obviously, 1000years before the time of Theophrastus, the civilisation in India as mentioned in Vedas was aware of living beings (*krimi*) that caused disease in man, cattle and plants, and sun heat and fire kill these poisonous creatures.

3

Plant pathology in medieval era

The medieval era starts after the fall of the Roman Empire (about 500 AD) to the discovery of America (1500 AD); and is known as the Dark or Middle Era in the history of plant pathology. The writings of this period show little that is new regarding plant diseases. This was the period when science and learning may be said to have slumbered, hence it is not strange that so fragmentary and unorganised knowledge, as phytopathology, at that time, should have had few additions even in the form of isolated observations. There is, however, one bright spot in the universal darkness, and that is the work of the Arabian

gentleman and agricultural encyclopaedist, Ibn-al-Awam (Whetzel, 1918 AD) who lived and wrote during the 10th century at Seville, Spain. Thoroughly familiar with the writings of Theophrastus, Pliny, and other ancient writers on agriculture, including those of ancient India, he was nevertheless an independent observer and thinker. His phytopathological comments are almost entirely on the diseases of trees and the vine. He describes the symptoms of many diseases briefly but with accuracy and gives extensive consideration to their control (Savastano, 1890 AD).

With the rapid decline of Roman governance, Catholic Christianity became the leading cultural force. During the first few centuries (about 400 AD–1200 AD) – often referred to as the Dark Age – little was accomplished. Populations decreased, agricultural lands were overrun by hostile armies and economics stagnated. But in other parts of the world, notably the near and far east, rich civilisations flourished. The revival of learning in medieval Europe had to wait till the 12th and 13th centuries. Partly through the rediscovery of ancient knowledge, partly through an improvement of economic and social conditions, science and technology began to advance. Towards the end of the middle age the centre of political, cultural, and scientific influence, formerly centred around the Carolingian Empire, shifted to the maritime nations of southern and western Europe, eventually leading to the discovery of new continents and unknown cultures (Orlob, 1971 AD).

Plant diseases as we know them today existed as the very fringe of medieval interest, and as an inevitable consequence, the plant pathological literature remained small.

3.1 In Europe

When the middle age began to take shape, a compendium of Byzantine agriculture, known as the *Geoponica* (probably written in ca. 650 AD, rewritten in ca. 950 AD), appeared (Cassianus, 1895 AD). Since the book contains material dating back to antiquity, it will not be mentioned here except for disease control, in which it was influential during the middle age.

The interest of the Romans in agriculture and natural history was marked as the last contribution of antiquity to early plant pathology. Palladius (ca. 330 AD) wrote a treatise on agriculture that was used by several medieval writers (Palladius, 1529 AD). Pliny (23 AD–79 AD), on the other hand, had produced an exhaustive encyclopedia in which he included a great deal of plant pathology. The tradition of encyclopedias was continued during the early middle age by such writers as Isidore of Seville (570 AD–636 AD), Rhabanus Maurus (776 AD–856 AD), the Venerable Bede (679 AD–735 AD), Alexander of Keckham (1157 AD–1217 AD), and Bariholomaeus Angkcus (ca. 1250),

but their books contained little or no pathological material. More often than not these writers were armchair naturalists with no other ambition, but to put nature in a dogmatic straight jacket (Orlob, 1971 AD).

By the 12th century a different kind of naturalist emerged who was more sensitive to the ways of nature. Vincent of Beauvais (1190 AD–1260 AD), for example, complained about the plight of the writer who was burdened with the many contradictions of ancient sources, and he admonished the reader to use his own judgment in sorting out the many complexions of men and animals and fruits (Beauvais, 1954 AD). The medieval naturalist was typically a member of the clergy or a monastic order who had maintained a keen interest in medical and agricultural botany. Two early contributions, Strabo's (808 AD–849 AD) Hortulus and Meung's (11th century) *Macer Floridus,* are landmarks of medieval botany, but again contain nothing that needs to be mentioned here (Gottfried, 1470 AD).

There are, however, several examples where the men and women of the church wrote about plant diseases. At the very beginning of the Christian era, the Byzantine church father Basil the Great (330 AD–379 AD), described a cereal disease recognisable as smut (Basilius, 1354 AD). More detailed were the botanical writings of Hildegard of Bingen (1098AD–1176 AD), a woman of extraordinary intellect but also of wild imaginative faculties. She developed a uniform system of pathology where all diseases of men, animals, and plants were attributed to cosmic factors. Each zone of the macrocosmos can emit disease in the form of fiery scales or vapors that blight and defoliate plants (Bingen, 1150 AD). To underscore such dramatic effects, the illustrator of one of Hildegard's codices created what seems to be the first illustration of sick plants (Bingen, 1170 AD).

Religiously oriented prescriptions are difficult to separate from the magical-superstitions. Caesarius of Heisterbach's (1180 AD–1240 AD) collection of contemporary anecdotes tells of a woman who, caring more for her garden than for the sacraments of the Church, crumbled up the host received at Holy Communion and sprinkled the crumbs over the cabbage to eliminate the plague (Heisterbach, 1851 AD).

The practice of seed treatment, sometimes mentioned in early literature, might go back to animistic ideas (Buttress *et al.*,1947 AD). Best known among the animistic traditions were the human sacrifices of the Aztecs, but these functioned primarily as rain-making rituals (Sahagun, 1950 AD). In several other countries, however, human sacrifices were symbolically expressed or really executed in connection with growth rituals. In parts of Europe, a straw man was burned on Easter Eve and the ashes scattered over the crops to protect them against blight and other maladies (Frazer, 1967 AD).

Less original and largely based on the agricultural book of the Roman Palladius (Palladius, 1529 AD), was the writing of Albertus Magnus (1193 AD–1280 AD), a Dominican of universal knowledge (Magnus, 1867 AD). He attributed a few plant disorders to functional deficiencies, but was primarily concerned with the control of pests.

Only a few manuscripts exist that can attest to the state of medieval agriculture. Certainly the most famous is the *Ruralium Commodorum,* a comprehensive text on virtually all aspects of agriculture, composed by Petrus Crescentius (1230 AD–1320 AD) of Bologna. Petrus plant pathology was based again on Palladius and presented nothing new (Petrus 14th century). The main merit of the book must be sought at a different level. A work that mentioned plant disorders and was the first agricultural handbook to be printed and translated into different languages was bound to spread the general awareness of plant diseases (Orlob, 1971 AD). The examination of an early 17th century edition of the *Ruralium Commodorum* found its plant pathology considerably expanded (Orlob, 1971 AD; Petrus, 1602 AD). Apparently editors and translators added to the original contents of the work and kept its usefulness up-to-date (Orlob, 1971 AD).

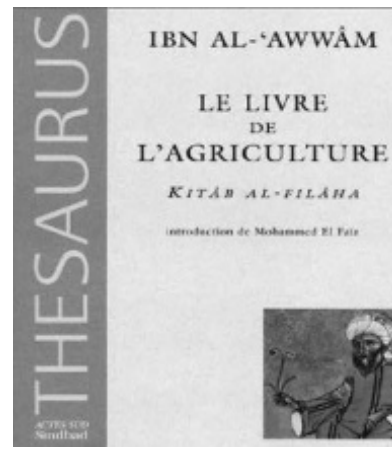
Of similar importance for early plant pathology was the *Pelzbuch* (book on grafting) of Gottfried of Franken (ca. 1350). Gottfried was primarily concerned about tree disorders, citing remedies from ancient or contemporary sources, and presenting his material plainly and forcefully (Gottfried (ca. 1470)). Like Crescentius' book, Gottfried's work was influential until the early 18th century. Perhaps the most original work of the European middle age was Konrad of Megenberg's (1309 AD–1374 AD) book on nature (Orlob, 1971 AD). As the title indicates, natural history rather than agriculture was the main theme. Plant diseases were incorporated in a comprehensive chapter on metereological phenomena treating such topics as rain, hail, thunder, honeydew, etc. (Konrad (ca. 1388)). Konrad's idea to relate plant diseases to the skies and weather was not new as Greek and Roman writers had alluded to this many centuries ago but his thorough description of mildew (more likely rust), its causes, nature, and terminology was the best that the middle age produced (Orlob, 1971 AD).

Additional phytopathological material can be found in the meteorological manuscripts of the 14th and 15th centuries (Orlob, 1971 AD). Storms, heavy rains, hail, and lightning were recognised as important crop-damaging agents (Dales, 1965 AD; Hellmann, 1904 AD; Sahagun, 1950 AD). Closely interwoven with the meteorological literature were astrological predictions that tried to foretell not only the fate of man, but also the welfare of his crops (Codex 2817 (14th century); Codex 317 (15th century)). Theological literature mentions plant diseases (ergotism) in prayer books (The Master of Mary of Burgundy, ca. 1480).

3.2 In central America

The discovery of America brought some additional material to light. In 1569 AD the Spanish friar Bernadino de Sahagun (1500 AD–1590 AD) completed an encyclopedia of Aztec culture, the famous *Florentine Codex.* Sahagun had obtained his information from pre-conquest sources and narrations of surviving Indians. What he tells us about plant pathology in early Mexico resembles much to what we know from other early civilisations (Sahagun, 1950 AD). The agricultural gods ruled the fields, acting through the intermediary of meteorological agents. In the arid climate of the Mexican highlands timely and sufficient rains were the most important growth factors. Other components of the weather were related to poor growth or disease, but the most interesting reference for the plant pathologist concerns two fungal diseases of corn, smut and ear rot.

3.3 In the Arabia



The advance of the Arabs into Spain had important consequences for medieval plant pathology. While most of Europe paid no attention to plant diseases, the Arabs were well aware of plant-health problems and recorded them in their literature (Orlob, 1971 AD). The great compiler of Moorish plant pathology was Ibn Al-Awam of Seville (12th century). In Chapter 14 of his agricultural treatise *Kitab al-Felahah,* various disorders and diseases of fruit trees and grapes were discussed at some length and several remedies recommended (Ibn Al-Awam, 1864 AD; Orlob 1971 AD). Unfortunately, the poor and repetitious organisation of the subject matter distracts from the overall value of the book. Other Arabs wrote about plant diseases too. Ibn al-Bassal (12th century), for example, mentioned the *germs of harm* that reach the tree and poison it (Ibn al-Bassal, 1955 AD).

More than many other people, the Arabs were masters of the magical arts. Astrology and alchemy prospered and advanced to semi scientific levels. The health sciences, too, were deeply dealt by magical concepts, as documented by many miraculous recipes. An important issue, although unrelated to disease control, was how to destroy crops or trees of an enemy. One source says that all plants struck with an axe immersed in goat's blood will cease

growing (Ibn Quayba, 1949 AD). Another recommends drawing a tabismanic figure on a plate, exposing it and tying it in desirable area and "the planetary judgment will descend and nothing will grow in this location as long as the figure remains" (Pseudo-MagrL, 1962 AD).

It is more difficult to ascribe a certain rationale to the many remedies in the literature. It is easy to spot remedies based on magical concepts. The principle of sympathetic magic entered the healing arts as the therapy of opposites. Ideas of sympathy and antipathy, for instance, seem to be behind Ibn Al-Awam's suggestion to control leaf blisters of peach (thought to be caused by hot manure) by exposing the rootstock and adding clay, which was considered cool and moist (Ibn Al-Awam, 1864 AD). The advice to prevent rust by fumigation could have a similar background because of an alleged antagonism between smoke and airborne agents (Magnus, 1867 AD). The smoke from herbs and other aromatic substances was already used in antiquity as a general disease preventive (Basilius, 1354 AD).

3.4 In the far east

In the far east little attention has been paid to the development of plant pathology outside the western world. Although the history of Asiatic science was accessible only to readers of the native scripts, a limited search revealed that the far east had accumulated knowledge of plant diseases in no way inferior to that of the west. The Chinese were expert agriculturists and mentioned plant diseases in some of their works (Han Yen-Chih, 1923 AD; Orlob, 1972 AD). Two Indian manuscripts on botanical and agricultural matters contain chapters on plant diseases. These sources, although of unknown age, seem to relate to the traditional beliefs of medieval India. Interestingly, plant diseases were approached with the concepts and terminology of medicine (Visva-Vallabha 1564 AD; Vrakysayurveda 1964 AD; Orlob 1971 AD).

The greatest difficulty in assessing the occurrence of specific plant diseases was the concept of disease specificity in general and of the specificity of plant diseases in particular which was foreign to the medieval mind. Different symptoms, if noted at all, were not related to different causes; crops or trees were simply sick and one or more major causes were held responsible for this. A clear recognition of leaf discolorations, wilts, or rots was also overshadowed by the facts that rustics were primarily concerned with the more dramatic and readily identifiable effects of frost, hail, floods, and insects.

British records kept between 500 AD–1500 AD, list 124 crop failures and all were attributed to impendent growing conditions or pests (Orlob, 1971 AD). A few examples:

984 AD: a summer of draught and heat with consequent damage to corn

1087 AD: a great famine following severe storms

1201 AD: a wet year where much hay and corn was spoiled

1248 AD: a plague of worms and grubs caused much damage to fruit trees.

It was not until 1780 AD that the same collection explicitly mentions a plant disease, stating that the crops were attacked by mildew and that the harvest, therefore, was light (Stratton, 1969 AD). The situation in other parts of the world was probably not much different. In medieval China frequent famines resulted from irregular rainfall and botanists recommended wild plants for use in such emergencies (Needham, 1968 AD). The Aztecs of Central America even kept smutted corn for similar purposes (Schultze, 1950 AD).

While calamities caused by adverse weather received the greatest attention, other disorders such as barrenness, poor growth, and untimely aging were described in some detail (Magnus, 1867AD; Cassianus. 1895 AD; Franken, Gottfried (ca. 1470); Ibn Al-Awam 1864 AD; Visva-Vallabha 1564 AD; *Vrakysayurveda*, 1964 AD). Then there were two problems that medieval writers picked up from ancient sources and perpetuated them with stubborn persistency. The first related to allelopathy, the influence of one plant on another. Crops were supposed to grow well when the sympathetic principle prevailed. But as soon as a plant growing close to another species suffered, antipathy was to blame (Magnus, 1867 AD; Petrus, 14th century; Gottfried (ca. 1470); Ibn Al-Awam 1864 AD). The second problem was to deal with transmutation, the change of one plant (e.g. barley) into another (e.g. darnel). Yields decreased and poor crop quality resulted (Magnus, 1867AD; Basilius, 1354 AD; Ibn Al-Awam, 1864 AD, Ibn Quayba, 1949 AD; Pseudo-MagrL, 1962 AD).

Mistletoe was the first plant pathogen to be recognised as such and the first pathogen for which a cultural control (by pruning affected branches) was recommended, by Albertus Magnus around 1200 AD, a great deal has been fantasised, said, written, and practiced about it than its importance as a pathogen would indicate. The striking visibility of true mistletoes on deciduous trees, and their ability to remain green while their host leaves fall for the winter, excited the imagination of people since the times of the ancient Greeks and inspired many myths and traditions involving the mistletoe plant through the centuries. The plant itself was thought to possess mystical powers and became associated with many folklore customs in many countries.

When it comes to infectious plant diseases, only scattered and disputable records exist. Needless to say, it is the actual description of a disease rather than its name that interests us here. Most disease names seem to have been, collective names much like blight and plague, and probably embraced the most heterogeneous diseases (Orlob, 1972 AD).

The rust-like diseases, usually described under the names of *erysibe, rubigo,* or mildew, existed in the medieval literature as a hold-over from antiquity (Magnus, 1867 AD; Petrus, 1602 AD). On the other hand, it can be quite certain that rust occurred, and it was only a good diagnostic description that was lacking.

The mildews, like the rusts, were confused with other diseases and no reliable records of them could be found even though the name mildew was occasionally used (Megenberg, (ca. 388)).

The smuts were more diagnostic than either the rusts or mildews. Basil the Great (Ilius, 1354 AD) made a good description of what seemed to be cereal smut (". . . the kernels are burnt and change their color and taste"), while the Aztecs recognised corn smut and linked it to a fungus (Schultze, 1950 AD).

Corn ear rot was another disease known to Aztec farmers. They had observed that a fungus develops on green or dried ears and that these become moldy and rot (Schultze, 1950 AD).

Peach leaf curl seems to have been the only infectious disease described by Ibn Al-Awam, if we take the shrinking and curling of leaves that he mentions as sufficient evidence (Ibn Al-Awam, 1864 AD).

Ergot was of outstanding importance in the middle Ages. Surprisingly, the first description of ergot sclerotia in cultivated grain did not appear before 1582 AD (Uarger, 1582 AD). It is only through the deadful human disease known as Holy or St. Anthony's Fire that we know of the widespread occurrence of *Claviceps purpurea,* in the middle age (Fuchs, 1834 AD).

It is evident that this listing cannot be taken as an undisputable proof for the occurrence of any particular disease. It is also clear that infectious plant diseases were not among the most important issues of medieval societies and that their impact was greatly underestimated. This is in contrast to human maladies, where good descriptions exist for such diseases as rabies, smallpox, typhus, bubonic plague, leprosy, and syphilis (Henschen, 1966 AD; Talbot, 1970 AD).

Ancient plant pathology, especially in the works of Theophrastus (370 BC–287 BC), taught that disease is due to internal or external causes and it should be regarded as a disturbance of the normal life processes of the plant. Medieval writers did not bother much about such subtleties, and usually left the causation and nature of disease untouched. Nevertheless, a few causal agents were mentioned from time to time. Among these, celestial-meteorological agents clearly were the most popular (Orlob, 1964 AD.) Perhaps they were best suited to accommodate the drive for a supernatural-anthropocentric interpretation of natural phenomena.

One proponent expressed it thus. Several noxious vapors exist "according to God's will." One kind is created by the moisture of the waterways and affects man and animal without killing them. However, this vapor kills the buds of trees, damages fruits, and discolors leaves (Bingen, Hildegard, 1911 AD). Conrad of Megenberg, related the cause of a disease he calls mildew, to a similar phenomenon. "The vapor that creates the mildew is very fine and burned by the sun which at the same time makes it rise into the air. If this vapor suddenly changes into droplets it falls upon the crop . . . and will burn the fruitful substance in the same way as hot ashes ..." (Megenberg, (ca. 1388); Orlob,1971 AD).

To medieval men, nature and causation of diseases revolved around two major principles: first, disease as the result of sin, as God's punishment; second, disease as test of human endurance, even as a reward and blessing (Probst, 1969 AD). Agricultural mythology and folklore seem to have flourished throughout the middle age, even though much of this surfaced only during subsequent centuries (Orlob, 1972 AD). An example to show how the diseased plant entered agricultural lore, is that several European countries had their tales of corn spirits, which were believed to glide through the fields and as their gowns touched the growing grain the heads turned black (Frazer,1967 AD).

In the New World – then still undiscovered – the Mesoamerican Indians recognised plant disorders. The Aztecs not only developed an elaborate farm religion but were also aware of the intimate interaction of plants with their environment. The rustic deities of a picture codex are shown to determine plant growth and health in conjunction with weather, soil, and pests (Mayer, 1901 AD). Another insight into natural phenomena was documented in the post-conquest *Florentine Codex* (Sahagun, 1950 AD), mentions two developmental stages of corn smut, an early stage called maize fungus (probably because the galls of corn smut resemble puff balls), and a late smutty stage. Unfortunately the causes of these diseases were not mentioned.

A monograph on oranges written by Han Yen-Chih (ca. 1170) states that a fungus or moss appears on the branches of old trees and that the fungus will spread and absorb the sap of the tree unless it is removed (Han Yen-Chih (ca. 1170), 1923 AD). Another source depicts a mushroom on a pine tree, explaining that the fungus is the *guest*, the tree the *landlord* or host as we would say today (Wang et al., 1918 AD). All this suggests that Chinese were familiar with the concept of plant parasitism.

Graeco-Roman plant pathology was primarily concerned with explaining the causes and nature of plant diseases while saying little about how to control them. This trend was reversed during the middle age. Medieval plant pathology consisted essentially of a long list of remedies for the prevention

or cure of various ills. As long as the actual causes of plant diseases remained unknown and field observations were not in vogue, disease control could make no headway. Not surprisingly, the remedies usually belonged to the realm of quackery, sanctioned more by tradition than by reason. Until the early 19th century the farmers' methods of controlling plant diseases were essentially those of medieval agriculture (Orlob, 1971 AD).

Probably one of the most influential, and certainly the most typical, work of medieval plant disease control was the *Geoponica,* an agricultural text based primarily on Hellenistic sources and the lore of husbandmen. The many recipes scattered throughout the book are interesting primarily for their quaintness rather than their potential effectiveness. A few examples may illustrate this (Cassianus, 1895 AD). Against rust, burn three crabs together with cuttle-fish. If fruits begin to rot treat the roots with ashes and vinegar. Against barrenness, split the roots and insert a stone or wedge. Transplants should be dipped in ox gall and the young trees will remain healthy. To prevent grain diseases and pests, strain the seed through perforated seal skin prior to sowing. Despite these and other absurdities, the *Geoponica* gives a few sensible recommendations. Manuring and change of soil were prescribed against unfruitfulness, barrenness, or old age (Cassianus, 1895 AD).

Like the *Geoponica,* Ibn Al-Awam's *Kitab al-Felahah* revolved around prophylaxis and cure. Most commonly, compounds such as vinegar, urine, ash, oil drugs were recommended against the various ailments of fruit trees and grapes (Ibn Al-Awam, 1864 AD). Scarification, i.e. cleaving roots or stems (to allow removal of bad sap) was frequently mentioned.

A major collection of remedies and cures appeared in Gottfried's of Franken *Pelsbuch* (Franken Gottfried, 1470 AD). For the most part the treatise resembles the *Geoponika*, except that it is somewhat less dogmatic and more open to new impressions. Gottfried was a practical man, well versed in the art of grafting, pruning, and tree nursing, and this is reflected in his tree pathology. For instance, against canker he recommends scraping off the diseased part clear down to the green wood, then dressing it with cowdung (Franken Gottfried, 1470 AD).

If one tries to sort out medieval control techniques according to methodology, chemical and physical procedures can be differentiated. Examples of some commonly used compounds have already been mentioned, but none of these seem to have possessed proven fungicidal properties. As to physical methods, scarification and tree surgery were often cited. Next, prophylatic and curative measures were recommended, but not clearly differentiated, usually the same treatment was suggested for either approach. The same applies to the treatment of different disorders; similar compounds or procedures were prescribed for a variety of conditions.

What was the reason for the unproductiveness of medieval plant pathology? It would be most convenient to blame the spirit or conditions of the period. However, as pointed out before, the middle age were not all that bad. Other areas, notably the medical and veterinary practice, expanded, if not through great scientific achievements then certainly through their growing importance (Davidson, 1967 AD). Furthermore, if the age was to blame, plant pathology should have blossomed in the 16th century Renaissance along with other sciences. Instead it remained almost at medieval levels for the next three centuries.

The main reason for the unproductiveness of medieval plant pathology, especially in the area of infectious diseases, was the difficulty of the subject matter that confounded medieval, Renaissance, and 17th century plant pathologists alike. Any rational being, unversed in pathology and confronted with a situation where plants suddenly changed their appearance and slowly declined, would be hard pressed to explain the cause of the abnormality or suggest a useful control. Whatever could be obtained from justifiable or unjustifiable reasoning has already been put down in antiquity. What was desired next was a growing familiarity with the real nature of plant diseases, the existence of infectious and invisible agents, and the life process of plants. But this required insights, scientific skills, and technical know-how which were unavailable for some more time.

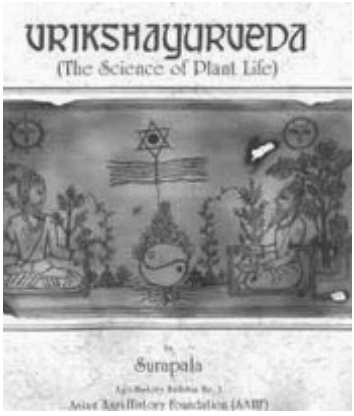
Medieval plant pathology was neither worse nor better than that of any other early period (Orlob, 1971 AD). It served the needs and aspirations of farmers and fruit growers with a host of primitive remedies and folklore. It was not until the late 18th century that the medieval approach to plant health problems gave way to a new phase of plant disease studies. This period was molded by men who combined a critical spirit with the belief that nature had to be consulted. The diseased plant, not the classical text, became the arbiter of plant pathology (Orlob, 1971 AD). Also in 1440 AD printing was introduced in Europe and this reflected interest in learning science.

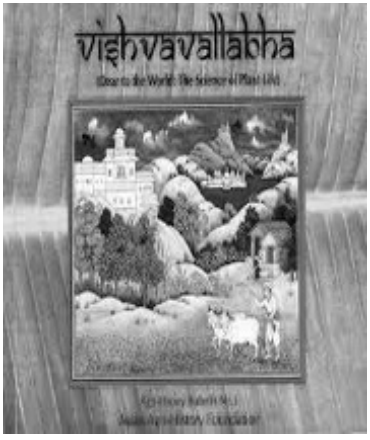
3.5 In India

Outside the western world, the most exotic remedies seem to have been used in India where elephant milk, tiger meat broth, perfumed water, honey, herbs, etc., were prescribed (Visva-Vallabha, 1564 AD, *Vrkysayurveda*, 1964 AD). The Indian recipes acquired a degree of sophistication, mainly due to the pseudo medical interpretations of plant diseases. Perhaps all health sciences were in the hands of the local wise man who administered both to sick people and diseased plants.

In addition, some Indian ancient literatures got compiled during the medieval era describing the plant diseases and their management practices. Dwivedi (2013 AD) in his work, *Thoughts for Plant Pathology in Sanskrit* literature published the translation of these ancient literatures and under the leadership of Y.L. Nene in the journal *Asian Agri-History* which publishes very educative and interesting articles related to the history of agriculture named *Crop Diseases Management Practices in Ancient, Medieval, and Pre-Modern India*.

The term *Vrikshayurveda* has been in use since ancient times. By the time Kautilya (296 BC–321 BC) compiled his *Arthasastra*, the term *Vrikshayurveda* was well-established and well-known. The document Vrikshayurveda, a very brief one, was compiled as *Brahat Samhita* by Varahamihira (505 AD–581 AD), *Sukraniti* was composed in around 8th century. The two texts compiled in the 11th century AD were Surapala's *Vrikshayurveda* (ca.1000) and *Vrikshayurveda* chapter in *Lokopakara* composed by Chavundaraya (1025 AD). In the 12th century AD, Chalukya King, Someshvardeva compiled an encyclopedia *Abhilashitarthachitamani* or *Manasollasa* in which a full chapter on *Vrikshayurveda* was included. In 13th century AD a text titled *Upavanavinoda*, was composed which deals with landscape gardening. *Upavanavinoda* was compiled by Sarangadhara, a courtier and scholar in the court of King Hammira.





In the court of great Maharana Pratap, a scholar, Chakrapani Mishra, compiled (ca. 1577 AD), adding his own experience, the text *Vishvavallabha*, which has contents similar to Surapala's *Vrikshayurveda*, with a good deal of additional information. There are controversies regarding the dating of *Krishiparasara.* It might have been composed anytime from 6th to 10thcentury AD. Chronologically the last text available is *Shivatatvaratnakara* (in Kannada) compiled by King Basavaraja of Keladi, now in Karnataka; it has a chapter on *Vrikshayurveda*.

On going through the *Sanskrit* literature, it becomes clear that the plant diseases and pests were well recognised by ancient Indian seers. They developed

eco-friendly ways of crop cultivation and organic agents as pesticides. Now modern agricultural science also accepts that traditional agronomic practices, organic manure and pesticides are far better than the modern synthetic chemicals. The great ancient Indian scholars have made valuable contributions to plant pathology. One can find references to the diseases of the plants and their treatment in Sanskrit texts. On any living beings, plants or animals or humans, general health and vitality is the first line of defence against disease. However, it is a fact that the plants do undergo ailments and diseases caused by natural forces and other aliens like pests and animals. The *Sukraniti* makes it clear that grains get spoilt by poisons, fire or snows or eaten by worms and insects.

Various Sanskrit texts contain chapters dealing with diseases and treatment of plants.

While discussing the diseases of the plants, Sanskrit texts came up with the fact that the diseases are basically of two types. They are as follows:

1. Internal diseases – These occur due to imbalance of doshas *(Vata, Pitta, Kapha)* and further divided into three types.
    a. *Vataj – Vataj* diseases occuring due to dry land.
    b. *Pittaj – Pittaj* disease occur when tree is watered with acidic and salty water.
    c. *Kaphaj – Kaphaj* disease occur when tree is watered with sweet and cold water normally in the spring and winter season.
2. External diseases – These occur due to worms, insects and unfavourable environment.

Varahamihira makes note of the natural causes of diseases. He narrates that trees get disease from cold weather, strong winds, and hot sun. Consequently their leaves become pale-white, sprouts scanty and sickly, branches dry and their milk oozing out.

Kasyapa was of the view that lack of the branches, stems, foliage, fruits and leaves loosing lustre gradually are the diseases of plants caused by cold, heat, rain, wind, roots of the trees being intertwined by the roots of other trees growing very close to it and elephants rubbing their temples against the trees.

3.5.1 Internal diseases and their symptoms

1. Vata diseases
    a. Stem becomes thin,
    b. Knots appear on leaves and stem and

c. Fruits become hard, less juicy and less sweet.

2. Kapha diseases

   a. Tree takes long time in bearing fruits,

   b. Paleness develops,

   c. Leaves are twisted,

   d. Improper growth of fruits and

   e. Juicelessness of fruits.

3. Pitta diseases

   Pitta generated diseases occur in summer, and on the arrival time of clouds when the trees are watered with bitter, sour, strong and salty things. Diseases caused due to imbalance of pitta are:

   a. Untimely yellowness of leaves

   b. Untimely falling of fruits

   c. Dryness of trees,

   d. Paleness of leaves, flowers and fruits and

   e. Decay.

   Sometimes the plants witness imbalance of all the three components i.e. vata, kapha and pitta. If so happens plants fall prey to "Pandu" disease. Surapala says that in some trees the balance of pitta, vata and kapha get disturbed and all these dosas lead to pandu disease of the plant.

4. Symptoms of pandu diseases

Due to this disease, paleness develops in fruits, stem, and branches of the affected trees.

3.5.2 External factors leading to diseases of plants

Vrksayurveda has discussed in length about the external factors that cause diseases in the plants. They are mentioned as below:

1. Attack of insects

   Sometimes trees start drying up due to the attack of insects. Due to this the leaves of the tree become pale.

2. Scorching heat

   Scorching heat also results in fading of leaves.

3. Furious winds

Sometimes due to furious winds trees are broken, uprooted and twisted. On several occasions cracks develop in them and they get broken up in two parts

4. Exposure to fire and lightning

   The trees are dried due to exposure to fire, lightning, dryness of soil and absence of water.

5. Striking by axe:

   The trees also go dry or their development is adversely affected when they are struck with an axe and injured.

6. Mistaken treatment

   All the trees become unfruitful due to faulty seeds, lack of treatment and mistaken treatment. They develop bareness and loose the capacity of production.

7. Excessive irrigation and attack by the ants

   Sometimes due to excessive irrigation (watering) indigestion develops within the tree. Due to attack by the ants, roots of the tree start suffering from bad smell. Dwarfing and twisting of tender leaves take place.

8. Wind, friction, shade and weeds

   Due to fire, wind, friction with other trees, staying in constant shade of other trees, becoming inhabitation of numbers of birds, growth of many creepers and weeds nearby; the development of trees are obstructed and they get damaged.

9. Hailstorm

There is reference of a crop in the Kuru country had been destroyed by hailstorms.

3.5.3 Treatment of diseases of plants

The ancient seers not only discussed the causes of the diseases, but also came out with number of measures to cure these diseases.

Surapala has pointed out that treatment is to be done in a careful manner keeping in mind various factors that led to the diseases of the trees. He says that treatment of trees should be done after examining the root causes of the diseases. Treatment should start only after proper diagnosis. Only those are to be contacted for treatment, who, are well-versed in the concerned field and have high intellectual capacity. Only with the consultation of such personalities, treatment is to be started.

The composer of the *Brahatsamhita*, Varahmihira has also discussed the matter more or less in the same manner. He says that after the trees show symptoms of disease, then the trees should be treated. At the outset, the trees should be cleared of ulcers and the like (i.e. whatever is colourless and wet) with a knife. Then a paste made of *Vidanga,* ghee and silt must be applied to those parts and they should be sprinkled with water and milk. If this is done they will be free from disease.

3.5.4 Treatment of plant diseases caused due to internal factors

1. Treatment of vata generated diseases

   For treating vata generated diseases, trees should be treated with the mixture of Aristha (horn of cows, hair of horses, sana, ghee, sisumara oil and marrow of Ankola).

2 Treatment of Kapha generated diseases

   Surapala has recommended various treatments for the kapha generated diseases.They are as follows:

   a. Use pancamula for treating kapha generated diseases.

   b. The kapha generated diseases should be treated with bitter, strong, and astringent decoctions made out of *panchamula* with fragrant water.

   c. To do away with kapha generated diseases in plants, the paste of white mustard should be deposited at the root and the trees should be watered with a mixture of sesame and ashes.

   d. Soil around the roots of the tree should be removed and replaced by new and dry soil.

3. Treatment of pitta generated diseases

   Cool and sweet substances are recommended for pitta generated diseases.

   a. Decoction of milk, honey, yastimadhu and madhuka is also recommended for treatment of pitta generated diseases.

   b. Decoction of fruits (triphala, ghee and honey) is also recommended for pitta related diseases.

3.5.5 Treatment of plant diseases caused due to external factors

Surpala recommended treatment of plant diseases caused due to external factors, which are as follows:

1. Attack of insect

When insects are found on stem and branches, then water the trees with cold water for seven days.

According to *Manasollasa,* there are two methods given for the destruction of worms and insects.

a. The trees should be perfumed with the smoke produced by burning *Nisa, Vidahga, Siddhartha* and *Arjuna* flowers mixed with flesh of *Rohita* fish. Besides killing insects and worms, it also helps in the luxuriant growth of flowers and fruits.

b. The insects and worms can also be killed if the trees are perfumed with smoke caused by burning of *Vidahga, Hingu, Sindhura, Marica, Ati visa, Vaca, Bhallataka* and the horns of the buffalo mixed together in equal quantities. This process also destroys all the diseases of the plants.

2. Attack of worms on tree

The worms that attack trees can be removed with help of paste prepared of milk, kunapa water, and cow dung mixed with water and also by smearing the roots with the mixture of white mustard, vaca, kustha, and ativisa.

3. Creepers adversely affected due to insects

If creepers get adversely affected due to insects, water mixed with oil cake should be sprinkled over it. By sprinkling the powder of ashes and brick dust, the insects on the leaves can be destroyed.

4. Plants wounded by animals

Sometimes plants get wounded by the animals for one reason or the other. Due to this plants fall prey to certain diseases. For curing them Surapala has said that if the trees are wounded by animals, then they get healed if sprinkled with milk and a mixture of *vidahga,* sesame, cow's urine, ghee, and mustard.

5. Cold and heat as cause of disease

There are several diseases that are caused due to excessive cold or heat. *Upavanavinoda* has come up with some preventive measures that would be helpful in curing diseases caused due to cold or heat. It states that one should take up the ashes of the trees struck by lightning and throw the same on the trees and plants. This spray of ashes would help the trees in withstanding the cold.

Vraksayurveda has also a cure for the plants affected from snowfall or scorching heat. According to him, if the trees are adversely affected due to

snowfall or scorching heat, they should be externally covered. Thereafter, they should be sprinkled with *kunapa* water and milk. They will get relief.

6. Rain as cause of disease

On various occasions it rains in such a way that plants get adversely affected and become prone to diseases. *Upavanavinoda* suggest sprinkling of a specific mixture on the plant to avoid diseases caused by harmful rains. This mixture consists of boiled rice of white variety mixed with curd and rock salt and it should be thrown round the trees.

3.5.6 Treatment of some specific diseases

1. When fruits are destroyed prematurely

It is seen that on various occasions fruits get destroyed prematurely for one reason or the other. Treatment of such trees as per *Varahmihira* is that when the fruits of a tree are destroyed prematurely, it should be watered with milk which has been cooled after being boiled, with horse gram, black gram, green gram, sesamum and barley. After being treated, thus, it will have abundant flowers and fruits.

If the fruits get destroyed then tree should be watered along with kulattha pulse, Masa, Mudga, Sesamum and barley mixed with cold water. Fruits and flowers could be obtained by doing so.

2. Treatment for broken trees

Some trees get broken either due to certain diseases or stormy conditions. *Vrksayurveda* has suggested treatment for this. It says that the broken trees should be smeared with the paste of the bark of plaksa and udumbara mixed with ghee, honey, wine, and milk and the broken parts should be firmly tied together with the rope of a rice stalk. Fresh soil then should be filled in the basin around the trees, sprinkled immediately with the milk of buffalo and flooded with water. They recover after this treatment.

Sometimes branches of trees fall off. For this also *Vrksayurveda* has prescribed the treatment. It says that if the branches fall off, the particular spot should be pasted with the mixture of honey and ghee and sprinkled over by milk and water so that the tree will have its branches reaching the sky.

3. Treatment for burnt branches and trees

a. Burnt branches

If the branches are burnt they should be cut off and the particular spots should be sprinkled with water mixed with milk. Thereafter, they should be smoked with crab shell. New seed-bud sprout out of that tree and healthy leaves are seen everywhere.

b. Burnt trees

When the tree gets burnt, then all sides of the tree should be plastered with padmini and mud. Thereafter, kunapa water should be sprinkled over it. After doing so, new branches of tree develop tending to touch the sky.

Upavanavinoda says that the treatment of the trees burnt with the fire is that, the trunk of the trees should be smeared with lotus bulbs pasted with kunapa water and milk. This makes the trees bear leaves which are bright green.

c. Burning due to lightening

If the tree is damaged due to a stroke of lightening, then the tree should be irrigated with water mixed with milk. The tree should also be plastered with the paste made up of the mixture of Vidari, sugar, nagajihva and Tila. If treated like this, leaves sprout from the tree.

4. Treatment for dry trees

a. Dryness due to burning

If the tree has gone dry after getting burnt from fire then plaster should be applied over it which has been prepared with a mixture of sweet water (water mixed with sugar), Tila, milk, padmini and mud. The dryness disease developed in the tree is then gone.

b. Dryness due to problems in soil

If the trees get dried due to problems in soil, then that soil should be replaced with new one. Thereafter, it should be watered mixed with milk.

5. Treatment for barrenness

a. Treatment 1

For those trees that are unproductive or barren, they should be watered with milk mixed with kunapa jala. After doing so, the tree is loaded with flowers and fruits.

b. Treatment 2

If the barren tree is irrigated with cold water mixed with Tila, Jau, Kulattha, Masa and Mudga, it becomes productive and loaded with flowers and fruits.

c. Treatment 3

In case where barrenness would be apprehended, the leaves and branches of the tree should be sprinkled over with a solution of cold

water, saturated with Vidanga, clarified butter, pulverised Mudga, Masa and kulattha pulse. A tree sprinkled with a solution of cold water and clarified butter, produces abundant fruits and blossoms.

6. Treatment for dotage

   Upavanavinoda says the old age trees that have reached their dotage through repeated production of flowers and fruits are to be treated with kunapa water and milk separately and then they will surely bear fruits.

7. Treatment for diseases related to water

Sometimes it so happens that excessive watering or lack of watering to plants leads to numerous diseases. Seers have suggested treatment for such diseases too. *Upavanavinoda* says that excess of water or lack of it makes the branches of the tree becoming dry. Cure for this is, vidahga, clarified butter and milk should be boiled together and the trees should be sprinkled continuously for a week.

Sometimes it so happens that trees are unable to digest water. Under such condition, the trees become pale, devoid of leaves, full of ants and smell like fish and at places the trees are devoid of barks. For this *Upavanavinoda* suggests the treatment. Firstly, the poisonous sap is to be removed from the trunk and then the affected places are to be treated. The paste of honey, vidanga powder and sesamum have to be applied to the affected place; that part is also to be covered with earth and then sprinkled with water and milk. The atrophy (Sosa) of a tree is cured when it is treated with the above mentioned paste and sprinkled with the mixture of sugar, sesamum, milk and water. Thereafter tree has to be fumigated.

3.5.7 Prayer and mantra based treatment

Parashara specifically gives the mantra for controlling grain destroyers to the feet of the revered preceptor, the meaning of which is as follows:

Let success prevail! The ever victorious feet of Ram (i.e. Rama himself). the Lord of Lords, the Emperor of Emperors, the revered one, commands from his heavenly abode situated on the peak of the Himalayas, the slope of which are white like the conch, the jasmine flower, the Moon – Hanuman, the son of Lord Wind, moving fast like wind, destroyer of invaders, standing on the seashore amidst hundreds and thousands of monkeys with his tail raised and claws harsh and strong, let there be well-being. Winds are blowing with great force in a section of a farm belonging to so and so hailing from such and such family/group. If the destroyers of crops such as gandhi, shankhi, pandarmundi, dhuli, shringari, kumari, madaka, etc. and goats, wild boars, pigs, deer, buffaloes, parrots, sparrows, winged insects, etc. do not leave that farm by your order, you shall strike them hard.

The mantra had to be written with the red lac-dye on a leaf and tied in the field. By doing so fear of diseases, insects and animals are done away with.

3.5.8 Materials and practices in *Vrikshyayurveda* in medieval era

Surpala's *Vrikshyayurveda* mentioned some materials (along with their properties) and practices that were supposed to be used in agriculture for the protection of crops. Some of these materials and practices need our attention. A few of these materials are described below in brief.

1. Application of milk and milk products

   Milk and ghee have been used for centuries. Glutamate, leucine and proline form about 40% of the total amino acids in milk. Recently, a report (Arun Kumar et al., 2002 AD) claimed that milk sprays induced systematically acquired resistance in chilli against leaf curl (a viral disease). Milk also has been used for controlling powdery mildews. The amino acid proline has been found to systemically induce resistance in plants (Niranjan Raj and Shetty, 2002 AD). High amounts of endogenous proline increase the contents of cytokinin and auxins. So milk treatment requires early attention and given an opportunity to rediscover its beneficial effects.

2. Application of cow dung

   The use of cow dung has been indicated since the time of Kautilya (ca. 300 BC). It was used for dressing seeds, plastering cut ends of vegetatively propagating sugarcane, dressing wounds, sprinkling diluted suspension on plants, etc. since ancient times.

   Cow dung is a mixture of dung and urine, generally in the ratio of 3:1. Cow dung consists of nitrogen, potash, sulphur. When seed is treated with cow dung in various ways, it gets coated with cow dung residue that contains cellulose, hemi-cellulose, micronutrients, metabolic nitrogen, epithelial cells from the animals, bile salt and pigment, potash, sulphur, traces of phosphorus and a large number of bacteria. This thin dry layer of residue on seed absorbs moisture from the surrounding soil to the advantage of the seed. The presence of bacteria in cow dung plays a significant role in the development of the seed. As these cow dung bacteria have the capacity to utilise cellulose, hemi-cellulose and pectin, so these can quickly colonise the area around sown seed and compete with the pathogenic fungi and bacteria and prevent them from attacking the seed. As Indian farmers are using cow dung for a long time, they are convinced of its utility. Now, it is important to take initiative as there is a lot to learn about

the role of cow dung in maintaining the seed health. Dried cow dung powders could also be applied to soil to promote bio-control.

Nene informs that it is good news to know that according to a newspaper report (15 March 2003 AD) that scientists at the Indian Institute of Technology at Delhi and Kanpur have taken up a project in researching cow urine.

3. Application of liquid manure (kunapa)

Kunapa's preparation involves boiling flesh, fat and marrow of animals such as deer, pig, fish, sheep or goats in winter, placing it in an earthen pot and adding milk, powders of sesame oil cake, black gram boiled in honey, decoction of pulses, ghee and hot water. Now the pot containing these materials is put in a warm place for two weeks. The resulting fermented liquid manure is known as *kunapa*. There is always a danger of passing on dormant pathogens to fields with plant-based composts, but there should be no such danger with application of kunapa water.

Firminger (1864 AD), who was known as the *Chaplain of the Bengal Establishment* mentions beneficial use of liquid manure for vegetable cultivation, but he has given no information about who first thought of liquid manure.

All the materials used in the preparation of kunapa need detailed research so that we might be able to provide acceptable scientific evidence to support recommendations made by Surpala.

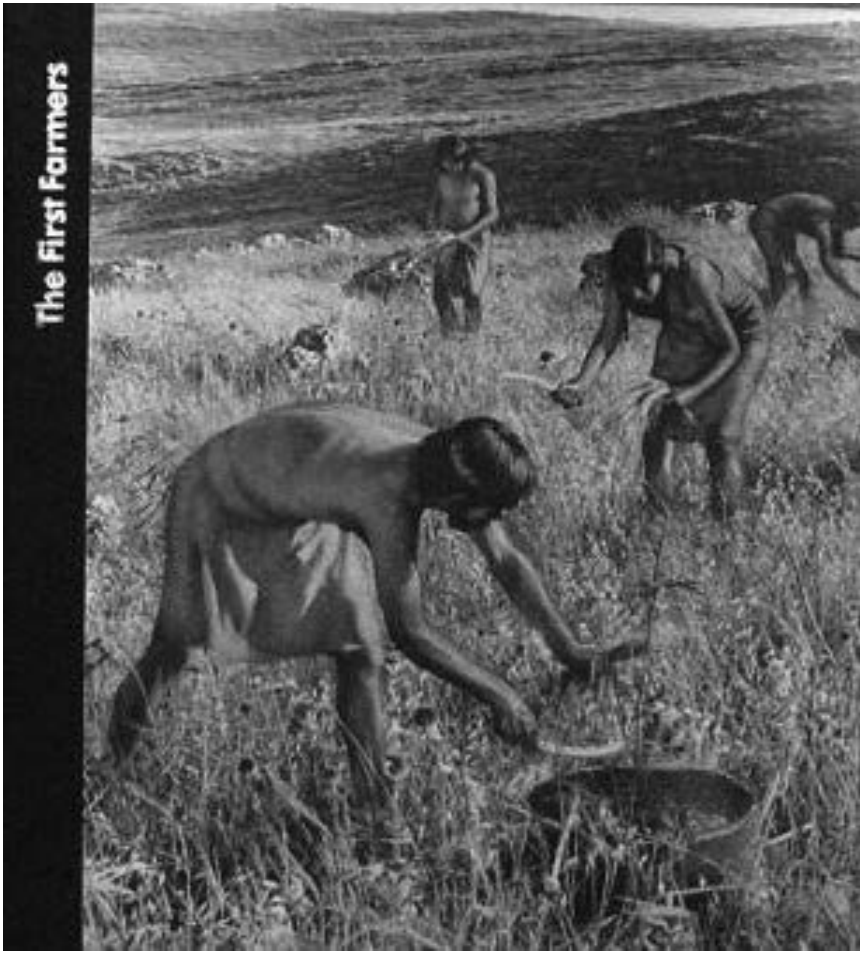
4. Application of some other materials

Some other materials mentioned by Surpala were animal fat, ash, brick powder, buffalo horn, cow horn, crab shells, fish meal, honey, horse hair, lotus mud, marrow, etc. All these materials were recommended by Surpala to control tree disorders. Some plant species like *Acorus calamus L., Oroxylum indicum, Solanum indicum L., Piper nigrum L., Embelia ribes* Burm F., etc. were also considered useful by him.

Now, we can say that there are many opportunities in researching our past technologies in agriculture. It is the need of the day to know about these methods and investigate them, so that we can utilise them at present.

4

Plant pathology in premodern era



Development of modern plant pathology had to wait for a sufficient foundation to be laid in botany and other sciences from which it derives its roots. This, in turn, had to await a change in intellectual climate that could make it possible

to break the bonds of scholarticism, dogma, and inertia and to initiate free inquiry into natural phenomena. Beginnings of the intellectual awakening of the Renaissance in Europe in the 14th century heralded this change. As the Renaissance progressed, there was a general awakening of activities in all fields. Printing, which was introduced in Europe during the middle of the 15th century, greatly facilitated learning, and the discovery of the western hemisphere. Several decades later it has opened the prospects that fired men's imagination and spurred their activity. The 16th century work, which was done largely in response to the urge of medicine, aroused a new interest in botany and added to the foundations for the great movement in description, naming, and classification of plants that proved to be the first step in the development of modern botany.

The development of optical apparatus in the 17thth century opened a new era in science. With extended vision, **Galileo** could look outward through his telescope and reveal a new cosmos and the early microscopists could look inward and discover a new world of the *infinitely small*. **Robert Hooke** (1665 AD) improved the compound microscope and saw the sections of cork and other plant tissues, which were made up of minute units, those he named as cells. **Antony van Leeuwenhoek**, a linen draper, surveyor, wine-gauger and Chamberlain to the Sheriffs of Delft, Holland, ground the finest simple lenses of his time as an avocation, discovered bacteria, yeasts (in 1675 AD–76 AD) and many other microorganisms, and opened up a new science of microbiology (Dobell, 1932 AD). He is commonly known as *the Father of Microbiology*, and considered to be the first microbiologist.

Sir David Brewster (11th December 1781–10th February 1868 AD) was a Scottish physicist, mathematician, astronomer, inventor, writer, historian of science and university principal.

Most noted for his contributions to the field of optics, he studied the double refraction by compression and discovered the photoelastic effect, which gave birth to the field of optical mineralogy. For his work, William Whewell dubbed him the *Father of Modern Experimental Optics* and *The Johannes Kepler of Optics*.

He is well-recognised for being the inventor of the kaleidoscope and an improved version of the stereoscope applied to photography. He called it as *lenticular stereoscope*, which was the first portable 3D viewing device. He also invented the binocular camera, two types of polarimeters, the polyzonal lens and the light house illuminator.

As the foundations of modern botany were laid, similar advances were made in other fields of science, and at last the time was ripe for the beginning of a modern science of plant pathology.

4.1 The development of the germ theory of disease in plants

The first great step towards establishment of the germ theory of disease, after the development of the microscope, was the experimental proof that fungi are autonomous organisms that reproduce by means of seed like bodies, rather than capricious products of spontaneous generation.

4.1.1 Theory of spontaneous generation

John Needham (1743 AD) did experiments with gravy and later, tainted wheat in containers. This was done in order to experiment with spontaneous generation. Needham was curious to know how this term was relevant. The experiments consisted of briefly boiling a broth mixture and then cooling the mixture in an open container to room temperature. Later, the flasks would be sealed, and microbes would grow a few days later. Those experiments showed that there was a life force that produced spontaneous generation. Today, it is now known that the boiling time was insufficient to kill an endospores of microbes and the cooling of flasks left open to the air could cause microbial contamination.

4.1.2 Germ theory

Louis Pasteur's (1862 AD) gave germ theory that replaced the concept of spontaneous generation of microorganisms in dead or dying organisms. de Bary's work preceded Pasteur's germ theory and should be credited to advocate the theory of Pasteur in fungal pathology.

4.2 Experimental proof of reproduction in fungi

Porta (1588 AD) saw the black spore dust of mushrooms and stated that it was their seed, but he had no proof. **Robert Hooke** (1665 AD) saw and figured teliospores of a Phragmidium taken from yellow spots on leaves. He, however, thought these spores were seed pods rather than seeds. He believed that the fungus initially arose spontaneously but might produce seeds for its further propagation. **Malpighi** (1675 AD1679 AD) also figured fungus spores, but regarded them as florets of an inflorescence, rather than as seeds. He thought, but did not prove, that fungi grew from seeds or fragments of themselves, rather than that they arose by spontaneous generation. **Joseph Pitton de Tournefort** (1705 AD), a prominent French botanist, confidently expressed the view that fungi could reproduce by means of eggs or seeds and cause the dangerous moldiness disease of plants in humid greenhouses in winter. He thought humidity hatched the fungus eggs or seeds in minute

crevices in plant surfaces, much as happened in moldiness of leather in cellars. He recommended keeping greenhouses drier to prevent moldiness. Thus, he clearly foresaw first, that fungi are autonomous organisms, rather than capricious creatures of spontaneous generation, and second, that they can incite disease in plants. However, he lacked the proofs, which Micheli was later able to produce for the first great proposition and Prevost for the second.

Pier Antonio Micheli, published in 1729 AD *Nova Plantarum Genera*, with financial aid of patrons, including Hans Sloane, President of the Royal Society of London, dealt mainly with higher plants but included his work on fungi, for which he is chiefly remembered. Micheli studied many fungi microscopically and identified their "seeds." He conducted an ingenious series of experiments with species of *Agarics, Mucor, Botrytis*, and *Aspergillus* to test the reproductive ability of their seeds. He scattered seeds of the *Agarics* on dead leaves which he incubated on selected sites in the woods, and cultured those of the other fungi on freshly cut surfaces of melons, quinces, and pears. He varied the environmental conditions of the experiments, made replications and repetitions, and provided non-inoculated controls. The experiments with fruits gave clear-cut and convincing results. The ``seeds" consistently produced crops of their own kind. He attributed the few aberrant growths on seeded or controlled surfaces to air-borne spores that tried to fall there. He figured spore clouds arising from *Lycoperdon* and *Fungoides* and clearly understood that spores floated in air. Thus, he took one of the first steps towards the overthrow of the theory of spontaneous generation.

4.3 Experimental proof on bunt of wheat is contagious

The next major advancement towards establishment of the germ theory of disease was **Mathieu Tillet's** (1755 AD) experimental proof that wheat bunt is contagious and it can be partly prevented by seed treatments. In well replicated and controlled plot experiments over a period of three years, he proved conclusively that application of the black dust from bunted wheat to seeds from bunt-free plants greatly increased bunt in the crop they produced and that certainly needed treatments, especially with a salt pepper solution and lime, partly prevented the disease. He did not realise, however, that bunt is incited by an organism. He thought the black dust contained a poisonous

principle that could be partly antidoted. The brilliant design of his plot experiments would be highly creditable today.

Tessier (1783 AD), a prominent French agriculturist, repeated some of Tillet's experiments on bunt of wheat and conducted others of his own. He also studied several other diseases of cereals. He confirmed Tillet's results of the contagious nature of bunt. He reported that the bunt dust placed on the germs of wheat seeds resulted in a greater number of diseased plants than when placed on other parts of the seeds. He thought that treating seed wheat with extracts of bunt dust increased bunt. He conducted many seed treatment tests for prevention of bunt, using chiefly lime, alone in water or with various materials added. He also employed several mechanical methods for partial cleansing of the seed, including washing with water. His results, which were carefully compiled, indicated partial control from many treatments, but were not sure of satisfactory control. He thought that reduction of bunt was due to reaction of the lime with the oily portion of the bunt dust. He did not recognise the parasitic nature of bunt, and concluded that the cause of the disease was unknown. He thought that soil moisture played a major role in the development of ergot of rye and that mists seemed to be the cause of rust of wheat, probably because of checking transpiration.

4.4 A period of classification of plant diseases and speculation on their causes

De Tournefort (1705 AD) divided plant diseases into two classes; the first is due to the internal causes and the second to external causes. He listed internal causes as too much sap, too little sap, bad qualities acquired by sap, and unequal distribution of sap to different parts of plants. Special interest was attached to his class of external causes, in which he included hail, frost, moldiness, plants hatched on other plants, insect injuries, and wounds. The prominent French botanist, **Adanson** (1763 AD), followed de Tournefort in classifying plant diseases in to two main groups, one attributed to internal causes and the other to external causes. He thought that mildew, rust and smut diseases were caused by impeded transpiration, and regarded the associated fungi as products of the plant sap. He thought that the black dust of bunt, which he linked to the powder of Lycoperdon, was a secondary and perpetuating cause of this disease.

In 1766 AD, grain rust occurred with great severity in Italy and was studied independently by two distinguished Italian scientists, **Felice Fontana** (1767 AD), a brilliant professor of physical and biological sciences, and **Giovanni F Targioni-Tozzetti** (1767 AD), a prominent physician and botanist, who was the successor of Micheli. On the basis of careful microscopic examinations, both these men concluded that the cereal rust diseases were caused by microscopic parasitic plants. Although their observations and interpretations

were remarkably ahead of those times, experimental proof of their thesis was lacking. In 1773 AD, **John Baptiste Zallinger** (Sorauer, 1909 AD), a professor of natural history at Innsbruck, Austria, went to an extreme in attempting to follow medical concepts and terminology. He was strongly of the opinion that fungi associated with plant diseases are products of the diseased plants, rather than causes of the disease.

Johann Christian Fabricius (1774 AD), a Danish professor and a devoted student of Linnaeus, placed plant diseases in an elaborate system of class, genera, and species. Probably because of the influence of Linnaeus, he was equivocal and far less advanced than de Tournefort and his followers, with regard to fungi as causes of plant diseases. He expressed the belief that the cause of rust and smut of cereals ``is one and the same." After calling attention to the belief of Linnaeus that black smut powder soaked in water for some days turns into small worms which are the true cause of smut, he states: ``A kind of movement is always observable when the black powder has been saturated; whether this is due to something animal, to something organic, or whether indeed it is the cause and not the effect of smut, is not absolutely certain. However, certain it is that the causes and symptoms of smut can never be better explained than by assuming something organised to be the cause."

John Turberville Needham (1743 AD) was an English biologist and Roman Catholic priest. He reported plant parasitic nematodes in wheat galls.

The last major attempt to classify plant diseases without knowledge of the true causal role of microorganisms was made by **Filippo Re** (1807 AD), professor of botany and agriculture at the University Modena, Italy, in a treatise on diseases of plants. He divided plant diseases into classes and genera according to symptoms and supposed causes. Like so many of his predecessors, he was much influenced by medical concepts and terminology. However, he included a class of *indeterminate diseases*. Of this class, in which he placed the rust and smut diseases, he wrote: "I have thus designated those diseases whose origin is either entirely unknown, or deduced from observations contradictory in themselves, or from hypotheses which, however brilliant, have no real foundation." After this excellent statement, he discusses the opinions of several prominent writers on the cause of rust diseases and adds his own, which he summarises as follows: "All this would lead me to lay down that the cryptogamic plants, the minute insects, or the exudations, whether dry or not, are rather symptoms of the disease itself, which is a result of excessive vigour or over-repletion."

4.5 Early development of mycology

The development of mycology was highly essential for the progress of plant pathology. Beginning with the work of **Carolus Linnaeus** (1753 AD), mycology entered a dominantly taxonomic period. Although Linnaeus worked little with fungi, he made an important contribution to mycology by including them in his Latin binomial system. His work is the starting point for modern nomenclature of the Myxomycetes and lichens. **Pierre Bulliard**, a talented French botanist and mycologist, wrote a major book on mycology, *Histoire des champignons*. This work was published in part, beginning in 1791 AD; the second part was completed by Ventenat after Bulliard's death and the complete work was published in 1809 AD–1812 AD. Bulliard recognised four orders based on the position in which the seeds were borne. He used coloured illustrations, which he prepared. The foundations of modern classification of fungi were laid chiefly by Persoon and Fries.

Christiaan Hendrik Persoon, devoted most of his time to the study of fungi. His *Synopsis Methodica Fungorum* (1801 AD) is the chief basis for all later classification of fungi and is the starting point for the nomenclature of the Uredinales, Ustilaginales, and *Gasteromycetes*. He thought that some fungi arose spontaneously and that some grew from spores. He regarded smut fungi as products of the diseased plants.

Elias Magnus Fries, designed monumental *Systema Mycologicum* (1821 AD–1832 AD) to include all the fungi then known. It is the starting point for the nomenclature of all groups of fungi except those that start with the work of Linnaeus or Persoon. Fries regarded the rust and the smut fungi as products of the diseased plants. Among many other prominent early mycologists were Nees von Esenbeck (1816 AD–1817 AD) of Germany, Leveille (1837 AD, 1846 AD, 1851 AD) of France, Corda (1837 AD-1854 AD) of Bohemia, and Berlseley, (1857 AD, 1860 AD) of England. Although early concentration on taxonomy undoubtedly delayed progress in other aspects of mycology, but it laid foundation for the essential basis for further development of the science.

4.6 Experimental proof that bunt of wheat is incited by a fungus and can be controlled by a fungicide

Prevost (1807 AD) clearly showed "that the immediate cause of bunt is a plant of the genus of the uredos or of a very nearly related genus". This work contains the first recorded adequate experimental proof and interpretation of the role of a microorganism in the causation of a disease. He gave an accurate and detailed description of the symptoms of bunt in its various macroscopic stages of development. He suspected and proceeded to prove that the *globules* in the bunted kernels were *gemmae* or spores of a cryptogam. He described and illustrated these spores in detail and made extensive studies of their germination and of the development of the *bunt plant* in relation to time, temperature, substrata, toxic agents, age and previous treatment of the spores, and concentration of spores. Having concluded that he was dealing with a microscopic plant, he proceeded to prove by extensive inoculation experiments that it is the immediate cause of bunt and to ascertain conditions which favour or hinder its infection. He pointed out "that the vegetation of this plant, as well as that of a majority of the uredos, begins in the open air and is completed in the interior of the plant that it attacks," suggesting for such organisms "the general denomination of internal parasitic plants." He observed germinated spores of the bunt organism in the soil and on the surface of wheat seedlings grown in infested soil. Although he did not succeed in observing the mode of penetration of the bunt fungus into the wheat plant or its growth in the wheat embryo, he correctly surmised that some ramifications of the bunt plant must penetrate into the very young wheat plant and later insinuate themselves into the embryo and fructify. He observed fructification in the embryo and germinated spores. In extensive and refined toxicological studies, he found that certain copper salts and distilled water in which metallic copper has been left and various other substances in solution would prevent germination of spores of the bunt fungus. He critically distinguished injurious and inhibitory forms and their lethal effects and experimented extensively with regards to relations of concentration of the toxic agent, time and temperature to the toxic effects. On the basis of this information and his extensive knowledge of the disease, he experimented for prevention of bunt. He made suitably controlled field tests in which inoculated seed wheat was planted after having received various treatments. Spores of the bunt organism from treated seed were tested for germination, and data were taken on the development of the disease on the wheat grown in the field from the experimental seed. Excellent control of the disease was obtained by dipping the seed wheat in a copper sulphate solution, and detailed practical recommendations were made for large-scale seed treatment. Prevost's work, which was remarkably comprehensive and well correlated, laid a firm foundation for nearly all branches of modern plant pathology. He developed methods for obtaining virtually pure cultures of spores

of the bunt fungus and for keeping them free from contamination by air-borne reproductive bodies of other microorganisms. He expressed his disbelief in spontaneous generation. He regarded the bunt plant as the immediate or direct, rather than the sole cause of bunt because he clearly proved that the fungus can incite the disease only under sufficiently favourable conditions. He thus recognised the conditioning or secondary causal relationship of environment.

This discovery of the cause and a means of prevention of bunt of wheat gave to the world a key to understand the cause and the prevention of all infectious diseases. It, therefore, ranks high among the great pioneering advances in science (American Phytopathological Society, 1956 AD; Keitt, 1956 AD). **Tulasne brothers** (LR and C Tulasne), of France, drew the attention of the other contemporary mycologists to Prevost's findings.

Franz Joseph Andreas Nicholas Unger was an Austrian physician and an eminent professor of botany. Although primarily a plant physiologist, he devoted much study to plant diseases. His best-known work on plant pathology was his *Exantheme der Pflanzen* (1833 AD). He thought that fungi associated with plant disease on the diseased plants are because of abnormalities in the plant juices, and were, therefore, products rather than the causes of disease. **Franz Julius Ferdinand Meyen** was a physician and a brilliant young professor of botany at the University of Berlin. He wrote on many aspects of botany, especially physiology and anatomy. His most important work is on plant pathology, *Pflanzen-Pathologie* (1841 AD), was published a year after his untimely death at the age of 36. Firmly wedded to the autogenetic theory, he regarded fungi associated with plant diseases as pseudo organisms which resulted from abnormal nutrition of the plants. **Justus von Liebig** (1853 AD a,b) was one of the most famous chemists of his time. A doctor of medicine, (though not active as a physician or biologist) he was a very influential opponent of the germ theory of disease. His ideas about the nature of disease were based on chemical theory rather than experiments with diseases. He told that fermentation, putrefaction, and contagious disease resulted from an active state of atoms, and that this active state of the atoms of one body could be transferred to those of another body in contact with it.

In Australia, **Collins** was the first to record disease in wheat in 1795 AD in a crop of bearded cape wheat "which was not worth the labour of sowing" at Petersham Hill, now the site of Sydney University. Early settlers referred to disease of wheat crops as blights or accidents, and the disease could not be identified.

In Asia, **Jahangir** (1605 AD–1627 AD), in his memoirs, described a disorder of marigold, which could be ascribed today to species of *Alternaria, Botrytis,* or *Sclerotinia.* Some documents of the early 19th century from the Mewar region of Rajasthan give description about powdery mildew *(chhachhia*) on different plants, and canker or anthracnose (*titari)* of orange.

In the second half of the 19th century parasitism of fungi was proven in Europe. *A Dictionary of Economic Products of India* was a monumental effort of **G Watt**, published during 1889 AD–1893 AD. This book contains a detailed description of disorders of crops covering the period from 1820 AD onwards. **Watt** (1889 AD–1893 AD) mentions various fungal diseases, viz; Ergots of barley, oat, pearl millet and horse gram; smut and rust (*Puccinia* sp.) of wheat; leaf rot of coconut (*Pellicularia koleroga*); rust of barberry; rust **(***Melampsora lini*) of linseed; rust (white rust) of mustard; late blight of potato; powdery and downy mildews of grape wine; root blight in tea; bunt of wheat; smuts and rusts of barley and maize; false smut of paddy; blight of cotton; *Cercospora* leaf spot of cotton in Madras (Chennai); powdery mildew of indigo; rust and smut of pearl millet in western United Provinces (U.P.); mildew (*Cercospora* sp.) of black gram; fungoid diseases (tuto, angare, nona, chittigabari, gandi) of betel vine in Bengal.

The microscope was introduced in about 1700 AD and since then, through observation of many biological materials, including filamentous fungi, an entirely new field has been developed.

5

Plant pathology in modern era

The effects of the Irish famine were not limited to Great Britain. Its impact was felt throughout much of the western world and on the history of modern era.

5.1 In Europe

5.1.1 The Potato Disease and the Irish famine

The devastating epiphytotic of the potato disease in the middle of the 19th century was one of the most tragic events in human history. It swept the whole

of Europe and USA, but it was catastrophic in Ireland. The years 1845 AD and 1846 AD were milestones in the misery of man. Over one million people died and one and a half million migrated. This tragic event was a blessing in disguise, in so far as it hastened the realization of the importance of plant diseases. Many committees and commissions were formed to report on the disease. The causal organism was identified by **Sepeerschneider** in 1857 AD (see Keitt, 1959 AD) and **De Bary** (1861 AD–1863 AD–1867 AD). It was *Phytophthora infestans.* The battle against the spontaneous origin of disease was largely won. The science of plant pathology, evolving for a long time, was born. **Berkeley**, the most prominent British mycologist of his time, had, after initial reservations, accepted the causal role of microorganisms. He described (1847 AD) *Oidium tuckeri,* to be the cause of the powdery mildew of vine, and this helped in quick acceptance of De Bary's announcement of *Phytophthhora* being the cause of the potato late blight. It is interesting that **Lindley**, the editor of the *Garden Chronicle,* which recorded the miseries of the potato epiphytotic with great passion, was a firm supporter of the spontaneous origin.

5.1.2 The foundation of modern mycology and acceptance of the concept that fungus can incite disease in plants

The **Tulasne brothers** in France and **De Bary** in Germany were the outstanding founders of modern mycology. The Tulasnes most important work dealt with the rust and the smut fungi and the Ascomycetes (1847 AD, 1854 AD, 1861 AD–65 AD). The Tulasnes (1847 AD) referred most favourably to Prevost's work and accepted his concept that fungi can incite disease in plants. Their crowning work was the superbly illustrated *Selecta fungorum carpologia*.

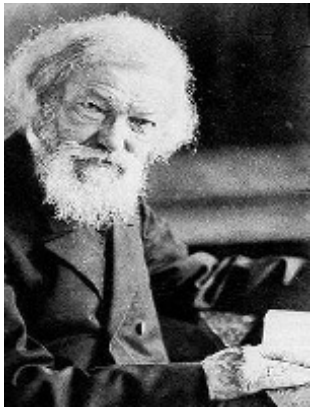
The Italian mycologist **P.A. Saccardo** (1845 AD–1920 AD) collected and brought together the scattered knowledge on systematic mycology under his massive 25-volume work, the *Sylloge Fungorum Omnium Hucusque Cognitorum* (1882 AD–1925 AD), whose first volume appeared in 1882 AD and the 25th in 1931 AD. With the publication of the *Sylloge*, as the work is usually called, systematic mycology took a big leap forward. *Sylloge* was not a mere compilation; it involved many difficult taxonomic decisions, which only a mycologist of Saccardo's stature could take. His classification of *Fungi Imperfecti* based on spore groups is extremely popular and has been in use ever since it was published in 1880 AD.

Heinrich Anton De Bary discovered *Heteroecious Nature of Rust Fungi* (1865 AD). He gave detailed account on life cycles of downy mildew genera. He studied about vegetable rotting fungi and damping of fungi. De Bary was devoted to the study of the life history of fungi. At that time, various fungi were still considered to arise through spontaneous generation. He proved that pathogenic fungi were not the products of cell contents of the affected plants and did not arise from the secretion of the sick cells. He studied the pathogen *Phytophthora infestans* (formerly *Peronospora infestans*) and elucidated its life cycle. The origin of plant diseases was not known at that time. De Bary spent much time studying the morphology of fungi and noticed that certain forms that had been classified as separate species were actually successive stages of development of the same organism. De Bary studied the developmental history of Myxomycetes (slime moulds), and thought it was necessary to reclassify the lower animals. He first coined the term Mycetozoa to include lower animals and slime moulds. In his work on Myxomycetes (1858 AD), he pointed out that at one stage of their life cycle (the plasmodial stage), they were little more than formless, motile masses of the substance called protoplasm. This is the fundamental basis of the protoplasmic theory of life. He wrote a book named ***Morphology and Physiology of Fungi, Lichens and Myxomycetes*** (1866 AD). He reported the role of enzymes and toxins in tissue disintegration caused by *Sclerotinia sclerotiorum.* De Bary was the first to demonstrate sexuality in fungi. In 1858 AD, he had observed conjugation in the alga *Spirogyra*, and in 1861 AD, he described sexual reproduction in the fungus *Peronospora* sp. He saw the necessity of observing the whole life cycle of pathogens and attempted to follow it in the living host plants.

He published *Untersuchungen fiber die Brandpilze* (1853 AD). In the first two parts of this book he reported thorough microscopic studies of the structure and development of numerous smut and rust fungi, including their relationships to the tissues of the diseased plants, and discussed their systematic relationships and classification. In the third part, he dealt with the relationships of these fungi to the smut and rust diseases. After a thorough discussion of literature and of his own observations, he concluded: “It has been shown that the smut and rust fungi originate not from the cell content or from the secretion of diseased cells and that they are not the result, but the cause of pathological processes.” He suggested destruction of diseased parts of plants as a method of preventing such diseases, but recognised that

this could not always be done. Hence, he wrote, "for agriculture a successful result will be obtained only by seeking in the main to prevent in every way the development of smut and rust fungi and therefore as far as possible destroying their spores, the smut dust. This seems to be accomplished by means of the various corrosives which the farmers use for disinfection of the seed undoubtedly and indeed chiefly by means of copper sulphate and lime."

John Robertson (1824 AD), in Ireland in 1821 AD, reported a careful study of peach mildew, in which he correctly interpreted the causal role of the fungus and successfully controlled the disease by repeated applications of a preparation of sulphur and soap in water, by means of syringe. This treatment, or modifications of it, came into use by gardeners, and recommendations of preparations of lime and sulphur soon followed (Lodeman, 1896 AD). The powdery mildew of the vine was first observed in Europe in glasshouses at Margate, England, in 1845 AD by **Edward Tucker**, a gardener (Berkeley, 1847 AD). After satisfying himself by microscopic study that he was dealing with a fungus similar to that of peach mildew, Tucker applied a preparation of sulphur and slaked lime in water to the diseased leaves by sponging or by rubbing it on with his hands. The mildew in his house was controlled, while in the next garden it developed destructively. As the disease spread rapidly and threatened the vineyards of Europe, many modifications of the sulphur treatment were tried. According to **Mares** (1856 AD), a French gardener, Gontier obtained excellent results in controlling the mildew in glasshouses in 1850 AD by applying sulphur dust to moistened vines by means of a bellows. In 1851 AD, another French gardener, Grison, reported successful control of the mildew by a diluted preparation made from sulphur and freshly slaked lime boiled in water (Henze, 1852 AD). In 1852 AD, Berman, a gardener in France, reported successful control of grape mildew by moistening the hot water pipes of glasshouses and powdering them with sulphur (Truffaut, 1852 AD). **Mares** (1856 AD) stated that decisive results were obtained in 1853 AD on 300 acres of vines at Thomery, France, by using a method proposed by R. Charmeux of dusting the dry vines with sulphur. With modifications, this method rapidly came into general use and saved the European vineyards.



Julius Gotthelf Kuhn (1825 AD–1910 AD) was a German academician and agronomist and he was one of the founders of plant pathology. He published first textbook on plant pathology *The Diseases of Cultivated Crops: Their Causes and Their Control.* In 1862 AD, he became a professor of agriculture at the University of

Halle. Kuhn published more than 70 papers on mycology and plant pathology over the course of his career.

One of his seminar papers published in the year 1858 AD was *Die Krankheiten der Kultergewachse*.

Many other advances of great biological significance were made in the early and middle 19th century. Among these were the synthesis of urea by **Woehler** in 1828 AD, the discovery of the cell nucleus by **Robert Brown** in 1831 AD, the founding of the cell theory by **Schleiden** and **Schwann** in 1838 AD–1839 AD, the contributions to plant embryology by **Hofmeister** in 1849 AD and 1851 AD, the works of **Wallace** and **Darwin** on the origin of species in 1858 AD and 1859 AD, and the discovery of the laws of heredity by **Mendel** (1866 AD) and **von Sachs**, (1875 AD).

5.1.3 The development of modern techniques

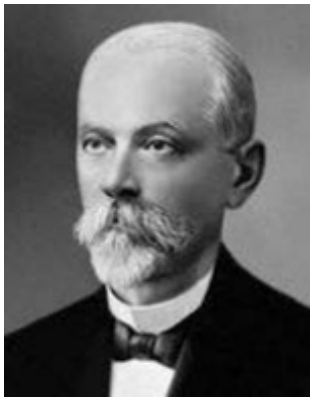
Oscar Brefeld (1875 AD, 1881 AD, 1883 AD), working with fungi, was the leader in the early development of modern techniques for growing microorganisms in pure culture. With the refinements made by Koch, Petri, and others, his techniques were the foundation for the pure culture methods currently being employed. After his earlier studies on the complete life cycles of saprophytic fungi, he gave major attention over a period of some 30 years to the smut fungi and diseases (1888 AD, 1912 AD; Brefeld and Falck, 1905 AD). He was the leader in tracing the life cycles of the cereal smut fungi and their role in the causation of diseases. In this work he made important contributions both to pure culture of smut fungi and to inoculation techniques.

5.1.3.1 Virological development

Adolf Edward Mayer was a German agricultural chemist whose work on tobacco mosaic disease played an important role in the discovery of tobacco mosaic virus and viruses in general.

Mayer published a paper in 1886 AD on the disease which he named as *mosaic disease of tobacco* and described its symptoms in detail. He demonstrated that the disease can be transmitted by using the sap from the affected tobacco plants as the inoculum to infect healthy plants. At that time, this disease was thought to be spread by a very small bacteria or toxins, yet some years later the tobacco mosaic virus (TMV) proved to be the culprit. Mayer concluded that the infectious agent was some sort of bacteria and erroneously claimed that he was able to obtain clear filtrate from the infected sap using filter paper in several repetitions. Regardless of the erroneous conclusion, Mayer's pioneer work on the tobacco mosaic disease served as an

important step in the discovery of viruses and led to the foundation of the field of virology.



Dmitri Iosifovich Ivanovsky (1864 AD–1920 AD) was a Russian botanist, the first man to discover viruses (1892 AD) and thus one of the founders of virology.

He discovered that both incidents of disease were caused by an extremely minuscule infectious agent, capable of permeating porcelain Chamberland filters, something which bacteria could never do. He described his findings in an article (1892 AD) and a dissertation (1902 AD).

In 1898 AD, the Dutch microbiologist **Martinus Beijerinck** independently replicated Ivanovsky's experiments and became convinced that the filtered solution contained a new form of infectious agent, which he named *virus*. Beijerinck subsequently acknowledged Ivanovsky's priority of discovery.

5.1.3.2 Plant disease control

By 1872 AD, **C.V. Riley** had been to France to observe the dying vines and talked to French scientists and growers as to the significance of the fact that American vines in France seemed to be spared except for some galls on the leaves. What Riley suggested was for the French vineyards to use American root stocks that have got adapted to the *Phylloxera* and graft them onto French grape plant. Some growers were convinced by Riley and began importing root stocks from America. However, to many growers this was not acceptable.

Pierre-Marie-Alexis Millardet (1885 AD) was a French botanist and mycologist born in Montmirey-la-Ville. He was responsible for protecting grape vineyards from downy mildew fungus (*Plasmopara viticola*). He accomplished this feat by implementing a fungicide consisting of hydrated lime, copper sulphate and water, a mixture that was to become known as the *Bordeaux mixture*. It was the first fungicide to be used worldwide, and is still being used today.

5.2 In America

Thomas Jonathan Burril (1839 AD–1916 AD) was an American pathologist who first discovered bacterial causes for plant disease. He introduced *Erwinia*

amylovora (called by him *Micrococcus amylovorus*) as the causal agent of pear fire blight. He showed that a bacterial organism was constantly and abundantly present in freshly blighted tissues and that he could incite the disease consistently by direct inoculations (1888 AD, 1881 AD, and 1884 AD).

5.3 In Australia

Plant pathology in Australia began in the 19th century as a result of sporadic rust epiphytotics which blighted wheat crops and devastated part of the vital supply of the pioneers. Soon after the first settlement was established in Australia in 1788 AD, Governor Phillip devoted all his spare time to the question of providing food for the young colony. The first crop of wheat was grown in the Sydney Botanic Gardens. Early settlers referred to disease of wheat crops as blights or accidents, and the disease could not be identified. **Joseph Holt**, a farmer, gave an accurate description of a rust-infected wheat crop at Dundas, New South Wales, in 1803 AD. After a few days of foggy weather and within 3 weeks of harvest, wheat was reduced in its value from £1400 to £20. "It covers the whole wheat straw with reddish powder……"

The early governors frequently mentioned crop losses in their despatches to the British. Governor King in his dispatch of 1804 AD reported: "Our last year's crop was much injured by rusts and smuts." The early records of rust attacks in the Australian colonies have been reviewed by Waterhouse, who showed that rust epiphytotics occurred in 1799 AD, 1803 AD, 1805 AD, 1829 AD, 1832 AD, 1860 AD, 1863 AD, 1864 AD, 1867 AD, and 1889 AD.

Not long after the founding of Victoria in 1851 AD, the Board of Agriculture (forerunner of the Department of Agriculture) was established. Within 5 years, three rust epiphytotics occurred, causing the Board to appoint a committee in 1864 AD to inquire into the cause and prevention of rust. **F. Mueller** (later Baron Sir Ferdinand von Mueller) Government Botanist and Director of the Melbourne Botanic Gardens, was chairman. The rust committee determined the effect of agricultural practices on the incidence of the disease and recommended early sowing and careful selection of wheat varieties as a mean of control. In Australia this first official scientific inquiry into a major plant disease came at the time when plant pathology was established in Germany as a scientific discipline by de Bary. Following an outbreak of wheat rust in 1867 AD in south Australia, the government of that colony appointed a similar committee.

William James Farrer graduated from Cambridge University in 1868 AD came to Australia the following year. He became a surveyor and, because of his travelling through the country, developed an interest in the problems of the man on the land. Farrer later recalled that "The idea of making improvements

in the wheat plant was taken hold of me as early as 1882 AD. … It was not until 1889 AD the first attempts at cross breeding were made." This work was carried out at his own expense on his property near Canberra until he accepted an appointment with the Department of Agriculture of New South Wales in 1898 AD. He was then able to give time to the production of a rust-resistant wheat variety. In this work, Farrer was encouraged by A.E. Blount of the Colorado Agricultural Experiment Station who was at that time producing new varieties of cereals by hybridisation and selection. Blount was not concerned with rust resistance, as this disease was not a problem under his conditions. In a letter to Farrer in 1885 AD, Blount said "I think four to six years, breeding of our standard wheat would almost entirely revolutionise them so that rust, smut and blight would be unknown … I will send you several kinds of my crosses to test." This is perhaps earliest intent of the cooperation in agricultural science between the United States and Australia.

According to Large, "the greatest single undertaking in the history of applied plant pathology was the attack of the rust on cereals. The mighty rust investigation soon began in Australia with a series of wheat rust conferences following the epidemic of 1889 AD." A brief history of the first conference has been given. Subsequent conferences were held in Sydney in 1891 AD, Adelaide in 1892 AD, Brisbane in 1894 AD, and Melbourne in 1896 AD. Watson stated "that the influence of Farrer's varieties was so great that the area sown with them rapidly increased as the overall production of wheat in Australia expanded. Federation became the leading variety and held that position until 1925 AD. He started his work when fundamental knowledge of the disease was lacking, not only in Australia but in other wheat producing countries as well. He was unaware of the extreme variability that was present in the organism and which was later to upset the breeding programmes of many countries."

Danie'l McAlpine arrived in Melbourne in 1884 AD and was appointed lecturer in biology at the University of Melbourne, and in 1890 AD, counsulting vegetable pathologist to the Department of Agriculture of Victoria. He was born at Saltcoats, Scotland in 1849 AD and, like Marshall Ward, studied biology under Huxley, and botany under Thistleton-Dyer, in the Royal College of Mines, South Kensington, England. There is little doubt that his appointment as vegetable pathologist was furthered by the 1889 AD rust epiphytotic and the program of experimental work approved by the first rust-in-wheat conference held in Melbourne in 1890 AD. He was chairman of the final conference in Melbourne in 1896 AD and, in the report, summarised the combined results of all sessions of the conference. McAlpine cooperated with Farrer by testing wheat under Victorian conditions. He published a systematic census of Australian fungi in 1895 AD, and in 1906 AD a critical monograph on rusts of Australia, followed by a paper on smuts of Australia in 1910 AD.

McAlpine published 226 scientific works, papers, and vullerins, 56 of which dealt with the fungal flora of Austria. In plant pathology there were three volumes (Diseases of Citrus in 1889 AD, Diseases of Stone Fruits in 1902 AD, and Diseases of Potatoes in 1911 AD) and 137 publications. An investigation into the nature and control of bitter pit of apples was reluctantly undertaken, and the results which were published in five reports between 1911 AD and 1916 AD included information on the influence of orchard practices on the incidence of the disease. McAlpine retired in 1916 AD and died in 1932 AD. Daniel McAlpine has been called *The Father of Plant Pathology in Australia*. He was a truly dedicated pioneering scientific worker during his 26 active years in Australia.

Nathan Augustus Cobb, who was born in Spencer, Massachusetts in 1859 AD, arrived in Sydney in 1889 AD. He has undertaken courses at the University of Jena in zoology under Haeckel, Lang, and Hertwig, and botany under Stahl, obtaining his Ph.D. in Helminthology. Cobb worked for one year at the Naples Zoological station before coming to Australia. The Department of Agriculture of New South Wales was established in 1890 AD, and Cobb was appointed vegetables pathologist the same year that McAlpine was appointed in Victoria. Cobb developed a formula for describing the anatomy of nematodes which were still used and produced about 12 papers on nematodes while in Australia. The proof that gumming disease of sugarcane was caused by a bacterium impressed Erwin F. Smith, who later supported Cobb's appointment to the United States Department of Agriculture. Cobb published an index of Australian fungi in 1893 AD. From 1891 AD to 1903 AD, he described and illustrated many plant diseases present in New South Wales. Before leaving Australia in 1905 AD, he published a monumental work on the nomenclature of wheat varieties. His experimental methods were outstanding. To replace the very indefinite adjectives previously used, he designed a scale of rustiness which was used by Farrer in connection with field observations on the degree of rust infection in his wheat. He endeavoured to determine the factors of importance to resistance to rust in wheat. He had a keen appreciation of error in field experiments, and lectured on this subject to department officers. He was a man with great practical outlook, capacity, and versatility who, in addition to his numerous duties, managed the government experimental farm at Wagga for 3 years. In 1897 AD he was commissioned to report on agriculture in overseas countries, and on his return in 1901 AD, published on such general agricultural matters as grain elevators and the California wheat industry. In 1905 AD, he went to Honolulu until he was called to Washington by the Bureau of Plant Industry of the US Department of Agriculture. According to Buhrer, **Cobb became the father of American plant nematology**. In fact he gave nematology its name. Cobb produced approximately 250 publications, 90 of which were produced in the New South

Wales Department of Agriculture. He died in 1932 AD while still actively engaged in nemotological studies.

A medical man, J**oseph Bancroft**, was probably the first to carry out a plant disease investigation in Queensland. In 1874 AD he discovered and described Fusarium wilt of bananas, noted the occurrence of varietal susceptibility, and recommended the use of disease-free planting material. He also cooperated with Farrer in his attempt to produce suitable rust-resistant wheat for Queensland.

Henry Tryon, who had been government entomologist in the Queensland Department of Agriculture since 1894 AD, also became vegetables pathologist in 1901 AD, holding the dual position until 1929 AD. Of the 136 papers he published, 30 were concerned with plant pathology, including two on the gumming disease and the top rot of sugarcane. According to Simmonds, he described for the first time in 1894 AD the bacterial nature of bacterial wilt of potato and tomato, which was subsequently confirmed by Erwin F. Smith. Tryon was the first to recognise the presence of late blight of potatoes in Australia.

The biology branch of the Department of Agriculture of New South Wales was established in 1913 AD with **G.P. Darnell** Smith as biologist, following the reorganisation of the Government Bureau of Microbiology. Darnell Smith showed that seed treatment with copper carbonate dust gave better control of bunt than did liquid bluestone-lime treatment, and that this dust treatment had no deleterious effect on the germination of wheat. This method soon became standard practice in Australia and, by 1925 AD, one tenth of the wheat grown in the United States was treated with copper carbonate powder. This discovery changed seed treatment methods throughout the world. Darnell Smith, before his retirement in 1927 AD, established a group of plant pathologist in the Department of Agriculture of New South Wales, who specialised on the crop basis, which was unique at that time.

Charles Clifton Brittlebank, after assisting McAlpine in Victoria for 5 years, was appointed as vegetable pathologist to the Department of Agriculture of Victoria in 1913 AD. He carried on this work single-handedly until 1924 AD, and also lectured in plant pathology at Melbourne University from 1923 AD–1928 AD. He published some 25 papers, and established a high-grade plant pathological diagnostic and consultative service for the primary producers of Victoria. Brittlebank became biologist in charge of science branch following W.E. Laidlaw, who had held this position since 1913 AD. On retirement he was engaged by the Council of Scientific and Industrial Research to produce a host and fungus index for Australia, which was later cyclostyled for limited distribution. He died in 1945 AD at the age of 82.

F. Maddox demonstrated in Tasmania in 1894 AD that floral infection takes place in loose smut of wheat. **H.M. Nicholls** was microbiologist of the Department of Agriculture of Tasmania from 1913 AD to 1935 AD, and published literature on black spot of apples and pears, and root rot of fruit trees.

F. Stoward was appointed as botanist and vegetable pathologist to the Department of Agriculture of Western Australia in 1911 AD. Up until that time the state had been dependent on outside help for its plant disease problems, particularly on McAlpine. Stoward was followed by H.A. Herbert in 1918 AD, and this marked the commencement in western Australia of the modern approach to plant disease problems. Hearbert was followed by J.C. Campbell.

During this era plant pathology began to develop in some of the Australian universities. The Botany School at the University of Melbourne was founded in 1906 AD with A.J. Ewart as the first professor of botany in Australia. **Ethel I. McLennan**, who was appointed to this school in 1915 AD, published on an endophytic fungus associated with the seed of *Lolium*.

Between 1890 AD and 1912 AD, plant pathological material in south Australia was referred to McAlpine and Cobb. Professor **T.G.B. Osborn's** appointment to the Chair of Botany in the University of Adelaide was contingent on an agreement with the government that he would act as botanist and vegetable pathologist to that state.

David Shepherd North joined the Colonial Sugar Refining Company as Sugar Pathologist in 1900 AD, and worked on leaf scald (*Xanthomonas albilineans*) and gumming disease (*X. vasculorum*), and on Fiji virus disease and downy mildew from 1907 AD to 1916 AD. He was notably successful in controlling these diseases in commercial crops of sugarcane, and his nine publications remain standard works. He retired in 1942 AD. The David North Plant Research Centre at Indooroopilly, Brisbane, was named after him.

5.4 In Asia

5.4.1 Historical development of plant pathology in Indian subcontinent

The first Indian universities that got established in 1857 AD were at Calcutta (Kolkata), Madras (Chennai), and Bombay (Mumbai) that emphasised taxonomy of fungi.

The first major study of a plant disease in the Indian subcontinent was by **Marshall Ward** on coffee rust in Sri Lanka in the 1870 AD onwards. At about the same time observations by Europeans on plant diseases of India

began to be made by members of the Government Medical Service including **D.D. Cunningham** (who proposed the new genus *Mycodea* for the parasitic alga, now known as *Cephaleuros virescens* responsible for red rust of tea and **A. Barclay** (still remembered for his studies on Indian rusts). In 1881 AD as a result of recommendations by the Famine Committee of 1880 AD, Imperial and Provincial Departments of Agriculture were established for the amelioration of agriculture. This development drew attention to the need for research, and an Imperial Agricultural Chemist was appointed in 1889 AD. Posts for an Imperial Cryptogamic Botanist and an Imperial Entomologist were created shortly afterwards.

The turning point for plant pathology was the appointment of **E.J. Butler** of Irish origin and a medical man by training as Imperial Botanist in 1901 AD. At first stationed at Dehra Dun, Butler moved in 1905 AD, as the first Imperial Mycologist, to the impressive newly-built Agricultural Research Institute at Pusa in Bihar where during the next two decades he laid a firm foundation for the Indian phytopathological service. The credit for laying the foundation of plant pathology goes to him, and he may aptly be called the *Father of Indian Plant Pathology*. The many important contributions to the plant pathology in Sri Lanka by **Tom Petch** between 1905 AD and 1934 AD will be remembered for a long time (Petch, 1921 AD, 1933 AD).

5.4.2 Fungal diseases

D.D. Cunninham in 1889 AD identified the causal organism of red rust of tea in Assam as *Cephaleuros vireseens.*

E.J. Butler stayed at the Indian Agricultural Research Institute for 16 years (1905 AD–1921 AD) and established a strong school of mycology and plant pathology. His book, published in 1918 AD, served as the major source of literature and inspiration to budding plant pathologists.

The research in forest pathology was initiated by **Bagchee** at the Forest Research Institute, Dehradun in the Himalayas. He reviewed in 1903 AD the work done on the coniferous rusts, root- and stem-rotting fungi, canker pathogen, nursery diseases, timber diseases, and the ecology and habits of forest fungi. He also listed rusts and polypores attacking forest trees. The principle diseases of oak in India were studied by **Bakshi**.

5.4.3 Bacterial diseases

Several bacterial diseases have long been known in India, and there are reasons to believe that some of them (citrus canker, leaf spot of mango, and black arm of cotton) may have originated here.

The first disease suspected to be caused by bacteria was bangle blight of potato in Bombay, Cappel reported it in 1892 AD as to be prevalent in Poona (Pune) and other places in Bombay, and considered the causal organism to be a fungus. Butler suggested in 1903 AD that the disease was similar to bacterial wilt of potato caused by *Bacillus solanacearum (*syn: *Pseudomonas solanocearum)*.

5.4.4 Plant disease control

Some of the historical events in the use of fungicides in India were in 1885 AD when **Ozanne** first used a fungicide copper sulphate for control of a crop disease known as sorghum smut. **Lawrence** used Bordeaux mixture for the first time in 1904 AD against *Cercospora* leaf spot of groundnut.

5.5 In Japan

After the great revolution in 1866 AD, the Japanese government did its utmost to introduce European and American civilisation, and the Ministry of Education invited eminent European and American professors from all branches of science and the arts to Japan. Plant pathology was introduced into Japan through Tokyo and Sapporo, Hokkaido.

Professor **Friedrich M. Hilgendorf** (born in 1839 AD in Brandenburg, Germany) came to Tokyo from Germany in March 1873 AD and taught botany and zoology in the medical school of the Kaisei Gakko, the predecessor of Tokyo Imperial University. Although Hilgendorf was a zoologist, in Tokyo he lectured on medical botany as well, and often touched on plant diseases (Yamanouchi, 1934 AD). After Hilgendorf returned to Germany, Professor **Hermann Ahlburg**, student of Julius von Sachs, replaced him in May 1876 AD. Ahlburg was a plant physiologist whose speciality was horticultural botany; however, he was very interested in plant pathology and mycology. He delivered special lectures on plant pathology to a private assembly of agriculturists, and was the first to describe Japanese K5ji-fungus, *Aspergillus oryzae,* under the name, *Eurotium oryzae* Ahlburg.

In 1875 AD, Shinnosuke Matsubara, a member of the above-mentioned assembly, was appointed as a teacher of the Tokyo Medical School and worked as an interpreter for Hilgendorf and Ahlburg. When the name of the school was changed to College of Medicine, Tokyo Imperial University, Matsubara was appointed associate professor. In 1882 AD he published a book on botany, containing one chapter on plant pathology. This may have been the first book concerning plant pathology published in Japan.

The Agricultural College at Komaba in Tokyo was established in 1878 AD. **Hikotaro Nomura** was the first plant pathology specialist in Japan, he introduced the new course of plant pathology at Komaba in 1880 AD, but afterwards this course was abolished as he joined the College of Science at Tokyo Imperial University to teach botany. From 1906 AD, he studied diseases of mulberry trees and silkworms at Tokyo Koto Sanshi Gakko (College of Sericulture). The fungus genus *Nomuraea,* and *Diaporthe nomurai* (the causal fungus of mulberry canker) have been named in his honour.

In 1881 AD, **Chujiro Sasaki** was appointed as a teacher of zoology and botany in the Komaba Agricultural College, where he studied diseases of silkworms and mulberry trees (Shirai, 1918 AD), lectured on plant pathology, botany and zoology. At that time (1882 AD) the Tokyo Botanical Society was established, and in February 1887 AD the first issue of the *Botanical Magazine* was published. This journal, the first botanical periodical in Japan, is still published and has often published papers on plant disease fungi.

Mitsutaro Shirai was a teacher of forest botany and plant pathology in Komaba Agricultural College. At that time the only reference books on plant pathology in the college library were the 1874 AD Sorauer's *Handbuch der Pflanzenkrankheiten* and the first edition (1882 AD) of R. Hartig's *Lehrbuch der Baumkrankheiten.* Therefore, in 1893 AD–1894 AD Shirai published a book of plant pathology, *Shokubutsu By Ori Gaku,* in Japanese language the most authoritative work at that time.

Manabu Miyoshi (1895 AD) worked on lower fungi. He studied the chemotropism of *Botrytis cinerea.* He studied the penetration of *Botrytis* hyphae in gold leaf or a thin film of mica floated on a nutrient solution. This may be the first report on mechanical penetration of the plant cell wall by fungal hyphae.

During this period, many pathogenic fungi found in Japan were identified and described. In the early years of Meiji (1894 AD), cherry trees along the Sumida river in Tokyo were said to be weakened by witches broom *(Taphrina cerasi).* Obuch recommended cutting off infected portions of the cherry trees with recommended therapy so as to recover their vigorous growth. This was probably the first written record of surgery on diseased trees in Japan (Shirai, 1914 AD).

An epidemic of cucumber downy mildew occurred in 1888 AD in Tokyo. **Ichikawa** (Tanaka, 1888 AD) reported it to be a species allied to the late blight fungus of potato. **Tamari** a professor of Komaba Agricultural College also studied this disease. He reported a method for its control and identified the causal fungus as *Peronospora cubensis.* Berk and Curtis, the same fungus found on wild cucurbitaceae in Cuba. Furthermore, Tamari confirmed that the conidia of this fungus germinated by zoospores.

A telegraph service in Japan was established about 1869 AD. A severe decay of wood poles led to their treatment with copper sulphate by the Boucherie process in 1880 AD. Creosote oil injection into railroad sleepers was begun in 1902 AD.

During 1894 AD to the end of the Meiji Era the foundation of plant pathology in Japan was laid. In April 1906 AD, a professorial chair of plant pathology was first provided in the Agricultural College of Tokyo Imperial University, and **Mitsutaro Shirai** was appointed as professor. This was one of the first professorial chairs for plant pathology in any university.

The Sapporo Agricultural College (*Sapporo* Nogakko) was founded in 1876 AD at Sapporo. **William P. Brooks** was invited from America as a teacher in 1877 AD, lecturing on plant pathology for about 12 years. **Shotaro Hori**, afterwards one of the authorities on plant pathology in Japan, was at that time a student in the college.

Kingo Miyabe (born in 1860 AD in Edo, Tokyo) graduated from the Sapporo Nogakko in 1881 AD. The school later was raised to the status of university. In 1886 AD Miyabe studied at Harvard University under Professor W.G. Farlow. After his return to Japan in 1889 AD, he was appointed professor at this school and lectured on botany and plant pathology. In 1907 AD, the year following establishment of a chair for plant pathology in Tokyo Imperial University, a similar chair was provided in the College of Agriculture of Tohoku Imperial University in Sapporo (afterwards renamed Hokkaido Imperial University), and in 1920 AD another professorial chair was provided. Kingo Miyabe and Seiya Ito occupied the two chairs, and Miyabe trained many plant pathologists.

In 1899 AD, the section of plant pathology in the Imperial Agricultural Experiment Station at Nishigahara, Tokyo (now the National Institute of Agricultural Sciences, Nishigahara) was established, and **Shotars Hori** and **Yeijiro Uyeda** were appointed leaders of the mycological and bacteriological laboratories, respectively. The government organisation for prefectural agricultural experiment stations was announced officially in 1893 AD, and between 1884 AD and 1911 AD an agricultural experiment station was established in every prefecture. Noteworthy experiments were carried out in this period, and the important contribution were made.

5.5.1 Discovery of flower infection by loose smut of barley

Formerly, loose smut of barley was thought to develop from seed-borne chlamydospores, and hot water or chemicals were used to disinfect seeds. Beginning in 1892 AD, **Sato and Yamada** carried out experiments in Kyoto prefecture to control loose smut of barley. After several experiments they

found that when plants were inoculated at flowering time, a large number of diseased plants resulted from the seed produced. Thus, it was recorded for the first time that flowers were infected by loose smut in barley. Independently, **Maddox** confirmed flower infection by loose smut in wheat in Japan, thus, establishing a scientific basis for Jensen's hot water seed treatment for the control of this disease. Furo-Yu Hitashi Ho (hot-water seed treatment) for loose-smut control was devised in 1896 AD by Kanjirs Shinohara and good results were obtained.

5.5.2 Discovery of insect transmission of rice dwarf virus

In the early years of Meiji era, a severe outbreak of dwarf disease in rice occurred in the Kansai district, especially in Okayama. According to Ishikawa dwarf disease of rice plants was first noticed in the Shiga prefecture by **Hatsuzo Hashimoto**, a conscientious rice grower. There was an epidemic of the disease in the Shiga prefecture in 1892 AD. He began making detailed observations of this disease in 1883 AD and suspected that the disease was in some way related to leafhoppers. In 1894 AD Hashimoto experimentally proved the causal relation of leafhoppers to rice dwarf, but did not publish the results, and the leafhoppers he observed were not identified. **Takata** gave the first report in 1895 AD of the relationship between the insect and the disease, and the leafhopper involved was identified as *Inazuma dorsalis* (Fukushi, 1935 AD). However, he did not discover that the disease was caused by a virus of which *Inazuma dorsalis* was a vector.

In 1895 AD the Shiga Agricultural Experiment Station was established, and Takata was appointed director. Research on virus transmission by insects was continued for more than 20 years. In 1900 AD, the Shiga Agricultural Experiment Station published reports that *Nephotettix cincticeps* (Tsumaguro-Yokobai) was the true cause of dwarf disease. Substantiating Takata's early claim, **Fukushi** later confirmed that *Inazuma dorsalis* was also a vector. During 1902 AD–1908 AD the Imperial Agricultural Experiment Station and the Shiga Agricultural Experiment Station found both infective and non-infective leafhoppers and also found that non-infective leaf hoppers from Tokyo became infective after feeding on diseased plants. From this came the conclusion that rice dwarf was not caused by leafhoppers but by certain unknown agents carried by them. This is now considered to be the first plant virus shown to be insect transmitted.

5.5.3 Confirmation of alternation of hosts in Cronartium quercuum

The phenomenon of alternation of hosts in rust fungi was first confirmed by **Oersted** and **DeBary** in 1864 AD–1865 AD. In Japan 1889 AD, **Shirai**

proved experimentally the alternate relationship in pine gall fungus, *Cronartium quercuum*. Shirai observed many galls produced on pine trees close to deciduous *Quercus acutissima*, *Q. serrata*, and *Q. variabilis.* In April 1898 AD, he inoculated aeciospores obtained from pine galls onto leaves of Quercus. Uredosori and teliospores developed on the lower surface of the leaves. The teliospores germinated immediately after they matured, forming sporidia. Thus, Shirai proved the genetic connection between Peridermium stage formed on pine galls and Cronartium stage on Quercus leaves.

5.5.4 Epiphytotic of false smut (Inakoji Byo) of rice plant

The occurrence of false smut (green smut) was long believed to be a sign of a heavy crop year, and severe occurrence was said to be conspicuous in years when weather conditions were favourable for growth of rice plants. During 1894 AD–1907 AD there were several epidemics of the disease. The fungus was first described by Cooke in 1937 AD as a smut fungus and named *Ustilago virens*, based on the specimen from India. The fungus had been described at a very early date in China, but not given a scientific name. **Tanaka** (1889 AD) had previously assumed the fungus to be *Ustilago virens* Cooke, but **Takahashi** transferred it to *Ustilaginoidea virens* (Cooke) Takahashi, and since then, this name has been generally accepted. **Yamashita** (1934 AD) detected alkaloids in infected grains and reported that the sclerotia of *Ustilaginoidea virens* resembled the ergot formed by *Claviceps*.

5.5.5 Bakanae disease of rice plants

Bakanae disease (characterised by elongation of seedlings) has been known from ancient times. In 1828 AD **Konishi** recorded its occurrence in his book ***Nogyo Yowa***, although he did not know its origin. **Hori** first reported that it is a parasitic disease, but erroneously identified the causal fungus as *Fusarium heterosporum Nees*, with the comment that he had some doubt about this identification. In 1916 AD, **Yosaburo Fujikuro** found the perfect stage and it was named *Lisea fujikuroi* by **Sawada** (1919 AD). **E. Kurosawa** showed in 1926 AD that culture media in which the fungus had been growing would induce the disease in rice plants. The active component, gibberellin, was isolated from the fungus and named by **T. Yabuta** in 1935 AD. The fungus was later put in the genus *Gibberell*a under the name of *G. fujikuroi* (Sawada) Wr. (Ito and Kimura 1931 AD).

6

Plant pathology in present era

The major trends, initiated in the latter half of the 19th century, progressed in several directions. The rediscovery of Mendel's laws of heredity in the beginning of this century had its impact, and the genetics of the host and the pathogen attracted attention of plant pathologists. The science of plant pathology grew multifarious in its scope. Some of the major aspects of study can be summarised as follows.

6.1 In Europe

The most significant contribution in different areas of pathology during present era are:

6.1.1 Advancement in phytobacteriology

Phytobacteriology after Smith's death (1927 AD), began to wane and could not keep stride with the advances in animal bacteriology or microbiology. However, it was strengthened in the second half of the century by **Braun** and his colleague **Pringle**, who proved that the crown gall bacterium *Agrobacterium tumefaciens,* caused transformation of plant cells, leading to their autonomous, rapid growth in culture. Today's enormous investment in plant biotechnological industry has its origin in Braun's work. He provided basics for the later discoveries, that the DNA transferred from the bacterium to the plant cell was the tumour-inducing principle. *Agrobacterium* was later used to introduce foreign genes into crop plants and more efficient methods of introducing DNA into plant cells are available now.

6.1.2 Bacteriophages

Frederick Twort (in 1915 AD) and **Felix d'Herelle** (in 1917 AD) were the first to recognise viruses which infect bacteria, which d'Herelle called bacteriophages (eaters of bacteria).

In the 1930 AD onwards pioneering virologists such as Luria, Delbruck and many others utilised these viruses as modelsystems to investigate many aspects of virology, including virus structure, genetics, replication, etc.

Frederick Twort
(1877–1950)

Felix d'Herelle
(1873–1949)

6.1.3 Advancement in plant virology

Early studies in TMV in the last few years of the 19th century led to the virus concept of plant diseases. **Zaitlin** and **Palukaitis** (2000 AD) have described the virus research in the 20th century into three phases: **the Classical Discovery Period** (the first 50 years, 1906 AD–1950 AD), **an Early Molecular Era** (1951 AD–1983 AD), and **the Recent Period** (1984 AD to present).

6.1.4 Classical discovery period (1906 AD–1950 AD)

A number of plant diseases were attributed to viruses, based on their mechanical or graft transmission and to the fact that no specific disease agents

were microscopically identifiable. **Holmes** in 1939 AD listed 129 plant viruses (in 1999 AD, the list has grown to 977 species). The other important events of this period were the discovery of cross-protection, development of bio-assay for TMV, crystallisation of TMV**,** discovery of RNA (ribo nucleic acid) in TMV, and elucidation of structure of TMV by X-ray crystallography.

***Cross protection*: Mc Kinney** (1929 AD) observed that tobacco infected with a virus causing a mild mosaic disease was protected against a more severe form of that virus. Cross protection was used to establish virus relationships, as only related viruses would show the response.

Development of bio-assay for TMV: The local lesion assay, developed by **Holmes** (1927 AD) enabled to quantify the virus in leaf extracts. This is the only assay that gives a count of viable virus particles. The other techniques of assay using serology and PCR (polymerise chain reaction) do not distinguish between viable and non-viable viruses.

Discovery of RNA in TMV: Bawden and *Pirie* (1938 AD) set the record straight by reporting the presence of RNA and with this the chemical nature of the virus was finally clinched. It was ribonucleoprotein. Other viruses purified later, like *Potato virus X* (PVX), and *Tomato bushy stunt virus* (TBSV), were also ribonucleoproteins.

Elucidation of structure of TMV: Rosalind Franklin (1955 AD), by X-ray crystallography, demonstrated the structure of TMV. The rod-shaped virus consisted of protein molecules tagged on an inner helical RNA so as to form a closed spiral.

6.1.5 Early molecular era (1951 AD–1983 AD)

This era saw many intellectual jumps, the more important being the discovery of RNA as the genetic material, sequencing of TMV coat protein, development of viral chemotherapy, elucidation of subgenomic RNAs, genome organisation and gene function, discovery of defective-interfering virus and sub-viral pathogens *viz.*; satellite viruses and viroids, use of protoplasts in plant virology, *in vitro* replication and replicases, and improvements in diagnoses.

RNA as the genetic material

Gierrer and Schramm in 1956 AD showed that the protein could be removed from the virus and that the RNA carried all the genetic information that

enabled it to cause infection and to reproduce the complete virus. It was shown subsequently that although the nucleic acid of most viruses infecting plants is single-stranded RNA, some viruses have double-stranded RNA, some double-stranded DNA, and some single-stranded DNA.

Viral chemotherapy

The 1950 AD decade saw an intense effort to cure plant viruses by chemotherapy, using nucleotide analogs (Matthews and Smith, 1955 AD). Unfortunately, the compounds damaged the host. It was beginning to look unlikely that commercially effective antiviral agents for plants will be found. **Matthews** (1987 AD), proved the use of apical meristem culture, often including a heat treatment, to get rid of viruses, but there is no chemotherapy.

In 1975 AD, **Kohler and Milstein** isolated the first **monoclonal antibodies** from clones of cells selected *in vitro* to produce an antibody against not only the whole virus, but at specific regions – epitopes – of individual virus antigens. In recent years, this ability has greatly increased our understanding of the function of individual virus proteins.

The sub-genomic RNA concept was established, when it was found that smaller, sub-genomic RNAs, associated with the virus were formed in infected plants, coded for the capsid proteins. The *in vitro* studies of translation of RNAs indicated that the viral's RNA behaved as messenger RNA (+ve sense nature). Later, I negative (-ve) sense and some ambisense (+, -) were reported. Double stranded RNA viruses were added to the list of plant viruses. Until 1968 AD, all plant viruses that were known were found to be RNA viruses. **Shepherd** et al. (1968 AD) reported double-stranded DNA in *cauliflower mosaic virus,* followed by the discovery of single-stranded DNA Geminiviruses by **Goodman** (1977 AD). Both the DNA viruses are used in plant genetic engineering.

Viroids: Diener, in 1971 AD, discovered a strange and new kind of plant pathogen associated with potato spindle disease, which he termed viroid. It paled off viruses into insignificance in its smallness of size and ingenuity of parasitism. Its physico–chemical structure has been worked out, though its replication and pathogenicity remain baffling. Its placement in and out of viruses has generated considerable controversy.

6.1.6 Recent period (1984 AD onwards)

The development and utilisation of technologies that allow viruses to be manipulated, distinguishes this period from the earlier periods. The recent

period is characterised by the ability to: (1) modify plant virus genomes, (2) detect nonstructural gene products, (3) determine the functions of viral gene products, and (4) modify plants using viral-sequences, providing novel types of resistance.

6.1.6.1 Enzymes and toxins in pathogenesis

The study, initiated by De Bary and Ward, was pursued vigorously by **Jones** who did pioneering work on pectic enzymes secreted by soft rot bacteria.


W. Brown


E.A. Gaumann


T.S.Sadasivan

Brown's series of papers on *Physiology of Parasitism* cut many new grounds and gave leads, which are still being pursued. By mid-1950 AD onwards investigations on the role of enzymes and toxins in disease started with exceptional zeal. **Gaeumann** in Zurich, **Walker** at Wisconsin, **Dimond** at New Haven, **Wood** at London and **Rubin** in USSR, contributed extensively to elucidate the role of enzymes and toxins in pathogenesis.

The studies on enzymes got a new dimension with the elucidation of cell wall structure by **Albersheim** and his associates (Keegstra et al., 1973 AD). The cell wall model showed specific covalent connections between the various polysaccharides of the cell wall *viz*.; cellulose, hemi-cellulose (glucan and xylan) and pectic polymers (Rhamnogalacturonan, homogalacturonan, araban, galactan and arabinogalactan). The studies on cell wall degrading enzymes had to be looked at a fresh, keeping in view the new polysaccharide fractions of hemi-celluloses and pectic polymers, which were no more polymers of only galactose. Cell wall fractions, called oligosaccharins by Albersheim, were shown to act as regulatory molecules or chemical signals (elicitors) for gene expression in phytoalexin synthesis, besides modulating growth, morphogenesis and reproduction.

Studies of toxins in plant disease were going on simultaneously with enzymes. After elucidation of fusaric acid in 1955 AD by **Sadasivan's** group as a vivotoxin, work on toxins went very fast with victorin and other host-specific toxins, especially those produced by *Alternaria* sp and *Cochliobolous*

(Helminthosporium) by **Pringle, Scheffer, Wheeler, Hanchey, Black, Sammadar** and several others. The great interest in mode of action of fungal and bacterial toxins resulted in the demonstration that: 1) some of the toxins were primary determinants of pathogenicity, and 2) the susceptible varieties contained receptors for toxins, which were absent or blocked in resistant varieties.

Y Silva et al. (2013 AD) worked on expression of a microbial serine proteinase inhibitor gene which enhances the tobacco defence against oomycete pathogens.

6.1.6.2 Genetics of host and pathogen in disease resistance

Biffen, in 1905 AD, proposed that: (i) resistance to yellow rust in wheat was inherited as a single dominant gene, like any other Mendelian character, and (ii) the resistant genes could be put into elite agronomic cultivars by back-crossing. This initiated work in relation to inheritance of resistance and virulence. It led to breed the resistant varieties for disease control. But when **Biffen's** rust-resistant varieties succumbed to infection in Australia, there was a wave of disbelief in genetically-bound disease resistance. However, the discovery of the physiological races of the pathogen broke the cobweb.

Biffen

Advances in the knowledge of sexuality of fungi by **Blakeslee** (1904 AD), **Cragie** (1927 AD) and **Dodge** (1927 AD) enabled genetic study of pathogenic fungi. The genetic studies with bacteria and viruses pushed microbial genetics to the forefront of life sciences.

6.1.6.3 Cloning of avirulence gene, and elucidation of gene product (avr protein)

Staskawicz and colleagues (1984 AD) cloned the first avirulence gene from *Pseudomonas syringae* pv. *glycinea* Race b, which when transferred to other races of this bacterium, conferred the ability to elicit a resistant response on soybean cultivars that have the resistant gene *Rpg 2*.This demonstrated that *avr* genes impose race specificity on a pathogen. By 1996 AD, over 30 such avirulence genes had been cloned from bacterial pathogens. The first fungal avirulence gene, *avr* 9, from *Cladosporium fulvum* was cloned in 1991 AD by **De Wit** and colleagues.

The products (proteins) of the avirulence genes serve as elicitors of resistance reaction in the resistant host lines. First such product was reported by Keen et al. (1990 AD) for *avr d* gene of *P. syringae* pv. *tomato* during interaction with resistant soybean lines. Several *avr* gene products (elicitors) have been isolated from different pathovars of *P. syringae* and *Xanthomonas.* Their mode of reaction remains unknown, but they do elicit hypersensitive response causing rapid death of host cells.

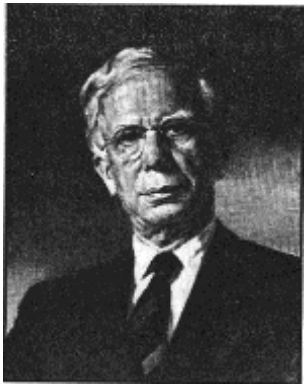
6.1.6.4 Cloning of resistance genes and elucidation of gene products

Several plant resistance genes, providing resistance against fungal, bacterial, and viral pathogens, have been cloned in the last decade, and their gene products have been examined for their mode of providing resistance. The first cloning of the resistant gene, *HM 1* from maize, was accomplished by **Johal** and **Briggs** (1992 AD). The *HM 1* gene provides resistance to *Cochliobolus carbonum* Race 1. It codes for a reductase that inactivates the toxin produced by the fungus. (This was, thus, not a classical *avr*-induced gene resistance). Soon (1994 AD), the second resistance gene *Pto,* which provides resistance to tomato against *Pseudomonas syringae* pv. *tomato*, was cloned. The gene encodes a protein similar to serine–threonine protein kinases. In 1998 AD, **Van Gigsegen** et al., identified a large gene cluster, the hip region in *Ralstonia solanacearum* that governed either the induction of hypersensitive response in non-hosts or the development of disease in susceptible species. Such *hip* regions have now been identified in several genera of plant pathogenic bacteria.

Only two plant genes encoding resistance to plant viruses have been isolated: (1) the *N* gene, specifying a hypersensitive response to TMV in several *Nicotiana* spp. (Whitham et al., 1994 AD), and (2) *Rx* gene from potato encoding extreme resistance to *Potato virus X* (Bendahmane et al., 1999 AD). The products of the *N* gene were counterparts of toll and interleukin receptors, whose importance in animal's innate immunity has only recently been recognised. Both *N* and *RX* genes also, like plant genes resistant to fungi and bacteria, contain conserved sequences, such as leucine-rich repeats, and several kinase domains, referred to as nucleotide binding site.

6.1.6.5 Fungicides in plant disease control

Research on fungicides developed on a fast pace. The team work of plant pathologists and chemists threw new light on pathogen-toxicant relations. The mechanism of fungicide action was explored by **Sisler**, **Cox**, **Ludwig**, **Lukens** and some others. Horsfall's book (1956 AD) entitled *Principles of Fungicidal Action*, distilled the available knowledge into a compact mass of theory.

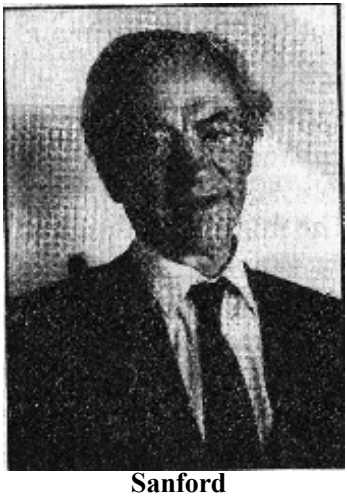


J.G. Horsfall

J.G. Horsfall: The sulphur and inorganic copper compounds were used as fungicides in the 19th century and it is remarkable that they still account for the larger part of the arsenal of plant pathology (**Rheim**, 1912 AD). **Tisdale and Williams** (1934 AD) revolutionised the world fungicide market by addition of organic mercurials to the field of organic fungicides by discovering the alkyl-dio-thiacarbamates that outranked all other fungicides. Alkylene bis-dithiacarbamates, the most widely used fungicide, was discovered by **Dimond, Heuberger and Horsfall** (1943 AD). The use of systemic fungicides started with oxathiins, was developed by **Schmeling and Kulka** (1966 AD). Now several systemic fungicides are available for use as therapeutants. The initial enthusiasm with antibiotics proved short-lived, which is evidenced by the fact that only 0.1% of the total antibiotics produced are used for plant disease management. The antibiotics that are used since long are streptomycin and oxytetracycline.

Nelson B. Lima et al. (2014 AD): They worked on comparative epidemiology of *Colletotrichum species* from mango in north-eastern Brazil. Different species exhibited different thermal requirements for maximum virulence in the fruits. Reduced mycelial growth observed in the presence of thiophanate-methyl, difenoconazole and azoxystrobin fungicides.

6.1.6.6 Biological control



Sanford

The term biological control was introduced by **Von Tubeuf** (founder of plant pathology in Europe), though it has been in practice since ages in the form of crop rotation and green manuring. It is another example of *art preceding science*. It was **Sanford** who in 1926 AD suggested that control of potato scab by green manuring was due to biological control, brought about by fall in population of saprobic bacteria. Thus, biological control entered plant pathology in a subtle way, but later it showed up complexities. Important contributions in this field were made by **Garrett, Baker, Cook, Ralph Baker, Chet and Kerr**.

6.1.6.7 Biochemical disease resistance

The chemicals responsible for resistance have always been most fascinating subject of study from the beginning of the century. **Magnus** (1900 AD), **Orton** (1908 AD), **Bernard** (1909 AD) were aware that anatomical features and special structures of plants were not of much consequence. They believed that the secret of resistance resided in physiological activities of plants, and displayed through substances toxic to the parasite *viz*.; alkaloids, glucosides and other secondary metabolites. **Hermann, Farkas** and **Kiraly**, and **Kuc** have made important contributions in the field of phenolics in plant disease resistance.

Rémus-Borel et al., 2005 AD stated that Si-mediated resistance to a pathogen is associated with higher Si deposition in leaves. In effect, this resistance can strengthen the mechanical barrier and activate biochemical defence responses by increasing the activities of defence related enzymes and the accumulation of antifungal compounds.

6.1.6.8 Hypersensitive reaction (HR)

Cruickshank and Perrin (1960 AD) isolated pisatin from pea pods, which ignited a flurry of interest in disease resistance. Two types of HR-inducing proteins, elicitors and harpins (products of avirulent bacterial genes), serve as signal to HR. The HR is now known to have intriguing parallels with humoral defense in *Drosophila* and innate immunity in vertebrates. It is very similar to programmed cell death in animals.

6.1.6.9 Systemic resistance (SR)

In 1961 AD, **Ross** observed *systemic resistance* in contrast to localised hypersensitive reaction and called it induced resistance. It is the phenomenon that a plant, once appropriately stimulated (by primary pathogen), exhibits an enhanced systemic resistance subsequently to a broad spectrum of

challenging pathogens. Because of this, SR induced resistance is commonly referred as systemic acquired resistance (SAR). Induced resistance has been synonymously used with acquired resistance, acquired immunity and immunisation (Kuc, 1983 AD).

Another type of induced resistance was reported by **van Peer** et al., in 1991 AD, when strains of non-pathogenic, root-colonising and plant growth-promoting rhizobacteria (PGPR) which were shown to induce systemic resistance in carnation and cucumber. To distinguish this enhanced defensive capacity, the induced protection was termed induced systemic resistance, ISR (Pieterse et al., 1996 AD; van Loon, 1997 AD).

SAR requires salicylic acid as the signal molecule and is associated with accumulation of pathogenesis-related proteins (PR-proteins). The mobile signal for SAR might be a lipid molecule (Durrant and Dong, 2004 AD). The ISR differs from SAR as it lacks the SA-signalling pathway and non-accumulation of proteins. SA pathway is substituted by plant hormones jasmonic acid and ethylene as signal molecules (van Loon et al., 1998 AD). Cross-talk between the two pathways (SA-dependent and SA-independent) provides great regulatory potential for activating multiple resistance mechanisms in varying combination.

6.1.6.10 Biochemistry and physiology of the diseased plant

The various metabolic processes affected have been pried to great depths to unravel the sequence of events in pathogenesis. The general conclusions are that in the diseased state the tissues disintegrate, growth and reproduction are adversely affected, host starves, water becomes deficient, and respiration is altered. All these studies have proved that the diseased state has a biochemistry of its own. The techniques are known how biochemistry and physical sciences have greatly contributed to these studies.


Waksman

6.1.6.11 Ecology of soil-borne plant pathogens

The root disease investigators realised that they were dealing not with one biological relationship between host and pathogen but with many microbial interactions as well. **Tabenhaus** and **Ezekiel** discovered in 1930 AD that the root disease fungus *Phymatotrichum omnivorum* could not grow in soil although it infected the finest rootlets. This observation had a great

future in plant pathology. It is said that the success in research depends on asking good questions. **Waksman** 1917 AD asked, Is there any fungus flora of soil? This succinctly suggested the state of knowledge about soil fungi at that time. **Waksman** in 1932 AD emphasised the important role that fungi played in the economy of the soil. Till then the fungi were not known to have any role in the decomposition of organic matter in the soil. The fungal spores present in the soil were thought to have drifted from the air. Later, fungi were proved to actually precede bacteria in colonising the dead and dying plant residues. Waksman determined which soil fungi were an integral part of the general soil microbiota and which were exotic. (Waksman was awarded Nobel Prize in 1943 AD for his discovery of streptomycin).

Reinking and **Manns** in 1933 AD observed that certain species of *Fusarium* were found in all the soils, while others were restricted to few localities only, dependent upon certain factors like presence of the host plants and soil conditions. The first group of species they named as soil inhabitants and the latter as soil invaders. **Garrett** (1950 AD) realised that this pattern of distribution could be made on the basis of distinguishing the soil fungi into two groups: (1) *soil inhabitants*, and (2) *soil invaders*. He renamed the soil invaders as root inhabiting fungi having little or no saprophytic ability to survive in soil. The two groups were distinguished by their competitive saprophytic ability. The concept of inoculum potential originated from root disease investigation and was consolidated by **Garrett** in 1956 AD.

The interesting field of rhizosphere studies was initiated in 1904 AD by **Hiltner. Lochhead** et al. (1940 AD) got evidence that the rhizosphere flora influenced the resistance and susceptibility of plants to root infections. Since **West's** (1939 AD) study of root exudates from flax seedlings, considerable work has been done on root to show their selective activity on rhizosphere microbiota. The important discovery of formation of antibiotics in soil *in situ* was of far reaching significance. Fungi static factors present in secretions of seedlings prevent spore germination in their vicinity.

There is much change in the limits and scope of rhizosphere since Hiltner defined the term as the zone of stimulated bacterial growth around legumes. Rhizosphere today is regarded as the zone of microbial proliferation in and around roots. It includes *ectorhizosphere* (microbes around roots), *rhizoplane* (on root surface), and *endo-rhizosphere* (in root epidermis-cortex zone). **Dommergues** (1978 AD) classified rhizosphere organisms as *beneficial* (symbiotic), *harmful* (pathogenic), and *neutral* (having no effect on the plant). **Salt** (1979 AD) coined the term minor pathogens for organisms, which were not necessarily parasites, and did not cause distinct symptoms. **Klopper and Schroth** (1978 AD) and **Suslow** et al. (1979 AD) coined the term rhizobacteria

for rhizosphere bacteria that can actively colonise the roots. The rhizobacteria have been studied extensively for their use in biological control and plant growth promotion.

6.1.6.12 Fastidious vascular pathogens

Mycoplasmas and rickettsia-like organisms: Mycoplasma-like organisms (MLOs) were discovered in 1967 AD by **Doi** et al. as causal agents of dwarf mulberry, potato witches broom and Aster yellows. These diseases till then were known as viral diseases. Electron microphotographs of thin sections of the diseased tissues showed the presence of wall-less, pleomorphic, mycoplasma-like bodies in the phloem. The same year the same group showed that the mycoplasma-like bodies and symptoms disappeared temporarily when the plants were treated with tetracycline antibiotics. Since then, MLOs that infect plants have been reclassified as phytoplasmas, and some of them that have helical bodies and are found in other environments besides plants are known as spiroplasmas.

Ishiie et al. (1967 AD) found that the symptoms of the diseases in Euphorbia plant disappeared on tetracycline treatment, but Penicillin had no such effect. These results and the typical mycoplasmal morphology strongly suggested that the causal agent was a wall-less, mycoplasma-like organism. It was a distinct surprise to the scientific world that the yellows group of diseases was caused by mycoplasmas. It opened a new field of mycoplasmatology in plant pathology. The technique introduced by Japanese workers *viz*.; electron microscopic examination of thin sections of diseased tissues for wall-less pleomorphic cells, and remission of symptoms on tetracycline treatment, have remained standard procedures for studying mycoplasmal etiology of plant diseases. The contribution of Doi, Ishiie and their colleagues were recognised by the award of a citation by *International Organisation of Mycoplasmatology* in 1982 AD.

The next major advancement was the culture of the MLO causing the citrus stubborn in 1971 AD by **Sagglio** et al. in France and Fudl-Allah et al. in USA. The MLOs appeared spiral in shape and were motile. **Davis and Worley** (1972 AD) observed a large number of similar helical and motile wall-less cells in the phloem sap of corn stunt diseased maize plants. **Davis and Worley** (1973 AD), inspired by the helical shape, coined the trivial name spiroplasma for this organism. When it was found that the citrus stubborn and corn stunt organisms were same; the term spiroplasma was used as the generic name, and the stubborn agent was designated as *Spiroplasma citri* (Saglio et al., 1973 AD). The corn stunt organism too was taken in axenic culture in 1974 AD. Such structures have been detected in leaf hoppers too, which serve as vectors.

Spiroplasmas have attracted the attention of cell biologists because of their helical morphology and unique type of motility achieved without the

support of a rigid cell wall. But all plant MLOs was not *Spiroplasma*. The non-cultivatable MLOs, which are not helical in thick sections of diseased tissues, are called by the trivial name phytoplasma. Since they are well characterised by molecular methods but not yet cultured, they are given the status of a *Candidatus* genus. Now, phytoplasmas are associated with over 200 diseases of crop plants.

After the discovery of plant mycoplasmas, the possible role of chlamydiae, rickettsiae and other groups of plant pathogens were speculated. But, unexpectedly flagellated protozoans were the next addition to the list of plant pathogens (Parthasarthy et al., 1976 AD). The concept of protozoan plant pathogens was not new to plant pathology, as these were originally reported as the cause of phloem necrosis of coffee by **Stahel** (1931 AD). However, it was forgotten until several reports appeared on coconut palms and oil palms. At present these phloem-inhabiting plant pathogens are restricted to the genus *Phytomonas*, whose five species are reportedly causing these diseases. (The phytopathogenic baterium, named *Phytomonas* in 1930 AD onwards was invalidated when it was found to be homonym of this protozoan.) Phloem-feeding homoptera are highly suspected to be the vectors of these pathogens (McCoy, 1982 AD).

Later, **fastidious bacteria**, strictly limited to xylem or phloem, were reported by **Hopkins** (1977 AD), **Nienhaus and Sikora** (1979 AD) and **Purcell** (1979 AD). These were not culturable on bacteriological media and their nature was revealed by electron microscopy. These were referred to as Rickettsiae-like organisms (RLOs). **Davis and Whitcomb** (1981 AD) called them eubacteria of unknown taxonomic affinity. These organisms appear to be serologically related to each other, but not to any existing taxa. **McCoy** (1982 AD) suggests that the term rickettsia-like should be dropped from future plant pathological literature, and all these organisms inhabiting the phloem or/and xylem tissues as special niche, should be designated as **fastidious vascular pathogens**. The common denominator of this group of organisms is their restriction to specific vascular tissues, and their fastidious nature, requiring highly complex and specific media for cultivation *in vitro* or, as in many cases, remaining uncultivated.

6.2 In America

6.2.1 On fungal, viral and bacterial pathogen and diseases

The noteworthy contributions in different areas of pathology during present era are of:

George F. Atkinson (1854 AD–1918 AD)

Atkinson was born on January 26th, 1854 AD in Raisinville, a small town in Monroe County, Michigan. Atkinson's research at Cornell spanned a broad range of topics, focusing mainly on fungi. Though best known for his work on mushroom taxonomy, he also studied plant diseases, made careful studies of *Endogone* species, studied evening primrose hybridisation, and speculated on evolutionary processes. He made careful developmental studies of a number of mushrooms, characterised chytrids, and elucidated the life cycles of plant pathogenic fungi. One paper addressed the intelligence of chytrid zoospores. He described several poisonous mushrooms for the first time, including *Amanita bisporigera*, the eastern Angel of Death. He rarely took credit via authorship in his students' work. He was an administrator for most of his time at Cornell, instead he must have limited his time for research, but this is not apparent in his strong publication record. He was also an active and effective teacher, a renowned expert on botany and mycology. He wrote several textbooks for college and high school students of botany, as well as articles about fungi for popular newsletters.

Atkinson was a pioneer in the use of photography for documenting fungi. He believed it to be a key tool in the standardisation of identification and determination. For mushrooms, which dried to shriveled shadows of their fresh selves, photographs are especially useful in documenting living specimens.

He corresponded extensively with **Charles Horton Peck** (1833 AD–1917 AD), who during his appointment as New York State Botanist described over 2700 species of American fungi, largely in isolation from European studies. Atkinson felt it was important to reconcile the similarities and differences among European and American fungi.

William Stanley

William Stanley, Jr. (November 28, 1858 AD–May 14, 1916 AD) was an **American physicist** born in Brooklyn, New York. In his career, he obtained 129 patents covering a variety of electric devices. He found the crystallised nature of TMV and showed that crystals were proteins. He was awarded with Nobel prize in 1886 for transformers.

Elvin Charles Stakman

Stakman (May 17, 1885 AD–January 22, 1979 AD) was an **American** plant pathologist who was a pioneer of creating methods for identifying and combatting diseases in wheat.

He also had a major hand in influencing **Norman Borlaug** to pursue a career in phytopathology. In 1938 AD, in a speech entitled "These Shifty Little Enemies that Destroy our Food Crops", Stakman discussed the manifestation of the plant disease rust, a parasitic fungus that feeds on phytonutrients, in wheat, oat and barley crops across the US. His discovery on special plant breeding methods created plants resistant to rust. His research greatly interested Borlaug, and when Borlaug's job at the forest service was cut short due to budget cuts, he asked Stakman if he should go into forest pathology. Stakman advised him to focus on plant pathology instead, and Borlaug subsequently re-enrolled into the University of Minnesota to study plant pathology under Stakman. Borlaug went on to discover varieties of dwarf wheat that helped to reduce famine in India, Pakistan, and other countries. Borlaug received the nobel peace prize for his work in 1970 AD.

Stakman (1914 AD onwards) contributed valuable information on delineating and cataloguing of physiological races.

Flora W. Patterson: The first woman mycologist at the USDA

Early in her career Flora Patterson published one of the few accounts of the leaf parasitic fungi belonging to the Exoascaceae that attacked living leaves. These fungi are now known as species in the genus *Taphrina* causing such diseases as peach leaf curl. The delicate line drawings of the naked asci accurately represent the structures still used in identifying these fungi. Patterson identified about 800 specimens in the U.S. National Fungus Collections. Many of these specimens were microfungi on exotic tropical plants grown in the USDA greenhouses, newly discovered pathogens on crop plants, or fungi found during inspection of agricultural commodities at ports of entry. Based on the specimens obtained while inspecting newly imported commodities, Patterson described a number of new species and one new genus. The cause of witches' broom of bamboo was described as a new genus and species, *Loculistroma bambusae.* She and her colleagues worked to develop control measures such as fumigation to control pineapple rot caused by *Thielaviopsis paradoxa*. Her most widely distributed publications were two bulletins, one entitled Mushrooms and Other Common Fungi and the other Some Common Edible and Poisonous Mushrooms, in which common fleshy fungi were described.

Beverly Thomas Galloway

The papers of Beverly Thomas Galloway, 1891 AD–1995 AD, occupy 4 linear feet, or 9 manuscript boxes, 218 folders, and 400 items. The collection was

donated to the National Agricultural Library on December 7, 1970 AD by Robert T. Galloway, the grandson of B.T. Galloway. Robert T. Galloway made an additional donation of a photograph album on October 25, 1995 AD. The articles and reports (Series III) reflect the research activities of Galloway as plant pathologist, and later as Chief of the Bureau of Plant Industry in the Department of Agriculture. He propagated the activities of the Bureau of Plant Industry, and discussed many agricultural issues of the American nation. His publications became especially numerous when he was Assistant Secretary of Agriculture (1913 AD–1914AD) and Dean of the New York State College of Agriculture, Cornell University in Ithaca, N.Y. Subjects of his publications included diseases of plants, protection of plants against alien enemies, marketing problems, and physiological and biochemical studies of plant pollens and their relation to certain allergic diseases.

Harold J. Brodie (1907 AD–1989 AD)

He joined the teaching staff on September 1, 1935 AD. Brodie, who eventually became a world authority on bird's nest fungi, taught mycology from 1935 AD until 1937 AD. During that period, he became interested in powdery mildews of grain (Brodie 1945 AD), and he assisted Coulson in the teaching of plant diseases. Brodie was the most highly trained mycologist, plant pathologist at McGill University.

Dickson

Dickson expanded undergraduate course options in plant pathology to include one on research in plant pathology. He also designed courses for graduate students because from 1921 AD onwards, postgraduate courses could be given only at Macdonald College. These included History of Plant Pathology, Pathologic Plant Histology, Diseases of Field Crops, Diseases of Fruit Crops and Diseases of Forest Trees and Timber. In 1922 AD, Advanced Plant Pathology was added, and there was a seminar in plant pathology. In addition, a reading comprehension of Latin, French and German was required. At that time, all postgraduate work was carried out under the auspices of McGill University's Committee on Graduate Studies.

Henry Luke Bolley

Henry Luke Bolley was the first plant pathologist in the North Dakota Agricultural Experiment Station. His work with soil borne pathogens was marked by his explanation of Flax Sick Soil, identifying the disease as being caused by Fusarium oxysporum (Schlecht. f. sp. lini (Bolley) Snyd. and Hans) and the root rot hazards from continuous cropping of wheat (caused

by Helminthosporium sativum). There are plots on campus, originally established by Bolley, that are still used to evaluate new germplasm and study these diseases. The flax wilt plot (Plot 30) was 100-years-old in 1997 and has been used continuously over the period of time. Bolley, truly one of the pioneers of plant pathology, also worked extensively behind the scenes to promote the USDA barberry eradication program.

George Baker Cummins (born on August 29, 1904 AD at Tecumseh (Nebraska))

He was a notable American mycologist and was considered an authority on the rust fungi. At his death on March 30, 2007 AD at Tucson (Arizona), he was the last surviving charter member of the Mycological Society of America.

George Baker Cummins was affectionately known as Mr. Rust and he was recognised throughout the world as the authority on the rust fungi, the Puccinales, which are the largest order of disease-causing organisms of plants. Cummins professional specialty for almost his entire career was the taxonomy, biology and geographic distribution of the rust fungi. His investigations of the rusts took him to the Philippines, New Guinea, continental China, the Himalaya, central and western Africa, and North and South America.

Outstanding contributions by Cummins included a study of the phylogenetic significance of the pores in rust urediniospores, a monograph of the genus Prospodium, an illustrated manual of rust genera, and studies of major groups of grass rusts, some cooperatively with H.C. Greene and J.F. Hennen (Baxter, 1962 AD).

Cummins was also a talented scientific illustrator and J.C. Arthur's 1934 AD *Manual of the Rust Fungi* contains his illustrations. There are hundreds of original illustrations by Cummins throughout the collection in the Arthur Fungarium.

Harold H. Flor (1955 AD)

Harold H. Flor was born on May 27, 1900 AD in St. Paul, MN. Dr. Flor's research on flax rust showed resistance in flax was dominant to susceptibility and the genes conditioning reaction occurred as multiple alleles at five loci. He then selfed and crossed many races of *Melampsora lini*, the flax rust fungus, and found virulence to be recessive to avirulence and inherited independently. His research suggested that for each gene conditioning resistance in the host, there was a corresponding

gene conditioning pathogenicity in the parasite. Furthermore, detection of these genes in the host or parasite was possible only when the other member of the pathosystem was present. Dr. Flor was the first to study simultaneously the genetics of the host and parasite, which allowed him to deduce what is popularly known as the gene-for-gene hypothesis.

Dr. Flor's interpretation of host-parasite genetic interaction has proven to be a critically important paradigm in plant pathology and of extraordinary utility in the breeding of disease resistant cultivars. It has been used extensively to explain genetic relationships in different rusts and in other diseases, as well as in diverse symbiotic relationships such as plants and herbivorous insects.

Flor's hypothesis was validated when a virulence gene of the parasite and resistant gene of the host were cloned and characterised in 1980 AD and 1990 AD decades. This was the culmination of a century of search for the resistant gene postulated by **Biffen** in 1905 AD.

Dr. Flor received many awards for his research contributions: the USDA Superior Service Award in 1957 AD, Outstanding Achievement Award from the University of Minnesota in 1962 AD, and Doctor of Science from North Dakota State University in 1963 AD. From the American Phytopathological Society, he received the Ruth Allen Award in 1966 AD, the Stakman Award in 1967 AD, and the Award of Distinction in 1980 AD. He was named Fellow in 1965 AD and elected President of APS in 1968 AD.

Richard L. Kiesling

The Plant Pathology Department at North Dakota State University was formally established on July 1, 1960 AD and Dr. Richard L. Kiesling as professor and chair was instrumental in making the department what it is today.

Dr. Kiesling was born on November 20, 1922 AD in Illinois. Michigan, Kiesling and John Grafius were responsible for the development of two new varieties of oats, Coachman and AuSable. The varieties were made available for the 1965 AD growing season. Throughout his career at NDSU, Kiesling was involved in many research projects:

- Epidemiology of barley covered smut
- Genetics of barley X barley covered smut interactions
- Black point of durum – epidemiology and resistance
- Root rot of wheat and barley – epidemiology and resistance
- Pinto bean disease – bacterial blight assay

- Potato virus disease indexing
- Establishment of pine in heavy alkaline soils in Cass County
- Root rot of wheat and barley.

William Farlow

In America, a fungus *Plasmopara viticola* was reported that grew on the native grapes. This fungus attacked the leaves of all species of grapes. However, it seemed to do little harm to the American grapes. In fact, it was even looked upon as beneficial by American growers. According to William Farlow, who was the mycologist at Harvard: Our native vines have a luxurious growth of leaves, and the danger is that in our short summers the grapes will not be sufficiently exposed to the sun to ripen. The *Plasmopara* appears at just the right moment to shrivel up the leaves so that the direct rays of the sun may reach the grapes. However, Farlow also added that if this fungus were to be introduced into Europe it would be a quite different story. In the moisture climate, the attack on the grapevines might prove as disastrous as the *Phylloxera*, and the fungus got inadvertently introduced into Europe.

Harry Marshall Ward

An outstanding contribution on the physiology of parasitism was his work on A Lily –Disease (1888 AD) incited by a *Botrytis* species. He made a thorough study of the fungus and of its penetration and invasion on host tissues. He obtained from pure cultures of the fungus a highly purified ferment that would break down healthy host tissues. Although he could not find a perfect stage of his fungus, he surmised that it was a *Peziza (Sclerotinia)*. Ward's belief was strengthened by his famous brume rust investigations (1902 AD, 1903 AD). Although later work did not support his interpretation of bridging hosts in his rust work, nor his conclusion that secretions produced by *Botrytis* dissolve the plant cuticle and thus permit penetration by the fungus, these mistakes in interpretation of pioneering experiments on very difficult problems were rare exceptions in his brilliant career.

Ward also warned about the dangers of monoculture. He observed that the continuous plantings of coffee trees over the island had created a perfect environment for a fungus epidemic. Rusts, like downy mildews, are obligate parasites and require living host tissue for their growth and reproduction. The rapid epidemic of the coffee rust was enhanced by many acres of the host plant. His warnings, unfortunately, were ignored, and most of the dead coffee trees were replaced with tea bushes. Luckily, no fungus immediately invaded the tea crop, and newly discovered fungicides were soon available to protect the tea from its fungal parasites. In an attempt to escape the rust disease, coffee production moved to the western hemisphere. Coffee had been grown in the Caribbean Islands since 1700 AD, but plantings quickly spread to the tropical highlands of Brazil, Colombia, and Central America.

The immediate death of cells surrounding the infection zone, noticed by **Ward** in resistant plants, which was later, termed hypersensitive response by **Stakman** (1912 AD).

Ward (1902 AD) was aware that anatomical features and special structures of plants were not of much consequence.

Erwin Frank Smith (1887 AD):

Erwin Frink Smith's work on bacterial diseases of plants began with successive studies on wilt of cucurbits (1895 AD), brown rot of solanaceous plants (1896 AD), and black rot of cruciferous plants (1897 AD). Outstanding among his later works are his Bacteria in Relation to Plant Diseases published in 1911 AD and his extensive researches on crown gall (e.g., Smith et al., 1911 AD1912 AD). In his controversy with Fischer, he (1899 AD a,b, 1901 AD) silenced the last doubters of the occurrence or the importance of bacterial diseases of plants. He is known as "Father of Phytobacteriology" in America.

Lime-sulphur has long been known and used as an insecticide and to a very limited extent as a fungicide in cases where disinfection of dormant trees is desirable, as in the case of the peach leaf curl. **Cordley of Oregon** in 1906 AD discovered that much diluted solutions of lime-sulphur might be used with safety and efficiency as a summer spray for apple scab.

Scott in 1907 AD devised and tested the so-called self boiled lime-sulphur mixture. He showed that it could be used successfully in the control of the scab and brown rot of peaches at the same time, without causing injury to the tender foliage of the peach. Such injury results as seen from the use of copper sprays or the solutions of calcium sulfides.

L.R. Jones (2010 AD) at University of Wisconsin started experiments on soil temperature in relation to plant disease development. Wiscosin soil temperature tanks were devised in which plants were grown in infested soil at a series of constant temperature for flax wilt and tomato wilt disease. He also contributed on etiology and control of potato blight.

Congressman Simmons of New York introduced a bill at its second session of the 62nds Congress to regulate the importation of planting material by the national government for nursery stock. This finally became a law in the form of the **National Quarantine Act of 1912** AD. This was the first national enactment aimed at the exclusion, from the country, of insect pests

and plant diseases. Other countries for years had various laws of this type, for the most part ineffective. The enactment of this measure, together with the establishment of a Federal Horticultural Board, marked a new period in the plant pathology not only for USA but for other countries as well.

J.C. Walker (1931 AD)

He worked on biochemical basis for disease resistance. The most convincing evidence was provided by **Walker** (1921 AD, 1929 AD) when he found that the resistance of coloured onion bulbs to smudge fungus, *Colletotrichum circinans*, was due to phenols – catechol and protocatechuic acid, present in the dry scales. These were absent in the susceptible white bulbs. Since then, the phenols have been found to play most important role in resistance, both in protection and defence.

Tanaka (1933 AD)

Isolated the first toxin to be recognised from *Alternaria kikuchiana* to cause leaf spot of pear.

Muller and Borger (1940 AD)

They studied the hypersensitive phenomenon in potato and coined the term Phytoalexin, the theory in 1941 AD. That's how phytoalexin serve as the basis of a disease resistance.

Meeham and Murphy (1947 AD)

Produced victorin (non-selective toxin) from *H. victoriae*. Twenty host selective toxin was recognised mainly from genera *Alternaria* and *Cochliobolus*.

Waggoner (1953 AD)

He coined the terms phytotoxin and pathotoxin.

Fraenkel-Konrat

Fraenkel-Konrat in USA simultaneously proved that RNA (like DNA) was a genetic material. Konrat (1955 AD) through his reconstitution experiments with TMV, proved that only RNA (and not protein) was infectious and possessed the viral properties.

Kassanis (1962 AD)

Kassanis in 1962 AD described a very small virus, still the smallest known in fact – found in association with some **isolates** of the larger tobacco necrosis virus (TNV). He found that it was dependent on TNV for replication. The complete nucleotide sequence of this small genome has subsequently been established. It contained only one gene which code for the coat protein. There is no genetic relatedness between the viral satellite, helper virus (TNV here) and its plant host. But the trilateral reaction is specific. These smaller particles

came to be known as satellite virus of TNV (SV–TNV). The satellite viruses enhance or retard the disease symptom caused by the helper virus and are put to use (with caution) in biological control of plant diseases.

Cook (1983 AD)

The rice false smut fungus was first described by Cooke in 1937 AD as a smut fungus and named *Ustilago virens*, based on the specimen from India.

There has been much debate on the scope and terms and limits of biological control (Baker, 1983 AD). Finally a definition given by **Cook** (1987 AD) has been widely accepted. Cook's definition in short is "*the use of natural or modified organisms, genes or gene-products to reduce the effects of pests and diseases*". Thus, genetically-engineered plants, resistant to pests fall in the category of biological control.

George A. Zentmyer (1913 AD–2003 AD)

He studied the biology and control of white pine blister rust and dutch elm disease. He became world known authority on *phytophthora*. He established new concepts chemotaxis, chemotropism and heterothallism. He also implemented biological control methods and determined the basis for their effect.

Myron K.Brakke (1921 AD–2007 AD)

A pioneer in virology and biophysical properties of viruses. He developed density gradient centrifugation, a tool for the purification of virus particles. He worked with the diverse array of virus – barley stripe mosaic, potato yellow dwarf, soilborne wheat mosaic, tomato spotted wilt and wound tumor virus.

6.3 In South America

6.3.1 Genome sequencing in fungi

The noteworthy contributions in different areas of pathology during present era are of:

James E. Galagan and co-workers (2003 AD)

The first complete genome sequencing of fungus *Neurospora crassa*. He worked on sequencing of *Aspergillus nidulans* and comparative analysis with *A. fumigatus* and *A. oryzae*.

Dean R.A. and co-workers (2005 AD)

They made the first complete genome sequencing of a plant pathogenic fungus *Magnaporthe grisea*. He worked on the role of transposable element clusters in genome evolution and loss of synteny in the rice blast fungus *Magnaporthe oryzae*.

The relative rates of evolution between proteins encoded by genes within TE clusters and outside of clusters were compared by computing the evolutionary distance to orthologous proteins in the *N. crassa* and *F. graminearum* genomes. This analysis assumes that all of the orthologous protein pairs diverged at approximately the same time while the divergence time may not necessarily be the same for all orthologous protein pairs, the mean time of divergence within and outside of the TE clusters should be similar.

David E. L. and Cooke (2012 AD)

They made genome analyses of an aggressive and invasive lineage of the Irish Potato Famine Pathogen. Polymorphisms, in combination with an extended biotrophic phase, may explain the aggressiveness of 13_A2 and its ability to cause disease on previously resistant potato cultivars.

André Mourão et al. (2013 AD)

He screened *cry*, *vip* and *cyt* genes in *Bacillus thuringiensis* strains collected from the Amazon biome in Brazil.

6.4 Devlopment of pathology in Australia

The noteworthy contribution in different fields of pathology during present era are:

6.4.1 On wheat pathology

W.L. Waterhouse

He continued the work on stem rust of wheat and published 20 papers on it. He showed the marked effect of environment on rust infection type and the significance of the barberry in the origin of new strains of wheat stem rust.

This followed the work of **Craigie** in demonstrating the function of the pycnia. The group at St. Paul, Minnesota used a culture homozygous for pathogenicity and of course recovered the same pathogenic strain from aecia as they had used in the initial inoculation.

Waterhouse used a strain heterozygous for virulence and recovered some very spectacular variants. About 1930 AD, Waterhouse began to see more clearly the role of resistant varieties in the control of cereal rust. He had, of course synthesised wheat lines earlier by crossing cultivars which combined the genes of resistance to the two groups of strains he had recorded. With the arrival of new rust from western Australia, combining new genes for virulence with new genes for aggressiveness, all his material became susceptible overnight. The arrival of rust resistance parents from overseas such as Webster, Hope, Gaza (*Triticum durum*) was a great stimulus for the work he proposed

on breeding. With rust in northern New South Wales and Queensland taking 25–30% of the crop. W.L. Waterhouse saw the need to intensify this work in the north-western part of New South Wales, where rust was a serious problem and began testing there. He was, in common with his associates elsewhere, not aware that breeding disease resistant plants would represent the new challenge it would turn out to be. It was not anticipated, for example, that releasing commercial wheat with single genes for resistance to rust would result in a steady succession of disappointments for the breeder. However, during the period 1940 AD–1950 AD, in spite of the war, investigations were made into the value and feasibility of having not one gene but several genes in the material released and this approach had been the basis for the success of the newest releases which combined several types of resistance. It is a great tribute of Waterhouse that during the latter period of his working career, we saw in 1947 AD–1948AD the last serious rust epidemic in New South Wales. Due to his basic approach to this problem, it has not arisen again in New South Wales for >20 years.

Upon the retirement of Waterhouse at the end of 1952 AD the rust work at the University of Sydney was continued by I.A. Watson, Professor of Agricultural Botany. A further outstanding advance was made in 1966 AD at the University when Williams et al. reported convincing evidence of the growth of the obligate parasite, *Puccinia graminis f.sp. tritici*, in culture media. The continuous attack on stem rust of wheat over an 80-year period has been remarkable contribution to plant disease control and to the welfare of Australia.

R.J. Noble carried out fundamental studies on the parasitism of flag smut of wheat. In 1924 AD Carne, who was situated in western Australia, obtained evidence that Nabawa wheat was resistant to flag smut, and within7 years it was sown on 32% of the five million acres of wheat. Breeding for resistance to flag smut was an unqualified success, and the resistance of Nabawa has been maintained for over 40 years. Gurka wheat was found in 1934 AD to be resistant to flag smut in Victoria, and a range of derivative varieties resistant to flag smut have since been bred in Victoria and in western Australia.

Farrer released, early in this century, varieties of wheat (Florence and Genoa) resistant to bunt. Because a cheap and effective fungicidal seed treatment was available for bunt control, most wheat breeders in Australia directed their attention to those diseases which were not controlled by fungicides.

A.T. Pugsley joined the Waite Agricultural Research Institute, and carried out the most comprehensive and successful breeding work against bunt in Australia. He has been Director of the Agricultural Research Institute at Wagga since 1953 AD. He identified races of *Tilletia*, studied the genetics

of resistance, and bred the bunt-resistant wheats Heron, Robin, Raven, and Yande. Disease-resistant or tolerant varieties have also been developed in sugarcane, potatoes, vegetables, and pasture plants.

J. Kuiper found a new race of *Tilletia foetida* in Victoria in 1965 AD which was tolerant of the organic fungicide, hexachlorobenzene. This is perhaps the first example of resistance of a pathogen to a fungicide developed under field conditions. The development of wheat variety resistant to the smut may overcome this difficulty.

6.4.2 On fruit disease

Fungicidal protection of fruits, vegetables, and other plants against infection by pathogens was utilised. Although there was no non-injurious fungicidal cover-spray, known at that time which could be used on apricot fruits without causing injury, apricots infected with scab (*Clasterosporium carpophilum*) were reduced from 70% to 2% with Bordeaux mixture in the autumn and at the pink-bud stage in August. It was later found that tetramethylthiuram disulfide could be used on apricot fruits without causing injury, and **Wade** also assessed it for brown rot control in Tasmania. It was shown in Victoria that dry-mix lime sulphur could prevent an epiphytotic of peach rust on the very susceptible variety, Thicle's Cling, and that it could reduce brown rot loss by about two thirds, but not anymore.

The epiphytotics of brown rot (*Sclerotinia fructicola*), was in evidence since the twenties, culminated in 1955 AD–1956 AD with heavy losses to growers of canning peaches. A combined work on this problem was made in 1957 AD–1962 AD by the Departments of Agriculture of Victoria, New South Wales, and Tasmania, and by the CSIRO and the University of Tasmania. **Wade** noted the possible occurrence of latent infection of brown rot in apricots, and **Jenkins and Reinganum** showed that it occurred in peaches as well as in apricot fruits. Dichlone applied to peaches at the early-full bloom and early-petal-fall stages reduced the occurrence of brown rot at harvest and in storage by eradicant action.

Simmonds of Queensland noted in 1941 AD, the existence of latent infection of tropical fruits, as did Adam in 1949 AD. **T.B. Kiely** of New South Wales found the perfect stage of the causal fungus of the black-spot disease of citrus, and showed that *Guignardia citricarpa* had an unrecognised latent period.

Hutton discovered that the overwintering ascigerous stage of apple and pear scab could be eradicated by post-harvest spraying of trees with mercurial fungicide. Because of mercury residues, use of this fungicide has not been encouraged, but it has opened up new avenues of research.

Fisher discovered the ascigerous state of the apricot freckle fungus.The most important single contribution to the Australian tobacco industry was the discovery by **Angel** et al. that fumigation with benzol vapor controlled blue mould in tobacco seedlings. Seedlings were grown at this time as far as 300 miles from tobacco areas in an effort to protect them from disease. Such precautions were rarely effective, and in the *Report from the Select Committee on the Tobacco-growing industry in Australia* (1929 AD–1930 AD) it was stated that, the growers have hitherto proved unable to deal with the blue mold disease, which in many cases wipes out the tobacco of a whole district two out of three seasons.

Adrian Loschiavo, Mark Sosnowski and Trevor Wicks (2013 AD) reported that the Eutypa die-back caused by the fungus *Eutypa lata*, is a major trunk disease of grapevines. Surveys have shown that eutypa die-back is widespread in premium wine growing regions of Australia. Their attempts to isolate the fungus from affected foliage have been unsuccessful because expression of foliar symptoms may occur 3–8 years after infection.

6.4.3 On root disease

Liliana R. Fraser of New South Wales identified the cause of a widespread citrus disease as *phytophthora* root rot, and introduced the use of the resistant *Poncirus trifoliate* as a root stock to combat this disease.

R.N. Hilton of the University of Western Australia was investigating die-back; in jarrah (*Eucalyptus marginata*) due to *Phytophthora cinnamomi* which has destroyed 4,62,000 jarrah trees and was threatening another 15 million trees. **G.C. Marks and F. Kassaby**, of the Forests Commission of Voictoria, investigated this fungus in nursery trees and shelter belts, along with **H.T. Hartigan** of the Forests Commission of New South Wales.

Root rots of wheat were first observed in south Australia in 1868 AD. Similarly **McAlpine** in 1902 AD recognised *Ophiobolus graminis* as a cause of take-all and dead heads in wheat. From the twenties to the end of the Second World War there was much activity in Australia on this problem by **Brittlebank** 1919 AD, **Samuel** 1923 AD–1933 AD, **Fish** 1927 AD, **Garrett** 1923 AD, 1935 AD–1938 AD and **Millikan** in 1940 AD, were concerned with *Helminthosprium* and *Curvularia*. **Samuel** and **Garrett** in 1932 AD, and **Hynes** in 1937 AD, investigated *Rhizoctocnia solani* on cereals, while **Geach** in 1932 AD–1933 AD was concerned with *Fusarium culmorum*. **F.C. Butler** published a comprehensive world review on the root-and foot-rot diseases of wheat, which contains some 70 Australian references.

Flenjte investigated *Rhizoctonia solani* after 1956 AD. **Magee** in 1957 AD recorded *Fusarium graminearum* as a crown-and root-rot pathogen of wheat,

and it was later studied, along with *Ophiobolus graminis* by **Chambers** and **Prince**. **Prince** also recorded *Pythium ultimum* associated with dead-heads of wheat in 1964 AD. *Ophiobolus graminis* and barley yellow dwarf virus were the most important pathogens in Victoria, causing losses of 40–60% in a few instances.

6.4.4 On virus diseases

During the twenties little was known about virus diseases in Australia. **Noble** studied the woodiness disease of passion fruit in 1928 AD. In the pioneering work of **Magee** in 1927 AD on bunchy top of bananas it was shown that it was an insect-borne virus disease. A scheme based on eradication and replanting with disease-free stock was initiated, and this industry was reestablished.

Samuel and **Pittman** carried out a joint investigation on spotted wilt of tomatoes, and Pittman showed that it was caused by a virus acquired only during the larval stage of *Thrips tabaci*, the first record of this type of vector. A second thrips vector, *Franklinella insularis*, was also found soon. **Dowson** in 1928 AD tried to increase potato yields by eliminating virus diseases.

Studies by **Magee** and **Adam** on potato virus diseases and their control eventually led to the initiation of potato seed certification schemes in the various states.

Pugsley and **Bald** studied the main virus diseases of the potato in Victoria. Bald contributed 24 papers on virus diseases of potatoes, symptom expression, reaction of variety s, host range s, effect on yield, and search for resistance during 1940 AD–1950 AD. Tobacco mosaic virus (12 papers) and tomato spotted wilt virus (7 papers) were also studied.

C.G. Hughes and **D.R.L. Steindl** joined **A.F. Bell** in the Bureau of Sugar Experiment Stations, Queensland, in the mid-30s. According to Hughes, the most important contribution of the Pathology Division was the discovery of ratoon-stunting disease and working out a method for its commercial control. Ratoon stunting has no external symptoms and there are internal symptoms. The effects of this disease vary with the variety and the season and on certain sensitive varieties there may be a total loss of crop. The discovery of this very infectious disease, which was subsequently found in practically every cane-growing country, offered one explanation of the sugar cane seed industry running out of cane varieties. This disease was controlled by heat treatment of setts material, in water at 50ºC for 3 h or in air at 54ºC for 8 h. The hot water is the more setts widely accepted method. In Queensland some 2,000–3,000 tons of plants are treated annually, but a much larger tonnage of disease-free plants come from special plots propagated from treated setts.

According to **P.B. Hutchinson**, Fiji disease of sugarcane has assumed some importance in New South Wales.

Grieve, while being with the University of Melbourne, showed that rose wilt and die-back are caused by a virus.

Rose Mushin conducted serological studies on plant viruses. **Stubbs and Grieve** first recorded carrot motley dwarf in 1944 AD and Stubbs studied its identification, epidemiology, virus vector relationships, and control in 1948 AD. Stubbs gradually expanded this work to include viruses of strawberry, citrus, grape, pasture, and cereals, while **R.D. Anderson** extended the work on virus diseases of potatoes.

Stubbs and O'Loughlin demonstrated climatic prevention of mosaic spread in lettuce seed crops in the Swan Hill region of the Murray Valley. Cooperative work with R.G. Grogan of the University of California resulted in the elucidation of the previously unrecognised lettuce necrotic yellow virus. Detailed studies of this virus, including its purification and characterisation by Chambers, Crowley, Francki and O'Loughlin have continued. Work on the strawberry virus complex by Stubbs, and subsequent provision of disease-free planting stocks of the Victorian Department of Agriculture led to a spectacular increase of 5–10-folds in yield. He has also been concerned with the identification of citrus viruses, and their elimination by heat theraphy. His work has involved inoculation with mild strains of the tristeza virus with a view to its protection against severe strains.

Simmonds of Queensland was one of the first researcher to use a mild strain against a plant virus, the passion fruit woodiness virus. Elimination of the viruses affecting fruit trees by heat therapy has been studied by **Stubbs** in Victoria, **R.S. Greber** in Queensland, and **G.P. Johnstone** in Tasmania.

There is probably more work done on virus diseases of fruit species in Australia than on other crops. **Johnstone and Martin** have shown that infection of apple with the green crinkle virus markedly reduces the produce compared with virus-free trees.

Fraser demonstrated the virus nature of scaly butt of *Poncirus trifoliata* which limited the use of root stock, and the previously unrecorded diseases of citrus, seedling yellows, and woody gall. **Pares** studied virus diseases of stone fruits, and **Hutton** investigated virus diseases of pome fruits. In 1957 AD, **Smith** reported the presence of barley yellow dwarf virus of cereals in Australia.

R.H. Taylor worked with **W.B. Hewitt** of the University of California on grape viruses in 1962 AD–1963 AD and continued work on serology, virus purification, and electron microscopy.

6.4.5 On bacterial diseases

Work on bacterial diseases of plants in Australia commenced with that of **Cobb** on the gumming disease of sugarcane in 1895 AD. **North** in 1926 AD described and named leaf scald disease of sugarcane caused by *Xanthomonas albilineans*. In 1935 AD, he published a paper on gumming disease of sugarcane by *X. vasculorum*, and on its dissemination and control, mainly by using resistant varieties or healthy planting material.

H.L. Jensen, who was appointed Maclaey biologist with the Linnean Society of New South Wales in 1929 AD, found that the *Corynebacteria* were numerically important group of soil bacteria and were active in the decomposition of organic matter in the soil. In a study of saprophytic mycobacteria and corynebacteria, he placed the organisms causing bacterial canker of tomato and bacterial wilt of Lucerne in the genus Corynebacterium. He also described strain of both *C. michiganense* and *C. insidiosum* isolated from soil under grass.

Cottrell-Dormer in 1932 AD described the causal organism of red-stripe disease of sugarcane, *Pseudomonas rubrilineans*.

An epiphytotic of halo blight of beans, caused by *Pseudomonas phaseloicola*, devastated the French bean seed-growing industry in Victoria in 1930 AD–1931 AD, severely damaging crops in New South Wales and other states when infected seed was used. None of the seed treatments listed by Adam were effective, and Pugsley demonstrated that the pathogen was under the third seed coat and in the cotyledon, which made effective seed treatment very difficult. The principle variety which was grown at that time was Canadian Wonder on which the bean-seed export had been built. This variety proved to be so susceptible to halo blight that it was no longer grown commercially. A strain of Canadian Wonder which was field-selected from a badly infected crop and had good field resistance was, unfortunately, inferior in its length of pod and other desirable horticultural characters. Varieties with field resistance, such as Richmond Wonder, Hawkesbury Wonder, and Windsor Long Pod, have been developed in New South Wales. Halo blight has also been kept under control by seed certification with a zero tolerance being enforced.

Studies by **Adam and Pugsley** showed colony variation, and phage reactions of the pathogen. They also identified other bacterial diseases. In 1936 AD Pugsley first used serological techniques for identification of plant bacterial pathogens in Australia.

Wilson's demonstration of improved means for detecting infected bean seed was the basis of the New South Wales bean-seed certification scheme.

Griev studied the relationship of *Ps. solanacearum* to water plants in 1941 AD. **Rose Mushin** also published on the physiology of plant pathogenic bacteria in the same year.

Harrison reported a typical strain of *Ps. solanacearum* producing symptoms somewhat similar to those recorded for the ring-rot bacterium, *Corynebacterium sepedonicum*. **Harrison and Freeman** found this strain to possess a common antigenic fraction with type 11 described by **A.C. Hayward** of University of Queensland.

Erwinia atroseptica has caused severe losses in Sebago and to a lesser extent in Sequoia potato varieties. According to Harrison, seed-piece decay and black leg have accounted for a loss of up to 50% of some potato crops, particularly in low, poorly drained areas.

Two important bacterial diseases have recently been identified in Australia. Lucerne bacterial wilt, caused by *Corynebacterium insidiosum,* was found in Victoria, and a survey showed it to cause early decline in Lucerne stands. The Hunter river variety was very susceptible to bacterial wilt. Serology tests at the University of California showed the similarity of Victorian and United States isolates. Resistant American Lucerne varieties have been introduced. Peach bacterial spot, caused by *Xanthomonas prino*, was first recorded in New South Wales on plum in 1929 AD and on peaches in 1960 AD. Infected peaches are unmarketable, severe defoliation may occur, and severe cankers form on the lateral branches. Other bacterial pathogens in Australia of economic importance include *Pseudomonas syringae, Corynebacterium michiganense, Xanthomonas juglandis, X. campestris, and X. phaseoli*.

Alien Kerr and Karen Gibb (1996 AD) reported the crown gall of stone fruit caused by the soil-inhabiting bacterium *Agrobacterium rhizogenes* and less frequently, *A. tumefaciens* in Australia and the pathogens are really natural genetic engineers because of the following wounding plant cells that are getting transformed. A small piece of DNA is transferred from a bacterial cell to a plant cell where it encodes the synthesis of plant growth hormones and opines. The growth hormones are auxins and cytokinins which stimulate the plant cells to grow and divide to produce the characteristic galls. Opines form a group of compounds found nowhere else in the plant kingdom. They consist of two common plant substances joined together in an unusual way.

They also reported that pathogenicity in *Agrobacterium* is dependent on the presence of a large plasmid, the tumour-inducing plasmid (pTi). Several kinds of pTi have been described and are usually defined by their opine characteristics. So there are octopine plasmids, nopaline plasmids, etc. Only part of a pTi, the transfer DNA (T-DNA) is transferred to a plant cell where the opines are synthesised. More than one opines and the corresponding catabolic genes are always present on pTi but not on the T-DNA. Most non-pathogens and

soil bacteria cannot utilise opines. A further sophistication of Agrobacterium is that some opines also induce conjugation and pTi transfer between bacteria. Not only do pathogens have a considerable ecological advantage over non-pathogens lacking pTi, but the latter are converted to pathogens.

They (1996 AD) further reported leaf scald of sugarcane (*Xanthomonas albilineans*) as one of the most devastating diseases of sugarcane in more than 50 countries and occurs in most sugarcane-growing districts of Australia. It was first recorded in Australia in 1926 AD and was probably introduced on clonal planting material at a time when quarantine restrictions were inadequate. They developed a technique for the elimination of *X. albilineans* from planting material. It consists of soaking setts in water at ambient temperature for at least 24 h, followed by hot-water treatment for 3 h at 50 ºC. They worked on a second approach as to grow resistant cultivars but unfortunately, they were not as productive as some susceptible cultivars. A new approach was being taken by them at the University of Queensland. It involved the genetic engineering of sugarcane. The strategy was to find genes that will inactivate the bacterial toxin and introduce them into high yielding leaf scald susceptible cultivars. If the gene is active in the plant, it should destroy the toxin and make the plant resistant to the disease. They found genes conferring resistances were cloned and their mode of action was investigated. Those that inactivated the toxins were engineered to ensure that they would be expressed in sugarcane and then transferred to 480 sugarcanes by means of a DNAgun or micro-projectile. Recent data indicate that susceptible cultivars have been made resistant by this method. It is a method that could have wider application in the control of bacterial diseases of plants.

Fahy and Persley (1983 AD) reported crown gall of grapevine was caused by another species of Agrobactertum, A. vitis, but the process of crown gall induction is the same as that described in other crops and species. However, A. vitis differs from A. rhizogenes and A. *tumefaciens* in several characteristics, including the fact that it lives systemically in the vascular tissue of its host. Fahy and Persley, (1983 AD) further reported the main causes of bacterial soft-rot of potato and other vegetables as *Erwinia carotovora* subsp. *carotovora, E. carotovora* subsp. *atroseptica* and *E. chrysanthem*. These bacteria are widely distributed and are the most common cause of soft-rot in Australia. They also worked on bacterial wilt of potato and other crops caused by *Ralstonia solanacearum* (previously named *Pseudomonas solanacearum*). The species consist of a large number of divergent strains which have been divided into biovars, based on biochemical reactions. The strains of *R. solanacerum*, biovar 3 strains have a very wide host range e.g. tobacco, tomato and ginger and are the most prevalent and destructive strains in tropical and subtropical Australia.

Fahy and Persley (1983 AD) also reported warm moist condition was ideal for spread and development of bacterial blight of cotton, caused by

Xanthomonas campestrts pv. *malvacearum*. The disease was first recorded in Australia in 1923 AD. In the period 1980 AD onwards, regular surveys of cotton crops showed that Race 18 of the pathogen, which is highly virulent on the popular Deltapine cultivars, was predominant in Australia and also that seed infection was a major factor in the epidemiology of the disease.

Hayward and Hartman (1994 AD) reported bacterial blight of peas caused by *Pseudomonas syringae* pv. *pisi* and *P. syringae* pv. *Syringae*, which was seed-borne and caused considerable, if sporadic, economic damage especially during spring and summer. They recently estimated the loss in Australia as $19m in one year. They distinguished two pathogens serologically or by inoculating stems of the susceptible pea cultivars, Rovar and Blue Prussian.

Buddenhagen (2006 AD) wrote the diagnostics manual for the bacterial wilt diseases of banana. The disease is systemic and so once infected, a plant must be destroyed in order to prevent further spread of the disease. He discussed various methods for the infield disinfection and tools have been devised (Buddenhagen and Sequeira, 1958 AD; Sequeira, 1958 AD; Stover 1972 AD; Wardlaw, 1972 AD). *Musa* spp., both bananas and plantains, and *Heliconia* spp. are susceptible to natural infections of the banana strains of *R. solanacearum* Race 2 (Sequeira and Averre, 1961 AD; Buddenhagen, 1994 AD). The comparison of the phenotypically based race and biovar more accurately reflects diversity in the *R. solanacearum* species complex. Race 1, defined as strains affecting tobacco, tomato, many solanaceous and other weeds, and certain diploid bananas (Buddenhagen, Sequeira et al. 1962 AD).

They stated that *Ralstonia solanacearum* Race 2 was first introduced in Australia in 1989 AD on infected rhizomes of *Heliconia* imported from Hawaii. Prior to December 1987 AD, *Heliconia* plants required a 9-month post-entry quarantine period, which included the screening of the imported material for *R. solanacearum* Race 2, however, after this date the post-entry quarantine period was reduced to only 3 months. The rhizomes, imported in April 1989 AD, from Oahu, Hawaii had been screened for the required 3-month quarantine period at a private post-entry quarantine facility in Cairns. In response to advice from the Austalian Quarantine Inspection Service (AQIS) that *R. solanacearum* Race 2 had been isolated from *Heliconia* plants in Hawaii.

6.4.6 On nematode diseases

After Cobb left Australia, plant pathologist without specialised training in nematology was largely dependent on information developed elsewhere for nematode control. **W. Laidlaw** worked as an early worker on onion eelworm.

J. Davidson first described the damage caused by *Heterodera avenae*, now regarded as of great importance in cereals in Victoria and south Australia.

Millikan's experiment on the control of nematodes led to the discovery of zinc deficiency in cereals.

The findings by **W. Carter** in 1943 AD in Hawaii that D-D was effective as a soil fumigant against nematodes stimulated studies on nematode control in Australia.

Seinhorst from Holland was appointed to work on nematode problems and to conduct a short school. **Seinhorst and Sauer** indicated the existence of a nematode complex on grapevines in Australia.

By 1955 AD, the Departments of Agriculture of New South Wales and Queensland had appointed plant pathologists in nematology and soon the work began in south and western Australia. A Victorian pathologist went to Rothamsted to study in the Nematology Department.

The CSIRO established a Section of Nematology in the Division of Horticultural Research in Adelaide. Specialised courses in nematology became available at the University of Sydney under **C.D. Blake** and at the University of Adelaide under **J.M. Fisher**. An extensive list of nematodes and their hosts recorded in Queensland showed the range of crops attacked by nematodes apparently introduced in Australia. Recent papers by **Meagher, Colbran**, and **Sauer** have described several new plant-parasitic genera. Most of the new Australian nematodes have not been shown to attack cultivated crops.

Much work is now in progress in nematology throughout Australia and an unpublished bibliography of Australian literature from 1890 AD–1945 AD shows 104 references and from 1945 AD–1969 AD, 232 references.

6.4.7 On plant quarantines

The outbreak of Phylloxera in Victoria in 1875 AD, and subsequently in New South Wales and Queensland, demonstrated the need for an effective Plant Quarantine Service. Legislative action in 1877 AD limited the spread of this insect. Interstate conferences were held in 1884 AD for the purpose of designing uniform legislation for a Quarantine Bill. This was based on part of the Public Health Statue of 1865 AD of Victoria, and the original basis can be traced in the form of the existing Quarantine Act of 1908 AD and 1969 AD.

The Federal Quarantine Service was established in 1909 AD in the Department of Trade and Customs, but in 1921 AD it was placed under the Ministry of Health with the Director General of Health as the Director of Quarantine. The Chief Officer administrates the Plant Diseases of Quarantine plants under the Federal Law.

The most important early action in relation to plant quarantine was instituted in 1916 AD when citrus canker *Xanthomonas citri* was found in the northern

territory. **Gerald F. Hill** supervised the destruction of infected trees, and the diseases were successfully eradicated.

E.A. Mackinnon was appointed Director of Plant Quarantine in 1927 AD. **T.H. Harrison** was appointed Assistant Director General of Health, Plant Quarantine, in 1947 AD, and plant quarantine policy in Australia was consolidated, with the 1941 AD report of the Australian Institute of Agricultural Science as a guide. J.R. Morschel succeeded T.H. Harrison.

The Australia is free from such major diseases and pests as citrus canker, fire blight of apples and pears; wart disease of potatoes, golden nematode, European corn borer, and Colorado potato beetle is a tribute to the plant quarantine service of this island continent.

6.4.8 On phytoplasmal diseases

Alien Ken and Karen Gibb (1996 AD) worked on phytoplasmas affecting wide range of crop plants including ornamental and weed plants causing diseases such as legume little leaf, witches' broom of lucerne, big bud of tomato, Australian grapevine yellows, green petal of strawberry, strawberry mycoplasma yellows, yellow crinkle of papaya (pawpaw), die-back of papaya, mosaic of papaya, bunchy top of peanut, little leaf of sweet potato, purple top wilt of potato, virescence (greening of flowers) of choko and sesame and phyllody (greening of petals) of foxglove. Some of these diseases are considered to be economically important and are described by **Padovan** et al. (1995 AD).

Big bud of tomato (TBB) was first described in 1933 AD as a virus disease and affected plants were reported as having a dense, tufted habit with an enlarged bladder-like calyx. It was reported to occur on tobacco, tomato and other species throughout Australia.

In Australia, papaya (*Carica papaga*) is affected by yellow crinkle, mosaic and die-back. Die-back is the most serious. It has been known since 1922 AD and frequently causes losses from l0% to 100% of trees in southern and central Queensland.

Australian grapevine yellows were first recorded in 1975 AD and a phytoplasma etiology was suspected after symptom regression following tetracycline treatment. Phytoplasma like bodies were also observed in electron microscopy of ultra thin sections of phloem from affected grapevines.

6.5 Development of plant pathology in Africa

The noteworthy contribution in different areas of pathology during present era are of:

6.5.1 On fungal diseases

Trends of investigation naturally developed regarding groups of causal agents of diseases. Since fungi were the first microorganisms shown to incite disease, the first great trend in plant pathology was mycological followed by virology and bacteriology, which have very less economic importance in agricultural crops in Africa.

Plant pathology in Africa began in the last century as a result of sporadic rusts, and mosaic in different crops. Epiphytotics caused heavy losses in Africa and devastated part of the vital supply of the pioneers in 19th and 20th centuries. There had not been enough development in plant pathology in 19th century due to undevelopment and poverty in Africa, but in 20th century Africa had more development in plant pathology.

Travadon, et al., (2012 AD), reported ascomycete fungus *Eutypa lata* causing Eutypa die-back of grapevine (*Vitis vinifera*). To decipher the cosmopolitan distribution of this fungus, the population genetic structure of 17 geographic samples were investigated from four continental regions (Australia, California, Europe and South Africa). Based on analysis of 293 isolates genotyped with nine microsatellite markers, high level of haplotypic richness (R=0·91–1) and absence of multilocus linkage disequilibrium among loci supported the preponderance of sexual reproduction in all regions examined. Nonetheless, they got confirmation of identical multilocus haplotypes with identical vegetative compatibility groups, in some vineyards in California and South Africa. They also suggested that asexual dispersal of the fungus among neighbouring plants could be a rare means of disease spread.

In this survey the greatest levels of allelic richness (A=4·89–4·97) and gene diversity (H=0·66–0·69) were found in Europe among geographic samples from coastal areas surrounding the Mediterranean Sea, whereas the lowest genetic diversity was found in South Africa and Australia (A=2·78–3·74; H=0·49–0·57). They examined samples from California, Australia and South Africa, which had lower genetic diversity than those of Europe. These findings suggested that human-mediated spread of the fungus may have resulted in its current global distribution.

Sosnowski (2009 AD) achieved control of eutypa die-back by protecting wounds from infection of the fungus, *Eutypa lata* or by physically removing infected wood. Wounds can be protected with fungicides, paints, pastes and biological control agents. It is also important to time pruning in order to avoid rainfall events and to maintain good sanitation. Controlling of established infections is achieved by removing infected wood and retraining of watershoots from below infection.

Nicolette Schinzl (2003 AD) worked on the fourth most important food crop in the world i.e. the humble banana and its relative where banana and plantain production in Africa is hampered by many constraints, having direct implications on issues of food security. This is compounded by the lack of resources and money to control diseases and pests by means of expensive chemical pesticides which are all too often ecologically unsound options anyway. In order to avoid pesticides he suggested the safe and affordable alternatives for controlling banana diseases and pests, a research in plant biotechnology in combination with plant molecular genetics.

He worked with The Banana Diseases Research Project (BDRP) as a part of the Forestry and Agricultural Biotechnology Institute (FABI) at the University of Pretoria in South Africa. FABI is a multi-disciplinary, postgraduate academic institution aimed at producing plant scientists sensitive to the agricultural needs of the African continent.

Another serious fungal disease in Africa is *Fusarium* wilt or Panama disease, which attacks the roots of the banana plant, affecting the vascular system required for mineral and water transport. It has evolved into three races that attack certain banana species only. In addition to this, once a crop of infected bananas has been harvested, the field from which it is taken cannot be used again for some time because of the fungal pathogen which remains active in the soil for years.

Akinsanmi et al., (2001 AD) reported the disease in Kenya, Rwanda, Uganda, and spread from southwards to Zambia, Zimbabwe in 1998 AD, and by, 2001 AD it got established in South Africa. In westward direction, the disease has been reported in Nigeria in 2001 AD. In this related study, a set of five differentials identified three races from 50 isolates collected at five locations in Taiwan and Africa by Yeh, 1983 AD–1985 AD. **Bromfield** (1984 AD) recommended a long term solution against soybean rust, as race specific resistances have been identified, but no immune cultivars were developed.

Kawuki et al., (2003 AD) collected information on distribution, epidemiology and management of soybean rust in Africa.

6.5.2 On plant virus diseases

In Africa, virus diseases of crops rank second to those caused by fungi in economic importance. Accordingly, high incidences and/or serious epidemics of virus diseases have become common place in most farming communities in Africa. These diseases include cassava mosaic, cocoa swollen shoot, rice yellow mottle, maize streak, banana streak, and banana bunchy top.

Otim-Napea et al., (2000 AD) highlighted the historical perspectives of plant virology in tropical Africa, with the emphasis on virus diseases

of important crops, their impacts, and the research and development interventions made to manage them. The lessons learnt from the occurrence of these diseases, the knowledge gaps, and the challenges to plant virologists in Africa are emphasised. They discuss the critical need for concerted efforts in research and development throughout Africa, so as to fully exploit the existing expertise, knowledge, facilities, and opportunities. This will eventually translate into improved agricultural production through the development and use of practical and viable technologies to manage crop virus diseases.

Recent epidemiological studies in different agroecological zones of Africa have revealed at least 30 relatively common viruses of plants, which produce significant economic losses in Africa. Cassava mosaic was probably the first virus disease reported in Africa (Warburg 1894 AD). Other early reports were of maize streak (1901 AD) and groundnut rosette diseases (1907 AD). These diseases have caused and continued to cause major economic losses in Africa. The rapid increase in production was severely constrained by the devastating cocoa swollen shoot disease which was first reported in Ghana in 1936 AD, although there is evidence that it had occurred earlier (Stephen, 1937AD; Dale, 1962 AD). It is believed that the disease was introduced to cocoa plantations by mealy bugs from wild indigenous trees of the West African tropical rain forest (Posnette 1947 AD, 1981 AD). The disease has seriously affected cocoa in Côte d'Ivoire, Ghana, Nigeria, Sierra Leone, and Togo (Owusu, 1988 AD).

In an attempt to control the disease, the authorities in the West African countries, especially Ghana, embarked on an ambitious eradication program (Jeger and Thresh, 1993 AD). By the end of 1970 AD onwards, over 160 million cocoa trees had died or have been eradicated as a consequence of swollen shoot disease and the number had increased to 190 million by the early 1990 AD. **Thurston** (1973 AD) estimated the losses, particularly in Ghana, have been devastating in his review on threatening plant diseases cited losses in cocoa production of over 20,000 tonnes in 1961 AD. **Owusu** (1988 AD) reported losses in yield of mature trees of 25, 50, and almost 100% in the first, second, and third years of field trials, respectively. Also, **Legg** (1979 AD) estimated that the similar losses have occurred in natural disease outbreaks. The decline in cocoa production from 118–110 tonnes to 38–90 tonnes in the eastern region of Ghana between 1936 AD and 1956 AD was largely attributed to swollen shoot disease noted by **Dale** (1962 AD).

Cassava mosaic virus disease (CMD) was first reported in Africa in what is now Tanzania by **Warburg** (1894 AD). The disease is now known to be caused by a number of similar cassava mosaic viruses of the genus *Begomovirus*; family: *Geminiviridae* (Bock and Woods, 1983 AD; Thresh et al., 1998 AD; Swanson and Harrison, 1994 AD), *viz.*; *African cassava mosaic virus* (ACMV) and *East African cassava mosaic virus* (EACMV). Rey and Thompson (1998 AD) recently distinguished *South African cassava mosaic*

virus (SACMV) and a variant (with properties of both ACMV and EACMV) referred to as the Uganda variant (UgV) by **Zhou** et al. (1997 AD), or as a strain of EACMV (EACMV–Ug) reffered by **Deng** et al. (1997 AD). On plausible assumptions, **Thresh** et al. (1997 AD) suggested an overall loss of 15–24% in Africa, equivalent to 12–23 million tonnes of cassava.

Sweet potato virus disease (SPVD) is the most serious disease of sweet potato in Africa reported by **Geddes** (1990 AD). He also stated it to be caused by dual infection with *Sweet potato feathery mottle virus* (SPFMV) and *Sweet potato chlorotic stunt virus* (SPCSV). Studies in Uganda, Nigeria, and Cameroon have shown devastating yield losses of up to 98%.

Groundnut rosette virus disease was first reported by Naidu et al. in southern Africa in 1907 AD and now seems to be widely distributed in east, west and southern Africa. He also reported 303 plant virus diseases in sub-Saharan Africa. The disease involves three pathogenic agents namely, *Groundnut rosette virus*, its assistor satellite RNA, and *Groundnut rosette assistor virus*, and seems to be restricted to Africa. **Misari** et al. 1988 AD; **Naidu** et al. (1998 AD) and **Oumarou** et al. (1990 AD) worked on occurrence i.e. sporadic and reported very devastating epidemics leading to high incidences and severities of the disease at various times in many African countries including Democratic Republic of Congo, Gambia, Malawi, Nigeria, Niger, Senegal, South Africa, Tanzania, and Uganda. In the review of groundnut rosette disease, **Naidu** et al. (1998 AD) cited examples of serious economic losses caused by the disease in northern Nigeria in 1975 AD, Zambia in 1995 AD, and a 23% drop in production in Malawi between 1994 AD and 1996 AD. Total losses in those years have been estimated at US$250 million and US$4.9 million for Nigeria and Zambia, respectively.

T.Alicaib (2000 AD) reported some of the factors that are responsible for the increasing importance of plant viruses in Africa. They are the rapidly changing agro ecosystems, an intensification of crop production, the vulnerability of the crop varieties grown, the latent nature of many plant viruses, the variability of many plant viruses, and the inadequate skilled manpower and facilities for virus detection, diagnosis, and control. Other factors are lack of improved technologies for virus disease control, laxity of bye-laws on plant virus disease control, and weak unsustained collaboration between African and advanced laboratories in areas of virus research and extension.

6.5.3 On genetics of host and pathogen in disease resistance

The examination of alleles from the *L* resistance locus and chimeric genes between alleles expressed in transgenic flax provided the first indication that the specificity of gene-for-gene interactions was determined by sequence variation of the LRR domain of the receptor proteins, and that the likely

origin of new resistance specificities in nature results from point mutations, re-assortment of the mutations, and duplication and deletion of LRR units by intragenic recombination. The flax rust system was among the first three systems to provide cloned resistance genes and the identification of a new class of immune receptor, cytoplasmic nucleotide-binding leucine-rich repeat (NB-LRR) proteins, specific members of which provided host plant resistance against fungi, oomycetes, bacteria, viruses, nematodes, sucking insects and parasitic plants (**Ellis** et al., 2007 AD).

Early stem rust epidemics initiated studies on pathogenic variability, epidemiology and host pathogen genetics in *Puccinia graminis tritici* (*Pgt*) was reviewed by **Loegering**, (1984 AD) and **Roelfs** (1985 AD). The specialisation of *Pgt* in different races has impacted strongly on wheat breeding and production. Numerous cultivars protected by single genes have become susceptible to stem rust, often with devastating boom-and-bust effects (Jin, 2011 AD; Kolmer et al., 2007 AD; Martens and Dyck, 1989 AD; Park, 2007 AD; **Pretorius** et al., 2007 AD). The occurrence of Race Ug99 of *Pgt* in East Africa with virulence for *Sr31* (Pretorius et al., 2000 AD) as a result of its adaptive capacity, fitness and virulence attributes (90% of the world's wheat is susceptible), the Ug99 race group has been recognised as a major threat to food security (**Flood**, 2010 AD; **McIntosh and Pretorius**, 2011 AD; **Singh** et al., 2011 AD; **Vurro** et al., 2010 AD).

Together with progress in the detection, genetic mapping and management of genes and quantitative trait loci (QTL) conferring resistance to Ug99 (Durable Rust Resistance in Wheat Project, http://www.globalrust.org), significant advances are being made in understanding the molecular basis of pathogenicity in *Pgt* (**Duplessis** et al., 2011 AD). Continued research on surveillance and race analysis, coupled with pathogen genomics, will enable the discovery, characterisation and utilisation of sustainable management strategies for resistance. The release and adoption of widely adapted resistant cultivars are essential for future and effective rust control globally (**Lowe** et al., 2011 AD; **McIntosh and Pretorius**, 2011 AD).

Despite many advances made since cassava moasic disease (CMD) was first reported over a century ago, CMD continues to be a significant threat to sustainable cassava production in sub-Saharan Africa. Although molecular studies on CMBs and host-virus vector interactions are advancing our knowledge on various aspects of CMD, one of the key gaps in knowledge on the epidemiology of CMD in sub-Saharan Africa is to define the role played by non-cassava plant species and alternative/reservoir hosts in the perpetuation of CMBs. Since cassava was introduced from South America in the 16th century, it is likely that CMBs endemic to Africa that infected indigenous African plant species became adapted to cassava. Thus, it is plausible that native plant species could act as alternative and/or reservoir hosts for CMBs and contribute

to continued virus evolution and future disease epidemics. Hence, concerted efforts are needed to enforce quarantine regulations in African countries to prevent dissemination of CMBs by propagation materials. A prerequisite to prevention is the availability of sensitive, reliable and rapid diagnostic tools for detection and differentiation of CMBs, including the recently developed multiplex PCR assay for detecting multiple CMBs in mixed-infected plants. Such assays will need to be constantly updated to detect newly evolving CMBs, as well as to incorporate detection of other important cassava viruses such as *Cassava brown streak virus* (genus *Ipomovirus*; family *Potyviridae*) in clean plant programs. The intensification of research efforts and the achievement of a synergy between traditional and novel approaches for CMD management will provide a holistic approach towards making cassava improvement an integral part of poverty alleviation in sub-Saharan Africa (**P. Lava Kumar**, 2011 AD).

Fen Beed (2013 AD) explained that banana *Fusarium* wilt is not new to the African continent. Foc Race 1 has first been reported from West Africa in 1924 AD. A second introduction occurred in 1951 AD when Foc Race 1 entered Tanzania. This strain is now widely spread in most banana producing countries in Africa. The majority of bananas grown on the continent, such as East African Highland Banana (EAHB), plantains and Cavendish bananas, however, are resistant/immune to Foc Races 1 and 2. For control of wilt he includes following measures: (a) containment of the outbreak in Mozambique to prevent spread on-farm, within the country and to neighbouring countries. (b) separation of other countries dependent on banana against future incursions by Foc TR4 through enhanced bio-security, frameworks, targeted awareness campaigns and training. (c) Management of banana Fusarium wilt in Africa by employment of resistant varieties and integrated disease management.

An African consortium (AC4TR4), was mobilised in November 2013 AD to deal with Foc TR4 (*Fusarium oxysporum* f. sp. *cubense* tropical Race 4) in Africa following its discovery in a plantation of export Cavendish bananas in northern Mozambique. AC4TR4 is a consortium of banana scientists of Stellenbosch University, IITA, Bioversity, Matanuska, NPPOs, NARs and other role players on the continent. Foc TR4 is one of many strains responsible for Fusarium wilt, a destructive disease commonly known as Panama disease.

6.5.4 On fungicides in plant disease control

Trevor Wicks (2002 AD) developed and evaluated management strategies for the efficient use of the new fungicide group, the strobulirins, for the control of powdery mildew, downy mildew and Botrytis bunch rot of grapes. Laboratory, greenhouse and field experiments were carried out to determine the protectant and curative limitation of the strobilurin fungicides Amistar and Flint. These

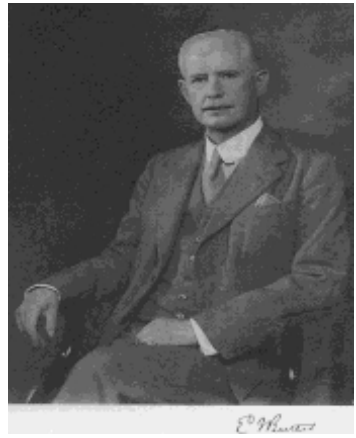
studies showed that they were most effective when applied before infection, and while they were effective against both powdery and downy mildew, they were less effective on Botrytis bunch rot. Field trials over three seasons of evaluating various sprays regimes were focused mainly on powdery mildew and these showed that excellent control of powdery mildew was obtained when four to six sprays per season were used and included either of wettable sulphur or DMI before and after two to three applications of a strobilurin around flowering. Where Flint and Amistar were directly compared Flint was the most effective fungicide for controlling powdery mildew whereas Amistar was more effective than Flint in controlling downy mildew. He also gave strategic use of sulphur in IPM Programs for Grapevines. Laboratory and field studies re-examined the efficacy of sulphur for powdery mildew and mite control, clarifying the effects of factors influencing the performance of sulphur to develop strategies that make optimum use of sulphur in IPM programs while minimising the potential for residues in grape products.

Herbert, J.A. and Marx, D. (1990 AD) conducted trials in South Africa, for methyl bromide which significantly reduced disease incidence of panama wilt, but was effective for only 3 years due to recolonisation of the fumigated areas by the pathogen. Although production was successfully reestablished at this site for 3 years after fumigation with a mixture of methyl bromide and chloropicrin, recolonisation of the site by the pathogen eventually negated this positive response.

6.6 In Asia: Development of plant pathology in India

6.6.1 Fungal pathology

E.J. Butler

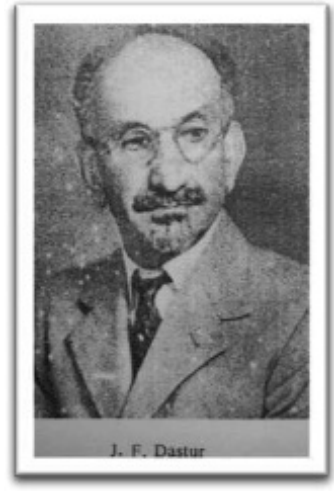


E.J. Butler stayed at the Emperial Agriculture Institute at Pusa, Bihar for 16 years (1905 AD–1921 AD) and established a strong school of mycology and plant pathology. His book, published in 1918 AD, on fungi and diseases of plants served as the major source of literature and inspiration to budding plant pathologists. Before Butler departed from India the book he published in 1918 AD remains a classic on the subject and about 200 diseases of Indian crops were included, which he studied in the field and in the laboratory. A disastrous earthquake

in 1934 AD badly damaged the Pusa Institute that it was decided to rebuild the Institute at New Delhi as the Indian Agriculture Research Institute which included a flourishing division of plant pathology.

Red rot (*Colletotrichum falcatunm*) causes enormous loss to the sugarcane crop in India. It was first studied by **Butler and Khan** at Pusa in 1913 AD. Infection was found to occur through setts, soil, and irrigation water. Simple practical schedules for its control were suggested by **Chona** (1947 AD), and the disease is now totally controlled by a hot-air treatment of the setts (Singh, 1968 AD).

Butler (1918 AD) first described the smut disease of sugarcane caused by *Ustilago sacchari*. The mode of infection of smut disease of sugar cane caused by *Ustilago sacchari* was studied by **Dastur** (1920 AD). Life history and method of perpetuation of the pathogen have been investigated by **Luthra and Sattar** (1938 AD). A monograph on potato diseases was written by Butler in 1903 AD. Other contributions include his classical work on genus *Pythium* and Chytridiaceous fungi.


J. F. Dastur

First Indian plant pathologist who was credited for his detailed studies on fungi and plant diseases is **Dastur J.F.** He studied the characters of *Phytophthora* and its diseases of potato and castor. Particularly, the pathogen *Phytophthora parasitica* from castor. **Dastur** (1915 AD) reported that potato blight due to *Phytophthora infestant, though* uncommon in the Indian plains is found on the hills, and caused severe damage to crops grown at an elevation of 6000 feet and above. Late blight was subsequently found to occur in the plains also. Dastur (1948 AD) described two new diseases of potato: leaf rot caused by a species resembling *P. parasitica*, and tuber rot caused by a new species, *P. himalayensis*.

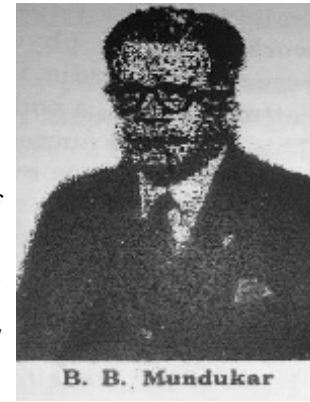
The studies on black tip of mango, aquatic fungi, saltation in fungi were carried out by **Sachindra Nath Dasgupta** (1902 AD–1990 AD). He also studied in detail saltation in fungi with reference to that occurring in Cytosporina, Phomopsis and Diaporthe. He described over 70 species of aquatic fungi.

Cereal rusts received attention in India as early as, 1907 AD when Milligan reported a heavy outbreak of rust on wheat in Punjab. Studies on the epidemiology of rusts and methods of their control comprise some of the most important contributions to plant pathology carried out in India. **Mehta**

initiated in 1929 AD a series of experiments on the annual recurrence of black stem rusts on wheat in India, and published his results in a monograph in 1948 AD. He was the first person to initiate studies on epidemiology of wheat rusts. He showed that *Berberis* and other alternate hosts do not play any significant role in the perpetuation of wheat rusts in India, and that there is no local source of infection to account for the recurrence of rusts in the plains, where the intense summer heat destroys all the rust inoculum of the preceding season. He showed that the main source of inoculum responsible for the fresh infection in the plains is derived from rust infection on the hills, where the crop is grown early. As a result of extensive surveys made periodically all over the wheat-growing areas of India, it was found by **Joshi** et al. (1971 AD) that the black stem rust, which survives in the Himalayas, is not an important source of infection; the immense bulk of inoculum comes from the south (Nilgiri and Pulney hills). He also highlighted the prevalence and distribution of several races of wheat rusts in India. He was a pioneer in establishing Flowerdale Rust Laboratory (now under DWR, Karnal) where pathotype analysis of rust is taken. In his honour, this laboratory launched the Newsletter *Mehtaensis* (named after Mehta) in 1981 AD. He published a monograph entitled *Further Studies on Cereal Rusts in India* in 1948 AD.

Mitra in 1937 AD recorded *Tilletia indica*, a new bunt (Karnal bunt) on wheat. The disease was thought, erroneously, to be soil borne. Later studies showed that it was air borne, and the infection was not systemic (Mundkar, 1943 AD).

Three plant disease epidemics stimulated greatly the growth of plant pathology in India. These were the great Bengal famine of 1942 AD known as *Helminthosporium* blight of rice; the severe wheat shortage in Madhya Pradesh during 1946 AD and 1947 AD due to wheat rust, and the red-rot epidemic in 1938 AD–1942 AD on sugar cane in Uttar Pradesh and Bihar state. Losses from diseases generally were enormous. Annual losses due to wheat rust alone have been estimated to be about 0.3 million tonnes of wheat worth about 32.2 million rupees. The total overall decrease in production of food grains due to fungal diseases has been considered to be about 5 million tonnes a year (Raychaudhuri, 1968 AD).


B. B. Mundukar

Dr. Mundkar B.B. was appointed as mycologist in the division of mycology at IARI, Pusa, Bihar (1932 AD). His work on control of cotton wilt in Maharashtra reduced losses to a large extent. The credit for identifying and classifying the smut fungi found in India also goes to Mundkur. He wrote a book *Fungi and Plant Diseases* in 1949 AD. He also wrote a

monograph in collaboration with Dr. M.J. Thirumalachar *viz*; *Ustilaginales of India; Supplements of Fungi of India* and *Genera of Rusts*.

The most important disease of rice in India was the *Helminthosporium* leaf spot caused to *H. oryzae* and occurred endemically almost every year (Singh, 1934 AD), and was responsible for the severe famine conditions in Bengal in 1942 AD. The nature and extent of damage caused by this disease were investigated, and trials laid for controlling the primary seed-borne infection by Padmanabhan et al. (1948 AD).

Green-ear disease is serious on bajra (*Pennisetuni typhoides*). **Chaudhari** (1931 AD) first showed that the oospores of the pathogen, *Sclerospora graminicola* in soil were responsible for its propagation. Artificial infection of the host could be accomplished only under very humid condition.

Sahay Ram Bose was known for his pioneering work in the study of bracket fungi and the discovery of antibiotics Polyporin and Campestrin. Bose published his findings in Polyporaceae of Bengal, a monograph in 11 parts with photo-prints, between 1918 AD and 1947 AD. Bose's work on Golgi Bodies in the basidia of *Polypraceae* attracted criticism from renowned botanist **J.B. Gatenby**. He investigated luminous fungi and ant-hill fungi from termite nests. Bose studied edible fungi in India and advised widely on their cultivation. He studied the effects of radiation on some polypores in culture in 1938 AD. Bose built up over the years a herbarium for bracket fungi by collections from different parts of the world. The herbarium, containing about 4,000 specimens of *Polyporaceae*, was entered in the list of World Herberia, Index Herbarium, Utrecht, The Netherlands.

Malformation is a devastating and somewhat mysterious disease of mango (*Mangifera indica*). During the last six decades, different researchers have attributed it to different causes: nutritional imbalance, virus, mites, etc. Affected shoots from mango trees have yielded *Fursarium moniliforme* (*Gibberella fujikuroi*) which reproduces the disease in healthy inoculated seedlings grown in the glasshouse and kept free from mites by **Summanwar** et al. (1966 AD). The disease has been shown to be systemic in branches, and in preliminary trials good results have been obtained with benlate and aphidan for control of this malady. It was suggested by Anupam Varma (1969 AD) that a judicious combination of pruning and application of insecticides, fungicides, and growth regulators may prove effective in controlling the disease.

Strong schools for fundamental pathology, especially the biochemistry of host–parasite interaction, were established at Lucknow and Madras University under the leadership of **S.N. Dasgupta** and **T.S. Sadasivan**, respectively. Dasgupta studied the role of enzymes in pathogenicity; a general high metabolic rate and higher level of several enzymes were shown in virulent strains by Varma J.P. (1964 AD). Sadasivan's school developed the concept of

vivotoxins and worked out the mechanism of cotton wilt caused by *Fusarium vasinfectum*. The production of fusaric acid by *F. vasinfectum* as a vivotoxin has been demonstrated by Kalyanasundaram in 1955 AD.

T.S. Sadasivan

It was shown by Sadasivan and Subramanian (1954 AD) that iron, although present in sufficient quantity in a living system, may be bound up with fusaric acid as a chelate and therefore, unavailable to the host plant. A correlation existed between the amount of fusaric acid produced in vivo and the amount of heavy material chelated. Further investigations revealed that chelating of zinc could also be one of the causes favouring wilt. Sadasivan (1958 AD) started the studies on bio-chemistry of host-parasite relationship.

The research in soil mycology and soil-borne plant diseases, focused on taxonomy and ecology of *Fusarium* was started by **Subramanian C.V.** His research contributions relate to mycology, plant pathology, and evolution. His fungal exploration of the tropics, especially southern India, the Kumaon Himalayas, the far-east, and Australia leading to discovery and description of many new genera and species, is a major contribution. His studies of the perfect states of *Aspergillus*, conidiogenesis based classification of Hyphomycetes and taxonomy and biology of fungi are widely recognised. Exploration of the western ghats brought forth an inventory of its remarkably rich mycodiversity. His main interests focused on the role of internal factors in evolution. His researches spanning four decades are summarised in the publications: *Hyphomycetes* (ICAR, 1971 AD) and *Hyphomycetes: Taxonomy and Biology* (Academic Press, 1983 AD). His contributions to hyphomycetology are well recognised in the recently published epitome. The Genera of Hyphomycetes is authored by Seifert et al. (2011 AD). He authored three books, edited/co-edited another five and published over 200 original papers. He also co-authored Soil Microfungi of Israel jointly with **S. P.Wasser** (2001 AD)

Certain newer diseases of wheat have come into prominence in late sixties because of new agricultural practices, such as extensive use of fertiliser, intensive irrigation, and use of the new high-yielding varieties. Leaf blight of wheat caused by *Alternaria triticina* is one such example (Raychaudhuri, 1969 AD).

Subramaniam Nagarajan's work on formulation of Indian Stem Rust Rules for *Puccinia graminis tritici* made it possible that no serious crop losses due to rust epidemics are observed today. He identified two new leaf rust resistance genes: *Lr*48 and *Lr*49.The life cycle of flax rust and identification of physiological races of cereal rusts were made by **Dr. Raghubir Prasada**. He also made significant contribution on *Alternaria* blight of wheat.

Rajender Kumar Grover conducted research on various aspects of plant diseases especially on bio-ecological interaction of fungi toxicants for efficient disease control. His contributions to the soil borne diseases management have been widely recognised throughout the world.

R.S. Singh was a dynamic teacher, researcher, writer in plant pathology at Pantnagar. He worked intensively on biological control and ecology of soil borne pathogens. He also wrote a classic book on *Plant Diseases* published in 1963 AD which is known as "Lighthouse of plant pathology" in India.

A. Mahadevan studied biochemical changes in diseased plants and enzymes. He wrote the book Growth Regulator, Microorganism and Diseased Plants.

C.D. Mayee, is a well known pathologist of 80s. He wrote the book *Phytopathometry*. His major contribution is on groundnut and sunflower pathology. He served as Vice-Chancellor of Marathawada Agriculture University, Commissioner of Agriculture to Government of India and Chairman, Agriculture Scientist Recuritment Board of Indian Council of Agriculture Research. He was also the President of Indian Phytopathological Society. On these various positions he made an emence contribution not only in plant pathology but also in agriculture as a whole.

6.6.2 On bacterial diseases

Coleman (1909 AD) was the first to establish the bacterial association with bangle blight in potato. **Mann and Nagpurkar** (1921 AD) isolated the organism, established its pathogenicity, and studied its biochemical and physiological properties. The pathogen has now been shown to cause brown rot or wilt of potato, which has been reported from Madras (Chennai), Uttar Pradesh, Bihar, Bengal and other parts of India. The same organism also affects banana and brinjal and causes wilt of tomato and tobacco. **Hutchinson** (1913 AD) developed his concept of toxins while working on this disease.

Hutchinson (1917 AD) first reported the yellow ear rot also known as tundu disease of wheat (*Triticum vulgare*), which had occurred in Punjab since 1908 AD.

Patwardhan (1928 AD) reported black rot of cabbage in India. **Patel** (1949 AD) reported that *X. campestris* on inoculation caused black rot of other cruciferous plants. Sever black-vein disease of cabbage was later reported in Bombay and West Bengal.

Chaudhuri (1935 AD) suggested that *Corynebacterium tritici* causes the ear cockle disease of wheat but later studies by **Vasudeva and Hingorani** (1960 AD) showed that the presence of both a nematode (*Anguilla tritici*) and the bacterium is necessary. Red stripe of sugarcane, caused by *X. rubrilineans*, was first reported in 1933 AD from India.

Although bacterial diseases were important and widespread, very little work was done until 1950 AD. **M.K. Patel** established a school of plant bacteriology at the College of Agriculture, Poona (Pune). He reported the first new species, *Xanthomonas uppalii* on *Ipomoea muricata* in 1948. Patel and his associates have established more than 40 new species of plant-pathogenic bacteria, mostly belonging to the genus *Xanthomonas*. Other active centers of plant bacteriology have since been developed at the Division of Mycology and Plant Pathology at the Indian Agricultural Research Institute, New Delhi, College of Agriculture, Poona, and IDHR, Bangalore, and many bacterial diseases of cereals, sugarcane, fiber, oil seeds, vegetables, legumes, and fruit trees have been studied in detail.

Gummosis of sugarcane caused by *X. vasculorum*, a very serious disease of the crop was reported from Madras in 1960 AD.

Black arm of cotton was reported in Madras in 1918 AD. Although **Uppal** found the disease to be of minor importance, severe epidemics were reported from Madras and Bombay. The disease has become very serious since the recent replacement of resistant diploid, rainfed, indigenous cottons (*Gossypium arboretum* and *G. herbaceum*) with high-yielding but susceptible irrigated, tetraploid cotton; (G. hirsuturiz and G. barbadense) (Verma and Singh, 1971 AD)

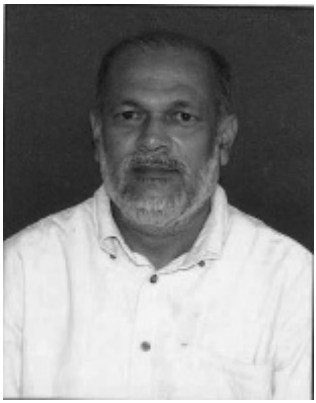
During 1970 AD and afterwards a strong group of plant bacteriologist existed at IARI, New Delhi. Dr. J.P. Verma and his student were working on cotton bacterium, Dr. P.N. Patel and Dr. Jindal were working on legume bacterium while Dr. Durgapal and Dr. B.M. Trividi were working on paddy bacterium.

Jeevan Prakash Verma started the pioneering work on *Xanthomonas campestris* pv. *malvacearum* causing bacterial blight of cotton. He laid a solid foundation of Indian Plant Bacteriology with his students. He wrote important book like The Bacteria which is used by undergraduate and postgraduate student and another book on Bacterial Blight of Cotton. He was the President of Indian Phytopathological Society and also editor of Indian Phytopath for several years. He founded a strong group of plant bacteriologist in India.

His students include R. P. Singh, S.G. Borkar, P.P. Sinha, S.D. Nafde, Datta Mujumdar, Satya Narayan and Jayshree. R.P. Singh devised the system of race classification of Xanthomonas malvacearum and reported 32 races of the cotton bacterium in India. P.P. Sinha worked on use of Phylloplane bacteria in the management of cotton bacterial infection while S.D. Nafde worked on pesticide resistant in the bacterium and curing of the resistanct gene. Datta Mujumdar, Satya Narayan and Jayshree worked on molecular aspects of the bacterium.

Among these students, **S.G. Borkar** rose to the position of Professor and Head in Agriculture University and kept contributing to the field of bacteriology.

The interaction between genotypes of *Xanthomonas campestris* pv. *malvacearum* and *Gossypium hirsutum*; Dynamics of phenols and diphenoloxidase contents of cotton cultivars during hypersensitive and susceptible reaction induced by *Xanthomonas* was studied by Borkar in 1980 AD–1982 AD (**Borkar** and **Verma**, 1987 AD; 1991 AD). Transmission of *Xanthomonas malvacearum* through spotted bollworms. (Borkar et al., 1980 AD) and through red cotton bug (Verma et al., 1981 AD) was also reported. Albinism in Xanthomonas due to plant phenolics was also reported by Borkar (Borkar and Verma, 1984 AD). Similarly effect of mixed race inoculums on changing of disease reaction (Borkar and Verma, 1984 AD) and role of EPS as water soaking disease reaction inducing factor was first time reported in India by Borkar and Verma (1989 AD).



Dr. S.G. Borkar went to plant bacteriology laboratory in Angers, France in 1984 AD to carry his post doctorate research in plant bacteriology. After his return from France he joined Jawaharlal Nehru Agriculture University, Jabalpur and subsequently Mahatma Phule Agriculture University, Rahuri in 1989 AD. In Mahatma Phule Agriculture University he laid a good foundation in plant bacteriology and trained several students in this field. His research area spanned widely with development of four rust resistant high yielding wheat varieties, use of probiotics in plant disease management, search for new antibacterial compound, antiviral compound, new biocontrol agent for nematode, yeast as biocontrol agent, tapioca granules and glucose globules are carrier for biocontrol agents. Development of Rahuri paste for bacterial blight of pomegranate, bacterial blight of pomegranate management protocol, TAPS as low cost universal medium for fungi and bacterial plant pathogens etc are his important contributions. He lead the wheat research in peninsular India as zonal wheat co-ordinator for more than 8 years with the development of rust resistant high yielding varieties for different sowing conditions and moisture regime.

His classical work is on the report of spines as new avenues of infection (1997 AD) and mycocyanin as new anthocyanidin group in fungi (2001 AD) suggesting the evolution of plant from fungi kingdom. He has discovered the new bactericide 4-(4-chlorophenyl) pyridine from medicinal plant. He suggested one Race - horizontal resistant model (1990 AD) to screen cotton varieties against bacterial blight disease. He searched differentials to formulate race identification scheme for bacterial blight of grapevine. He has written two important books in plant bacteriology i.e. Laboratory Techniques in Plant Bacteriology and Bacterial Diseases of Crop Plants. Beside to these, three other books on the subject of Pathology and Agriculture Microbiology was contributed by Borkar. He has published 90 research papers and guided 28 students for their M.Sc and Ph.D. degrees. He made a first documentary film on plant pathology in India (for this film a commendation certificate was awarded by Indian phytopathological society in 1993 AD) and established a plant disease museum for general public at Dhule in Maharashtra. He is the founder of the plant quarantine laboratory as well as eco-friendly disease management laboratory in MPKV, Rahuri with electron microscope facilities, which are being used by researchers from different laboratories. He has six patents to his credit for his fundamental inventions. He is a known plant bacteriologist in India and abroad.

The important work on canker disease caused by *X. citri* was carried out by Luthra and Sattar (1940 AD) at Punjab. The disease is known to have originated in India, as the disease specimens (available at Kew, England) were collected from Dehra Dun in the Himalayas during 1827 AD–1831 AD. The disease in India is now well established in citrus orchards all over the country.

The leaf spot of mango was first reported from Poona and Dharwar in 1947 AD by Patel and Kulkarni although, it probably occurred much earlier as the similar spots were observed on leaves in the herbarium of the Forest Botanist, Dehra Dun, (on the samples collected in Bihar in 1881 AD and at Dehra Dun in 1908 AD).

Srivastava and Rao (1969 AD) worked on the bacterial blight of rice in India as the disease was reported in 1959 AD in Bombay province and in a short time became very serious and widely distributed, attacking the commonly cultivated varieties. Variation in isolates of the pathogen in *X. oryzae*, has been studied by these scientists. Indica type rice varieties like IRRI 69/469, 70/470, and M. Sung Son and **Japonica type** Tainan-3 and Chianunsl-242 have been reported to be resistant.

Several new and important bacterial diseases have been described in recent years on bajra and ragi (Rangaswami, 1961 AD), castor bean (Patel, 1951 AD), sesamum (Rangaswami, 1961 AD), and legumes (Rangaswami, 1961 AD). A complete list of bacterial diseases is given by Rangaswami in his book Plant Diseases in India. **Y.S. Kulkarni** worked on bacterial leaf spot of castor and stem canker of pigeon pea.

In recent years (2005 AD onwards), bacterial blight of pomegranate known as oily spot disease of pomegranate created havoc in pomegranate orchards in Maharashtra, Karnataka and Andhra Pradesh causing losses of 100 million rupees per year. The bacterium Xanthomonas axonopodis pv. prunicae is very aggressive and developed resistance to several antibiotics, due to which the infection could not be controlled. Similarly all the available varieties and germplasm tested were found susceptible to the bacterium at Mahatma Phule Agriculture University, Rahuri. The varieties like Nana and Daru, thought to be resistant at Banaglore, were also found susceptible in the tests carried out at Rahuri University by **Borkar and Raghuwanshi** during 2010 AD. The pathogenic variability was also shown to exist within different parts of Maharashtra (Raghuwanshi et al., 2012 AD). Therefore, strategic research on bacterial blight of pomegranate was carried out at **Dr. Borkar**'s laboratory in Mahatma Phule Agriculture Univeristy, Rahuri by S.G. Borkar and K.S. Raghuwanshi and their students with the development of oily spot disease management protocol and Rahuri paste.

M.V. Naidu (1972 AD) reported bacterial leaf spot of grapevine caused by Xanthomonas viticola from college of Agriculture farm at Tirupati. **S.G.**

Borkar (2002 AD) reported races of Xanthomonas viticola prevalent in Maharashtra and gave the system and differentials for the identification of races of this bacterium.

Professor **B.P. Chakravorty** worked at Maharana Pratap Univeristy of Agriculture and Technology at Udaipur. He led the bacteriological work in the region of Rajasthan covering the crops like castor, cluster bean, pigeon pea and cowpea. **Dr. Ram Kishan** in Indian Institute of Horticulture Research, Bangalore led a group of bacteriologist and worked on bacterial leaf spot of mango and bacterial leaf spot of grapevine.

A group of bacteriologist from Haryana Agriculture University, Hissar discovered the use of bleaching powder for the control of bacterial blight of rice and subsequently it was used as seed treatment to control the bacterial infection in some of the crops.

The recent trend in plant bacteriological work in India mainly includes the molecular work on important plant pathogenic bacteria at IARI, New Delhi and some other laboratories.

6.6.3 On viral diseases

Plant viruses did not receive much attention in India as compared to fungal and bacterial diseases. One of the earliest records of virus disease in the country is the spike disease of sandal wood, first reported by **Coleman** in 1917 AD as a graft transmissible virus disease, now known to be caused by phytoplasma.

One of the first viral diseases to be investigated in India in late 30s was tobacco leaf curl transmitted by the whitefly *Bemisia tabaci* (Purthy, Pal and Tondan 1937 AD). The virus–vector relationship was studied and the virus has been shown to have a very wide host range, including tomato (Vasudeva, 1948 AD), papaya (Nariyani, 1956 AD) sunhemp, chilli (Mishra, 1931 AD), and a number of weeds and ornamental plants.

The next two decades were mostly devoted to investigations on dissemination and control of a number of virus diseases of economic crop plant such as sugar cane mosaic (Rafai, 1931 AD), stenosis or small leaf of cotton (Uppal et al.); Yellow vein mosaic of bhindi (Capoor and Varma, 1950 AD); tomato leaf curl (Vasudeva and Samraj, 1948 AD); *Katte* disease of cardamom (Varma and Capoor, 1955 AD); papaya mosaic (Capoor and Varma, 1959 AD); leaf curl (Nariani, 1956 AD) and virus diseases of temperate fruits (Azadf and Sehgal, 1958 AD). The investigations were mostly concerned with the vectors and sources of resistance. As a result, resistant varieties were reported for several viruses, and other sources of resistance in wild species such as *Carica cauliflora* for papaya mosaic (Capoor and Varma, 1961 AD), *Abelmoschus manihor* var *pungems* for yellow vein mosaic of bhindi (Nariani T.K. and Seth

M.L., 1958 AD); *Lycopersicon peruvimum* for tomato leaf curl (Nariani T.K. and Vasudeva R.S., 1963 AD); varieties Icbinose and Kairyonezumegaishi of *Morus ulba* and Oshimasho and Kosen of M. *latifolia* for mulberry mosaic (Raychaudhuri et al., 1965 AD) were reported. The whitefly, *Bemisia tabaci* generally were found to be an effective vector for a number of virus diseases of crop plants and they carried several viruses simultaneously.

The large number of virus diseases affecting cereals like *Eleusine* (Rao, 1965 AD) wheat (Raychaudhuri, S.P. and Ganguly, B. 1968 AD) pearl millet (Raychaudhuri, 1967 AD), bajra (Seth, M.L. 1971 AD) ragi (Yaraguntaiah and Keshava, 1969 AD); legumes like bean (Yaraguntaiah and Nariani, 1963 AD); pea (Sreenivasan and Nariani, 1966 AD); urid (Sahare, K.C and Raychaudhuri, 1963 AD) mung and sannhemp (Nariani, T.K. 1960 AD); broad bean (Azad R.N., 1961 AD) cowpea (Chenulu, 1968 AD), and plantation crops such as cardamom (Raychaudhuri and Chatterjee, 1961 AD), citrus (Vasudeva and Capoor, 1958 AD) and coconut (Summanwar et al., 1969 AD) have been studied. Fundamental problems such as purification, morphology of virus, serology, tissue culture, inhibition, and virus–vector relationships have received increasing attention.

A notable feature of these findings is the elucidation of the cause of some serious diseases of complex etiology such as citrus die-back, coconut root wilt, and sandal spike. The citrus die-back was shown to be caused by a complex in which greening disease (now shown to be due to a phytoplasma transmissible by psylla, *Diaphorina citri* (Capoor, 1967 AD) and fungi such as *Colletotrichum gloeosporioides*, *Diplodia natalensis*, *Curvularia tuberculata*, and *Fusarium sp*.) play a major role. The coconut root wilt, which has seriously affected the economy of the coconut industry in south India, has been associated with a rod-shaped virus (Summanwar et al., 1969 AD) and the sandalwood spike with a phytoplasma.

Antisera and particle morphology has been used in identification and detection of plant viruses in recent years, and fundamental studies on purification and serology have received attention with the introduction of the ultracentrifuge in most laboratories. A number of viruses, such as mosaic streak of wheat and tungro of rice (Raychaudhuri et al., 1969 AD); various cucurbit mosaics (Shanker et al., 1969 AD); cowpea mosaic (Chenulu,1969 AD); coconut root wilt (Summanwar et al., 1969 AD); barley mosaic, have been purified, their particle morphology studied and antisera prepared.

Tissue-culture techniques opened the way for investigations concerning host parasite relationships and inhibition studies, as well as production of virus-free plants by meristem-tip culture. A number of viruses such as chilli mosaic, sunhemp mosaic, tobacco mosaic, and potato virus X, have been

successfully eliminated in tissue culture (Raychaudhuri and Mishra, 1965 AD) and attempts are in progress to get virus-free plants of citrus by tissue-culture technique.

During the last few years inactivation of plant viruses has been attempted with plant extracts, microbial growth products, anti-metabolites, growth regulators, and ultraviolet and gamma radiations, to find some effective inhibitors of viruses for their control. Thiouracil and 8-azaguanine have proved effective in reducing virus concentrations and suppressing symptoms in several Cases (Raychaudhuri and Mishra M.D., 1965 AD). Inhibition of plant viruses has also been reported by extracts of chilli, acacia, Datura, deodar, and papaya latex (Raychaudhuri and Prasad, 1965 AD) and growth products of *Trichothecium roseum*, *Aspergillus niger*, and *Bacillus subtilis*, and ultraviolet and gamma radiations (Raychaudhuri and Prasad, 1965 AD)

Professor Anupam Varma has made important contributions to fundamental and applied aspects of plant virology, which are widely quoted and acclaimed. He has established a strong school of molecular virology in the country, which has provided lead to the understanding of the complexities of the devastating diseases caused by begomo, ilar, poty and tospoviruses in horticultural and field crops. Professor Varma's researches have played a key role in developing technologies for the management of viral diseases, through conventional and biotechnological approaches. An advance centre of plant virology was established by Anupam verma at IARI, New Delhi in 1990 AD onwards. He was also the President of Indian Phytopathological Society. He made a strong foundation of plant virology in the country.

6.6.4 On mycoplasma like organisms

Studies have been focused on diseases in sandalwood affected with spike disease, brinjal affected with little leaf (Varma, 1969 AD) and greening disease of citrus. The mycoplasma of the sandalwood spike and the greening disease of citrus have been cultured in the laboratory (Nayar and Ananthapadmanabha, 1970 AD) The effect of tetracycline antibiotics has been studied on these diseases, and suppression of symptoms reported (Nariani and Raychaudhuri, 1971 AD).

6.6.5 On non-parasitic diseases

Luthra (1921 AD) described *Striga* as a root parasite of sugar cane. Since then three species (*S. densiflora*, *S. enphrasioldes* and *S. lutra*) have been reported on sugar cane, rice and sorghum by Kumar in 1940 AD. Among other genera common in India are *Dendroplithoo* spp. (especially on *mango*) and Orobanche (on brinjal, tomato, cauliflower, turnip, etc.).

A school for the study of deficiency diseases was established in 1946 AD by **S.N. Dasgupta** at Lucknow University. The main work done was on black tip necrosis of mango, although this disease was first recorded in 1921 AD by **Woodhouse** and it became prevalent by the 1930 AD onwards.

It was observed quite early that orchards situated near the brick kilns sustained greatest damage. Both Sen and Dasgupta reported the production of limited necrosis when healthy mangoes or trees were subjected to coal fumes. The earlier observation that SO_2 could produce mango necrosis could not be confirmed. There was evidence that ethylene, another constituent near the brick kiln fumes, and a mixture of ethylene and SO_2 in certain proportion when administered in large doses continuously for a number of days produce typical black tip. It was found that ethylene in small dose (1:10,000) induced ripening, while heavier concentrations (10:10,v/v) produced dark brown lesions around lenticels all over the skin of the fruit. Finally the spots coalesced, forming dark brown patches. Other known deleterious constituent gases from brick kiln fumes, e.g., carbon monoxide and fluorine also failed to produce typical symptoms (Dasgupta and Sen, 1960 AD). It was, however, demonstrated that a spray with boron in the form of borax could successfully prevent necrosis. **Mango necrosis** is finally designated as a **boron-deficiency disease** (Dasgupta, 1959 AD).

Vasudeva and Raychaudhuri (1954 AD) reported a serious **disease of guava** from Rajasthan, characterised by severe reduction in leaf *size*, interveinal chlorosis, suppression of growth, die-back of shoots, and cracking of fruits. Foliar sprays and applications of zinc sulphate in soil, and shoot or trunk injections cured the disease. The disease was concluded to be due to **zinc deficiency**.

Yeshwant Laxman Nene reported "Khaira" disease of rice at Pantnagar due to zinc deficiency (1965 AD). He laid a strong foundation of plant

pathology at J.B. Pant University of Agriculture and Technology at Pantnagar. He made remarkable contributions in solving the mystery of chickpea wilt complex. He described two viral diseases (leaf crinckle and leaf curl) affecting mungbean for the first time. He also served at International Crop Research Institute for Semi-Arid Tropics in India as Deputy Director General of the Institute. Dr. Nene's book on Fungicide in Plant Disease Control is a well known title for the students of plant pathology.

6.6.6 On plant disease control

Some of the historical events in the use of fungicides in India were in 1885 AD, when **Ozanne** first used copper sulphate as fungicide for control of a sorghum smut **disease**. **Lawrence** used Bordeaux mixture for the first time in 1904 AD against *Cercospora* leaf spot of groundnut. **Coleman** claimed control of *Phytophthora omnivora* var. *arecae* on arecanut with Bordeaux mixture plus resin in 1915 AD.

Cotton anthracnose was controlled by seed dressing with an organo mercurial fungicide after delinting the seed with sulphuric acid (**Nene**, 1971 AD), **Narsimhan** (1930 AD) suggested the use of linseed oil in Bordeaux mixture for improving coverage and tenacity.

Use of antibiotics for the control of diseases of crop plant was fairly new in India. One of the crowning achievements of **Sahay Ram Bose** was his discovery of two antibiotics obtained from higher fungi *i.e*. Polyporin from *Polistictus Sanguineus* and Campestrin from *Psalliota Campestris*. **M.J. Thirumalachar** discovered several antifungal antibiotics like Aureofungin, Haymycin, Streptocycline and Antiamoebin. Lately, Hindustan Antibiotics has taken a leading role in the commercial manufacture of antibiotics and one of their products that has been used extensively as a seed dressing, a spray on standing crop, and a post-harvest dip is aureofungin.

As far as the biological control of plant diseases is concerned, till date 26 microbes have been included in the schedule to the insecticide act 1968 AD for production of microbial biopesticides. *T. viride, T. harzainum, Pseudomonas* sp., *Beauvaria bassiana, Metarrhizium anisopliae* and *Bacillus subtilis* are important biocontrol agents for management of various pest and diseases in India (Singh, 2012 AD).

6.6.7 On molecular and serological plant pathology

Since 1980 AD, a great emphasis has been laid on determining the genetic variability and relationship among the plant pathogens involved in disease development. Advances in molecular plant pathology have also provided a new set of diagnostic tools and techniques like PCR techniques, RFLP (Random fragment length Polymorphisms) and use of PCR with DNA hybridisation

that are used to detect and identify pathogens. Development and utilisation of detection and diagnostic methods based on enzyme-linked immunosorbent assay (ELISA), polymerase chain reaction (PCR) and microarray are gaining importance in Indian Plant Pathology in recent years.

Guleria et al. (2007 AD) used 19 *Rhizoctonia solani* isolates from rice growing regions of India with two marker i.e. RAPD and ISSR for molecular characterisation of genetic variability in this pathogen. **L.C. Bora** carried out genomic characterisation of microbial antagonists *viz.*; *Bacillus cereus*, *Citrobacter freundii*, and two fungal bioagents, *viz.*; *Aspergillus flavus*, and *Trichoderma viridae*. **M. Singh** developed a single step immuno-chromatography assay for rapid detection of quarantined Karnal bunt (*Tilletia indica*) disease of wheat.

T.S. Thind reported molecular characterisation and management of metalaxyl resistant populations of *Phytophthora infestans* in India. **D.K. Ghosh** studied molecular diagnostics and characterisation of major virus/ virus-like pathogens *viz*; citrus tristeza virus (CTV), Indian citrus ringspot virus (ICRSV), citrus yellow mosaic badna virus (CiMV), citrus exocortis viroid (CEVd) and citrus greening bacterium (HLB) infecting citrus in India by serodiagnosis using pathogen specific polyclonal/monoclonal antibodies and nucleic acid based diagnosis by PCR/RT-PCR techniques.

S.C. Dubey et al. developed probes and real time PCR assay for detection of *Rhizoctonia solani* infecting pulse crops. **R. Kapoor** et al. produced cocktail of polyclonal antibodies using bacterial expressed recombinant protein for multiple virus detection. **R. Ghosh** et al. studied molecular and morphological diversity in *Rhizoctonia bataticola* isolates causing dry root rot of chickpea (*Cicer arietinum* L.) in India. A total of 94 isolates collected from *R. bataticola* infected chickpea plants from different agro climatic regions of India. They were analysed with amplified fragment length polymorphism (AFLP) and rDNA-internal transcribed spacer (ITS) region sequencing. **R. Kumar and K.K. Mondal** investigated the possible role of *XopN*, one of the conserved T3SS effectors across phytopathogenic *Xanthomonas*. The use of *xopN* null mutant (Xap Δ*xopN*) for *Xanthomonas axonopodis* pv. *punicae* modulates cell-wall-associated immune response to induce bacterial blight in pomegranate. **A. Bhattacharyya** studied genetic diversity and pathogenic variability of *Fusarium oxysporum* f. sp. *cubense*

6.6.8 On marker assisted pyramiding of disease resistance genes

Marker assisted pyramiding of disease resistance genes termed as Breeding by Design can help to control the pathogen, which recurrently and rapidly develop their new virulence. Efforts are made in India under Asian Rice Biotechnology network (ARBN) to pyramid resistance gene against bacterial

blight of rice. Rice is among the first crops where marker assisted pyramiding of disease resistance genes were initiated. Rice varieties developed by using MAS have now been released for commercial cultivation for the first time in India. The variety amended as Improved Pusa Basmati-1 was developed by using conventional plant breeding approach integrated with MAS and two bacterial blight resistance genes Xa13 and Xa21 were incorporated in Pusa Basmati-1 (**Gopalakrishnan** et al., 2008 AD).

Another variety of rice resistant to bacterial blight was developed in non-basmati type rice in India by using MAS. PCR based molecular markers were used in a backcross breeding program to introgress three major bacterial blight resistance genes (Xa21, Xa13 and Xa5) into samba mashuri from a donor line (SS1113) in which all the three genes are present in a homozygous condition (**Sundaram** et al., 2008 AD).

These two reports successfully demonstrate the application of marker assisted selection for targeted introgression of BLB resistance genes into basmati types and non-basmati types variety of rice in India. So, there is a call for taking up such initiatives for other host pathogen system for control of serious loss causing diseases. Molecular markers can also help in assaying the germplasm for presence or absence of a particular disease resistance gene. Cloning of disease resistance genes by tagging approaches can identify the function of specific genes by uncovering a specific pathotype.

6.6.9 On transgenics research

Development of disease resistance through transgenic research is yet at primitive stage in India. Non-availability of resistance to plant pathogens can be overcome by research and transfer of resistance genes from other sexually incompatible species which is possible using genetic engineering approach. Disease resistance transgenic has been developed in banana and tobacco by transferring a synthetic substitution analogue of a short peptide, Maganin (**Chakarbarti** et al., 2003 AD). Tobacco plants transformed with the peptide showed enhanced resistance against *Sclerotinia sclerotium, Alternaria alternata* and *Botrytis cinerea*. Transgenic banana plants showed resistance to *Fusarium oxysporum f.* sp. *cubense* and *Mycosphaerella musicola* (**Kumar and Gupta**, 2012 AD). However, it remains to be seen how these plants perform under natural disease conditions.

6.6.10 On plant quarantine

In India, the Directorate of Plant Protection, Quarantine and Storage (DPPQS) under the Ministry of Agriculture implements the plant quarantine regulations for bulk consignments and National Bureau of Plant Genetic Resources under

Indian council of Agricultural Research (ICAR) is the nodal agency for safe movement of germplasm including transgenics. This work under the Plant Quarantine Regulation of Import in India order 2003 AD which came into force from 1st January 2004 AD, under the destructive Insect and Pests Act of 1914 AD. Although, the regulations are now in place, there are number of issues related to quarantine of germplasm as it has been drafted more for facilitating bulk imports than for exchange of germplasm. Under this order a pest risk analysis (PRA) has been made mandatory and the various schedule of the order give lists of crops for which a generic PRA is given.

Dr. Ravi Khetrapal headed Plant Quarantine unit at National Bureau of Plant genetic resources at IARI, New Delhi for more than 7–8 years and drafted several regulations while intercepting the pathogens. He also served as Regional Director, south-east Asia Region of Common Wealth Agricultural Bureaux. He was also the President of Indian Phytopathological Society. He immensely contributed in the development of rules and regulations of Plant Quarantine and restriction of exotic pathogens in the country.

Maximum planting material is imported in the jurisdiction of Mahatma Phule Agriculture University in western Maharashtra. Dr. S.G. Borkar designated as inspection authority of Plant Quarantine where he has intercepted strawberry bacterial leaf spot pathogen in strawberry, rust of chrysanthemum on chrysanthemum and dry rot pathogen of garlic as quarantine pest. Due to his efforts these were not established in the western Maharashtra region.

6.6.11 Extension of plant pathology in India

Besides teaching and conducting research on different aspects of plant pathology, Transfer of Technology and Plant Clinic was established for the benefit of farmers by Plant Pathologist of India. The real dalliance for Transfer of Technology and Plant Clinic started when **Srivastava** joined as Extension Pathologist in 1976 AD. He organised and chaired a special session on Plant Health Clinic during 9th ICPP at Turin, Italy in 2008 AD and at International Conference in Globalised Era at New Delhi in 2010 AD. Presently he offers free service to growers and scientists on diagnosis and control of pests, and establishment of plant health clinic through his web portal www.xsgrowth.com. (M.P. Srivastava, 2014 AD)

6.6.12 Contribution of well known Indian pathologist

Several other Indian plant pathologists who made noteworthy contribution in their respective area of research are highlighted in tabular form.

Name of the Pathologist	Area of Research
A. Mahadevan	Physiological plant pathology
A. Narain	Minor diseases of rice and toxins
A.P. Mishra	Helminthosporium diseases
Abrar Khan	Nematode diseases of vegetables and control
A.K. Sorbhoy	Fungal taxonomy and *Rhizotonia* diseases
Amerika Singh	Wheat diseases
A.N. Mulkhopadhyay	Biocontrol of plant pathogens
Anupam Varma	Viral diseases
B. Chandra Mouli	Tea diseases
B.K. Bakshi	Forest pathology
B.L. Jalali	Mycorrhiza and integrated disease management
B.B. Nagaich	Viral diseases
Bharat Rai	Wilt diseases of pulse crops
B.L. Chona	Sugarcane diseases
B.L. Chopra	Diseases of cotton
B.M. Singh	Plant disease management
B.N. John	Mycology and mycorrhiza
B.N. Uppal	Cereal smuts, downy mildews of millets and maize
B.P. Chakravarty	Bacterial diseases
B.P. Singh	Viral diseases, Potato diseases
C. Manoharachary	Post-harvest diseases and mycorrhiza
C.V. Subramaniam	Wilt of cotton
C.D. Mayee	Groundnut diseases, sunflower diseases and plant disease forecasting
Chtreshwar Sat	Biocontrol of plant diseases
Gopal Swarup	Nematology
C.S. Venkata Ram	Tea diseases
D. Ganguly	Helminthosporium disease of rice
D. Kumar	Mycorrhiza

D. Lalitha Kumari	Fungal biotechnology of antagonistic organism
D. Suryanarayana	Downy mildew diseases
Dharam Vir	Fungicides
D.J. Bagyaraj	Mycorrhiza and biocontrol
D.K. Aron	Soil-borne diseases and biocontrol
D.N. Srivastava	Bacterial blight of paddy and diseases of fruits and vegetables
D.V. Singh	Bunts of wheat
G. Rangaswami	Bacterial diseases, authored *Diseases of Crop Plants in India, Bacterial Plant Diseases in India*
G.C. Luthra and A. Sattar	Gram blight and wilt, solar heat treatment against loose smut of wheat
G.S. Shekhawat	Bacterial diseases of potato
H. Chand	Bacterial diseases
H.K. Saksena	Rhizoctonia diseases and gram rot
T.K. Nariani	Viral diseases
Hari Om Agrawal	Viral diseases
H.C. Arya	Downy mildew of pearl millet.
H.N. Vallla	Viral diseases
H.S. Shetty	Induced resistance in pearl millet
H.S. Sohi	Diseases of ornamentals and fruit crops and mushroom culture
I.K. Bagchee	Forest pathology
J.L. Kaul	Diseases of fruits
J.P. Verma	Bacterial diseases specially bacterial blight of cotton
J.S. Booty	Powdery mildews
J.S. Chauhan	Diseases of oil seed crops
J.S. Grewal	Diseases of pulse crops
K. Jindal	Bacterial diseases
K.C. Alexander	Sugarcane diseases
K.G. Mukerji	Mycorrhiza and biocontrol
Kishan Singh	Sugarcane diseases
K.J. Shrivastva	Onion and garlic diseases
K.M. Safeeulla	Diseases of pearl millet

K.M. Vyas	Soil-borne diseases
K.P.V. Mason	Coconut diseases
K.S. Bhargava	Viral diseases
K.S. Bilgrami	Mycotoxins
L.M. Joshi	Wheat rusts
M. Mitra	Helminthosporium diseases, discovered the new bunt of wheat caused by Neorossia irulica
M.N. Kamat	Ascomycologist. Control of plant diseases and author of *Introductory Plant Pathology*
M.V. Pagvi	Indian Ustilaginales. *Physodenna. Tapharina marulans* and rusts
M. Narasimhan	Phytophthora diseases, sandal-spike. downy mildews of grasses and cereals, heteroecism in rusts, entomogenous fungi and biocontrol of insect pests
M.K. Hingorani	Bacterial diseases
M.K. Patel	Bacterial diseases, advocated a new family Phytobacteriaceae to include all phytopathogenic bacteria
M.M. Payak	Maize diseases
M.N. Khare	Pulse diseases
M.P. Srivastava	Post-harvest pathology
M.S. Chatrath	Fungicides
M.V. Naydu	Viral and bacterial diseases
M.W. Khan	Powdery mildews
N. Prasad	Fusarium diseases and Phytophthora blight
Narayan Rishi	Viral diseases of legume and their management
M.U. Thirumalachar	Monograph *Ustilaginales of India. Uredinales of the World.* Cercosporae. *Physoderma. Cephalosporium* etc. At Hindustan Antibiotics Ltd., Pimpri, Pune. He discovered a number of antibiotics such as Haymycin, Aureofungin, Antibiotic 226, Antiamoebin, etc.
P. Fatima Rao	Soil-borne diseases
P. Vidhyasekaran	Physiological plant pathology
P.K. Koshy	Nematode diseases in plantation crops
P.R. Mehta	Diseases of cereals and millets and plant protection

Pushkarnath	Potato diseases
R. Jeyarajan	Biocontrol and viral diseases
R.K. Saksena	Cytology of Pythiaceac and ecology of soil fungi
R. Kalyansundaram	Wilt diseases, fusaric acid as a wilt toxin
R.N. Tandon	Physiology of fungal pathogens and post harvest diseases of fruits and vegetables
R.P. Asthana	Control of smuts of millets
R.P. Singh	Bacterial diseases
R.A. Singh	Rice diseases
R.D. Parasher	Bacterial diseases
R.J. Singh	Rice diseases
R.K. Grover	Fungicides
R.K. Hegde	Phytophthora diseases of arecanut coconut and Piper nigrum
R.P. Purkayastha	Phytoalexins and induced resistance
R.P. Thakur	Diseases of millets
R.S. Drivedi	Soil mycology and root diseases
R.S. Singh	Organic amendments and control of soil borne pathogens. Author of the books: *Plant Diseases* and *Diseases of Vegetable Crops*
R.S. Vasudeva	Root rot of cotton and viral diseases of potato
S.B. Mathur	Seed pathology
S. Malti	Betelvine diseases
S. Mukhopadhyay	Viral diseases
S. Nagarajan	Wheat diseases
S.P. Raychaudhuri	Viral diseases of crop plants
S. Singh	Viral diseases
S. Sinha	Blasts
S.Y. Padmanabhan	Diseases of rice
S.G. Borkar	Bacterial blight of cotton and wheat rust diseases
Satyavir	Sugarcane diseases
S.B. Chattopadhyay	Diseases of rice and revised the book entitled *Fungi and Plant Disease* (originally written by B.B. Mundkur).
S.B. Saksena	Mycologist of eminence, discovered *Saksenia* which is a human pathogen, root diseases and biocontrol

S.B. Sharma	Nematode diseases
S.K. Saxena	Nematode diseases
S.M. Paul Khurana	Viral diseases of potato
S.N. Das Gupta	Black tip of mango
S.P. Kapoor	Viral diseases
S.S. Aujla	Wheat diseases
S.S. Clutha	Pearl millet diseases
S.S. Sokhi	Vegetable diseases
Sudhir Chandra	Post harvest pathology, mycorrhiza and biocontrol
T.S. Ramakrishnan	Contributions on genera *Pythium, Phytophthora, Colletotrichum* and the rusts and his monograph on *Diseases of Millets* published by the ICAR
T.N. Lakhanpal	Tree diseases
T.S. Sadasivan	Established the school of Mycology and Plant Pathology at Madras University, Madras (now Chennai) and it was here that the foundation of research on soil borne pathogens was laid and also the physiology of wilting, specially cotton wilt, was investigated and the control of wilt diseases by trace element amendment
Uma Kant	Insect galls
U.P. Singh	Diseases of pulse crops
V. Agnihothrudu	Tea diseases
V. Mariappan	Rice diseases
V. Muniyappa	Viral diseases
V.K. Gupta	Diseases of apples
V.N. Pathak	Post-banal diseases of fruits and vegetables
V.P. Agnihotri	Pythium, sugarcane diseases
V.P. Bhide	Bacterial diseases
V.R. Mali	Viral diseases
V.R. Reddy	Viral diseases
V.S. Ahlawat	Viral diseases
W.M. Khan	Powdery mildew
Y.S. Kulkarni	Bacterial diseases of plants
Y.L. Nene	Khaira disease of rice and authored *Fungicides in Plant Disease Control*
Y.R. Sarma	Diseases of plantation crops

6.7 Development of plant pathology in Japan

6.7.1 Expansion stage (1913 AD–1926 AD)

Discovery of the pathogenic nature of fungi led to an intensive study of these organisms as the cause of plant diseases. **In the Taisho Era**, studies on the physiological nature of pathogenic fungi appeared gradually, and plant pathologists were not necessarily limited to simple etiological works of diseases but establishment and practice of control measures for the diseases were also studied.

Bakane disease (characterised by elongation of seedlings) has been known from ancient times in Japan. **Hori**, first reported that it is a parasitic disease, but erroneously identified the causal fungus as *Fusarium heterosporum Nees*, with the comment that he had some doubt about this identification. In 1916 AD **Yosaburo Fujikuro** found the perfect stage and it was named *Lisea fujikuroi* by **Sawada** (1919 AD). **E. Kurosawa** (1926 AD) showed that the culture media in which the fungus had been growing would induce the disease in rice plants. The active component, gibberellin, was isolated from the fungus and named by **T. Yabuta** in 1935 AD. The fungus was later put in the genus *Gibberella* under the name of *G. fujikuroi* (Sawada) Wr. (Ito and Kimura 1931 AD).

In October 1914 AD, **Umenojo Bokura** started a monthly magazine entitled **Ochu-Gai Zasshi** (Journal of Plant Protection). At that time, Bokura was a pathologist in the Plant Pathology Section of the Imperial Agricultural Experiment Station at Nishigahara. This journal continued until 1943AD and contained articles written in Japanese, covering both the fields of plant pathology and economic entomology. It contributed a great deal to the progress and dissemination of knowledge of plant diseases in Japan.

The Phytopathological Society in Japan (Nihon Shokubutsu Byorigaku-Kai) was established in February 1916 AD, and Mitsutaro Shirai was elected as the first president.

Kakugoro studied species of fungi related to sclerotial diseases of rice plants and the pathogens of hollow stalk (black leg) and bacterial wilt of tobacco plants. A book, **Sakumotsu Byogai Zuhen** (Illustrated Monograph of Crop Diseases) published in 1934 AD, gained large public favour. **Hazime Yoshii's** work on pathological anatomy and physiology and his book, **Anatomical Plant Pathology** (KaibO Shokubutsu BySri Gaku), published in 1947 AD is still a prominent textbook for graduate study.

A chair for plant pathology was provided in Kyoto Imperial University in 1924 AD with **Takewo Hemmi** as professor. He contributed on the Morphological and Physiological Studies on Anthracnose Fungus.

Since the opening of international commercial activities, many agricultural crops have been imported and were sometimes contaminated with plant pathogens and pests. Thus, new diseases or new races of pathogens were imported, bringing about great damage to crops. Fire blight bacteria of apple, anthracnose fungus of peach, late blight fungus of potato, crown gall bacteria, powdery mildew and downy mildew of grape were introduced with contaminated imported seedlings. Effective plant quarantine service was thus required and in 1914 the government announced regulations for plant quarantine and quarantine stations were established at Yokohama and other areas.

6.7.2 Active stage (1927 AD onwards)

In this period plant pathology in Japan made steady progress as a science of plant disease and plant medicine and was not limited to simple applied mycology.

6.7.2.1 Advances in experimental epidemiology

Hemmi (1933 AD) made extensive investigations on the relationship between environmental factors and the occurrence of rice blast. He reported that blast disease of rice plants was caused by *Pyricularia oryzae*, which was the most important and destructive plant disease in Japan, being widely distributed throughout the country and causing great loss in yield. Hemmi, further elucidated the relationships between occurrence of rice blast disease and environmental factors such as soil water, atmospheric humidity, and temperature.

The environmental conditions in Hokkaido are unfavourable for cultivation of rice plants as exemplified by the low soil and air temperatures and peat-moss soil. Thus, rice crops there suffer from severe blast disease every year. Beginning in 1927 AD, **Ito** and **his co-workers** in Hokkaido Imperial University and Hokkaido Agricultural Experiment Station concentrated on the problem of controlling blast disease. They concluded that the most effective protective measures were seed disinfection and treatment of diseased carryover rice straw by scattering them in fields and using a cover spray of Bordeaux mixture. Cultivation of resistant cultivars and soil improvement were also recommended. Ito practiced his theory in the Sorachi district of Hokkaido in 1934 AD to prevent primary occurrence and spread of the disease. The result was very successful and may be the first case of using a cover spray in a rice field to successfully control blast disease. In 1949 organic mercury fungicidal sprays replaced the Bordeaux mixture without noticeable spray injury (Akai, 1958 AD).

6.7.2.2 Advances in virus disease investigations

In the beginning of the **Showa Era** (1927 AD) virus diseases of plants became the subject of research because of their scientific interest and economic importance and rice dwarf and stripe diseases of rice plants, tobacco-mosaic disease, and others were also brought to light by many investigators.

Fukushi (1933 AD) demonstrated virus transmission through eggs of insect vectors in rice dwarf virus. After the Second World War, electron microscopic techniques were introduced into various fields of biological sciences and in virology these techniques helped to elucidate the fine structures of viruses. Serological and immunological approaches to characterisation of viruses have also been performed in many laboratories and institutes.

Histopathological observations of diseased plants have also progressed. The Kaib O Shokubutsu By Origaku (Anatomical Plant Pathology) published by **Yoshii and Kawamura** in 1947 AD is the first book concerning pathological anatomy of plants in Japan.

With the help of electron microscopy extensive anatomical and cytological studies on host–parasite interaction were made with physiological and biochemical analyses. The mechanism of resistance has also been discussed from various viewpoints.

An Institute for Plant Virus Research was founded at Chiba in 1964 AD.

6.7.2.3 Discovery of mycoplasma-like organisms

An especially noteworthy event in Japan was the discovery of mycoplasma-like organisms in plants infected by yellows-type diseases, which had been hitherto considered to be virus diseases (Doi et al., 1970 AD).

6.8 Development of plant pathology in the People's Republic of China (PRC)

Plant pathology and entomology in the People's Republic of China (PRC) are combined into one discipline *i.e. plant protection*. This developed following the Cultural Revolution of the 1960 AD onwards. Among other subjects most scientists in China have become practitioners within their specialty with relatively few remaining as researchers in the more basic sense.

The function of plant protection and indeed all other areas of agricultural sciences are primarily one of the *extension* applied or mission-oriented research supported by comparatively little basic research. Indeed, the current total extension effort in China may be greater than that of any other country.

Plant protectionists on the commune are involved in diagnosis and disease forecasts; application of fungicides, insecticides, or biocontrol agents and implementation of demonstration plots. Often they are technicians with only limited formal education or peasants with considerable experience and some education. Facilities for culturing plant pathogens are not available at most communes, although fungi, edible forms, and those used for production of antibiotics are cultured on some.

Most agricultural colleges are relocated in rural areas usually at some distance from major metropolitan centres with construction of new facilities and development of new curricula. The model that may eventually evolve in most colleges is a department of plant protection with two divisions, one of plant pathology and other entomology.

6.8.1 Important fungal diseases dealt with

Wheat scab (caused mainly by *Fusarium roseum Graminearum*), leaf rust, stem rust (*P. gramims* f. sp. *tritici*), and stripe rust (*P. striiformis*) all were important in the past but currently are under control. Common bunt has been virtually eliminated.

On soybeans, *Alternaria* leaf spot, frog eye leaf spot, *Mycosphaerella* leaf spot, downy mildew, brown spot, and stem rot occur.

Few serious diseases of sorghum prevalent are sooty stripe (*Ramulispora sorghi*), which produces a large leaf spot similar to that caused by *H. turcicum*. Leaf spots caused by *Cercospora sorghi* are also present.

The key pathogens on different crops in the Shanghai area are, *Elsinoe fawcettii on* mandarin orange, *Gloeosporium laeticolor* on peach; *Physalospora piricola* and *Venchuria pyrina* on pear and downy mildew on cabbage.

A late leaf blight-resistant cultivar of potato, Aquila, developed in 1965 AD at the Academy of Agricultural Sciences in Shanghai, is planted to minimise possible losses from late blight.

The fungal diseases of cotton causing loss to varying degrees in Shensi and Kiangsu provinces are damping-off, (*Rhizoctonia sp., Fusarium moniliformc* and *Pythium* spp), anthracnose (*Colletotrichum gossyppii*;), alternaria leaf spot, (*Alternaria macrospora*) *phyllosticta leaf spot*, (*Phyllosticta gossypina*), verticillium wilt (*Verticilium alboatrum*), fusarium wilt, (*Fusarium oxysporum* f. sp. *vasinfactum*) ascochyta blight, (*Ascochyta gossyppii*) and boll rots (*Diplodia gossypina, Aspergillus niger*).

6.8.2 Important viral diseases dealt with

Virus diseases are relatively common, but only in a few instances they appear to limit crop production in China. Intensive efforts on insect control and such cultural practices as early removal of virus-infected plants and weed hosts in the vicinity of fields have had an important effect in reducing the prevalence of virus diseases. Careful selection of healthy plants for propagation for seed at each commune tends to favor continued selection for resistant material, as well as, to reduce the hazard of dissemination of viruses. Yellow dwarf, the most important, was in epidemic in 1973 AD in the Peking area and is endemic throughout northern and north-eastern China.

Basic studies were carried out at the Institute of Biochemistry in Shanghai on black streak stunt of rice, witches' broom of jujube, mulberry stunt, yellow shoot of citrus and rosette dwarf of wheat. Initial studies using electron microscopy indicate that mycoplasma-like organisms may be associated with the mulberry yellow dwarf disease and the citrus disease.

6.8.3 Fungi for food and medicinal purpose

Mushrooms continue to be a main and important component of the Chinese diet and one or more types of mushrooms may be incorporated as ingredients in some dishes served at most meals. In the Shanghai area, many communes are engaged in large-scale production of *Agaricus bisporus*. Some of the local mushroom production is apparently for markets abroad. ***Lentinus shiitake (L. edodes), Volvaria violacea*****,** and ***Auricularia judae*** were also under cultivation in the communes. The communes were growing ***Ganoderma lucidum*** and ***Tremella fuciformis*** as sources of drugs. Extracts of the former are used to reduce hypertension. It is grown in jars containing pig manure and other wastes. ***Tremella fuciformis*** is grown on logs of ***Pterocarya stenoptera***. Mushroom growing is a large industry in itself in the PRC and provides valuable supplemental incomes for many communes.

6.8.4 Quarantines and planting material

The Chinese insist upon pathogen-free seed at all levels including the central, provincial, county, and commune. This was made very clear in 1974 AD when several cargoes of US wheat were refused at the port of Shanghai because they were found to contain spores of the dwarf-bunt fungus. Some provinces including Kirin, also have a quarantine against wheat seed from fields affected by the take-all pathogen *Gaerinzannomyces- graminis* var. *tritici (=Ophiobolus graminis)*, which is not seedborne.

At the Kiangsu Academy of Agricultural Research in Nanking, a bacteriophage specific for *Xanthomonas oryzae* is being used to detect X. *oryzae* in seed and crop residues and as a possible means to prevent its growth in paddy water.

Large-scale seed potato production was originally restricted to northern provinces in Manchuria and inner Mongolia. It was not possible to maintain productivity when initial attempts were made to use potatoes grown in southern provinces for seed.

The Plant Inspection and Quarantine Laboratory in Shenzhen city is now one of the largest laboratories in this field in China (Xiang C.Y. et al., 2011 AD) Two national and regional key laboratories have been built, top ranking both in inspection capacity and routine inspection. In 2010 AD, more than 70,000 imported products were inspected and more than 30,000 pest-infected products were intercepted. By carrying out scientific research and cooperating domestically and abroad, the laboratory is leading the way in the development of standard methods for standard pest testing using morphology, molecular biology and immunology.

6.9 Development of plant pathology in Sri Lanka

The first Sri Lankan fungi to be recorded were *Peziza ceylonische* and *P. lembosa* described by **Houttyn** in 1783 AD. Later work by **Berkeley, Broome, Petch** and **Bisby** (1950 AD) has raised the number of described species to over 2,000 in about 640 genera. **T. Petch**, a British mycologist worked at the Royal Botanical Gardens in Peradeniya between 1900 AD and 1925 AD also documented various plant pathogens and other fungi.

There are detailed records of the serious damage caused by coffee rust (*Hemileia vastatrix*) in 1870 AD, which defoliated the plants, and virtually ended coffee production in the island, causing the British to change their drinking habits and tea became more popular (Daniel 1993 AD). The coffee rust epidemics led to the beginning of plant quarantine.

Plant quarantine began in 1869 AD, after the coffee rust disease wiped out nearly all the coffee plantations in Sri Lanka, and Indonesia banned import of both coffee and coffee sacks from Sri Lanka. **This was Asia's first plant quarantine regulation**. In Sri Lanka, British scientists in the Department of Agriculture at Peradeniya began to quarantine plants in 1880 AD onwards, and Sri Lanka became a centre for the identification of pests and diseases affecting crops, as countries in the region began sending material for identification. After the establishment of the Central Agricultural Research Institute (CARI) at Gannoruwa, Peradeniya, all plant quarantine activities were carried out there by the departments of Entomology and Plant pathology.

Certain groups of fungi have been studied more intensively than others, because of their prominence, or practical importance. The number of records in Uredinales are, *Uredo* (58 species), *Puccinia* (41 species), *Coleosporium* (3 species), *Uromyces* (19 species), *Hemileia* (1 species), *Melampsora* (4 species), *Pucciniastrum* (2 species), *Aecidium* (20 species), *Blastopota* (1 species), *Cerotelium* (1 species), *Cystospora* (1 species), *Diorchidium* (3 species), *Phragmidium* (4 species), *Ravenelia* (8 species), *Scopella* (1 species); making a total of 168 species (Berkeley and Broome, 1870 AD–1877 AD; Petch, 1908 AD–1948 AD; and Petch and Bisby, 1950 AD). In Ustilaginales, 25 species belonging to 7 genera and in Exobasidiales 2 species belonging to single genus were recorded.

Sri Lanka has a century long tradition of research into phytopathogens (Ainsworth, 1976 AD). Numerous handbooks (UNESCO: Man and the Biosphere (MAB) National Committee for Sri Lanka publications) on the fungi parasitic on plants, associated with insects, or found in soil were published in the 1970 AD onwards and 1980 AD onwards (Coomaraswamy 1979 AD, 1981a AD; Koomaraswamy and De Fonseka 1981 b AD; Koomaraswamy and Kumarasingham ,1988 AD) and several countrywide checklists have been published (Coomaraswamy, 1979 AD).

Plant pathology divisions in major research stations have been established to carry out research on the pathogens affecting the crops. They work on issues such as diversity, ecology and the management of diseases. With the rapid advancement of research in fungal pathogens, the older checklists and countrywide databases such as the diseases of cultivated plants in Ceylon by Abeygunawardhane (1969 AD). Fungi parasitic on the plants of Sri Lanka (Coomaraswamy, 1979 AD) quickly became outdated (Cai et al. 2011b AD; Ko Ko et al., 2011 AD).

6.10 Important diseases and noteworthy work in Asia

6.10.1 Bacterial blight of rice

Bacterial blight is one of the most destructive rice diseases in Asia and has historically been associated with major epidemics. It occurs in China, Korea, India, Indonesia, the Philippines, Sri Lanka, Myanmar, Laos, Taiwan, Thailand, and Vietnam. In the late 70s, epidemics due to bacterial blight were reported in India. The advent of rice varieties bearing genes with resistance to the disease has changed the perception about the disease. The incorporation of host-plant resistance genes in rice varieties, their adoption and deployment in the world's main rice-producing environments are probably one of the most significant evidences of the role of plant pathology in agricultural development. Bacterial blight nevertheless remains an important concern and

many countries will not endorse the release of new rice varieties unless they carry resistance to the disease. Whenever susceptible rice varieties are grown in environments that favour bacterial blight, very high yield losses, as much as over 70%, may be caused by bacterial blight. It is particularly serious in hybrid rice, and therefore, active breeding programme at national and commercial level have developed and released some hybrids that have resistance to the disease by International Rice Research Institute, Philippines.

6.10.2 Rice blast fungus: genomics and beyond

Y.H. Lee of Korea elucidated the molecular mechanisms of fungal pathogenesis and interactions between rice blast pathogen *Magnaporthe oryzae* and its host plant rice at the genomic level. In an attempt to understand the molecular mechanisms of rice blast, he had been taking both forward and reverse genetics approaches. Their researches were using reverse genetics approach focus on identifying and characterising the genes involved in signal transduction pathways leading to appressorium formation, genes encoding transcription factors, and genes that are required for post-penetration stages. For forward genetics studies, they carried out a large-scale insertional mutagenesis of the *M. oryzae* strain KJ201 via *Agrobacterium tumefaciens*-mediated transformation, generating over 25,000 mutants. He also, developed high throughput phenotype screening system which enables rapid and robust assay of mutant phenotypes. Those mutants are stored and maintained in the Center for Fungal Genetic Resources.

R. Terauchi of Japan analysed the whole genome of rice – *Magnaporthe* interactions. The studies on whole genome sequence (WGS)-based gene isolation methodologies reported the interactions between AVR-Pik and Pik as well as AVR-Pii and Pii genes.

6.10.3 Rice tungro

Many farmers in south and south-east Asia describe rice tungro disease as a cancer disease because of the severe damage it causes and the difficulty in controlling it. The most important of the 14 rice viral diseases, tungro, was first recognised as a leafhopper-transmitted virus disease in 1963 AD. However, tungro, which means degenerated growth in a Filipino dialect, has a much longer history. It is almost certain that tungro was responsible for a disease outbreak that occurred in 1859 AD in Indonesia, which was referred to at the time, as *mentek*. In the past, a variety of names have been given to tungro, including *accep na pula* in the Philippines, *penyakit merah* in Malaysia, and yelloworange leaf in Thailand. During 1960–1970 AD, a series of large-scale outbreaks of tungro were recorded in India, Thailand, Indonesia, Malaysia, and Philippines. Rice farmers and poor consumers suffered severe hardships

during these outbreaks, and the threat to food security in the regions affected gave the disease a high political profile.

6.10.4 Citrus greening (*huanglongbing*)

1919 AD	First reported in southern China
1921 AD	First report of disease in the Philippines, but it was thought to be related to zinc deficiency
1928 AD	A disease under the names, yellow shoot or greening depending on region, was observed in South Africa
1941 AD–1955 AD	Most extensive work on greening in southern China was conducted
1956 AD	Lin Kung Hsiang (researcher from China) concluded that greening is a graft transmissible infectious disease, not related to physiological disorders (e.g. nutrient deficiencies, water logging, etc.) or soil borne diseases
1960 AD onward's	HLB first appeared in Thailand
1966 AD	Philippine and Indian researchers recognised the similiarities between the mottle leaf or citrus die-back disease and HLB in China and Taiwan
1967 AD	Philippine researchers demonstrated mottle leaf or citrus die-back could be transmitted by the Asian citrus psyllid, *Diaphorina citri*
1995 AD	The official name of the disease became huanglongbing (HLB) at the International Organisation of Citrus Virologists (IOCV) at the 13th conference of the Organisation in Fuzhou (Fujiam, China)

6.10.5 Cotton leaf curl (CLCV)

This disease is also called leaf crinkle. A virus causing leaf curl of cotton was first recorded in Nigeria in 1912 AD. In Pakistan, this disease was first time recorded in 1967 AD at Multan Punjab on some cotton plants. It was considered a minor disease until 1987 AD, but in 1991 AD–1992 AD, it became severe and since 1992 AD–1993 AD caused a huge production and monetary loss to the nation. In Sindh, this disease was first reported during 1996 AD at Ubauro, district Ghotki, and it reached up to New Saedabad, district Hyderabad, during 1999 AD–2000 AD.

Intensively studied cotton leaf curl virus (CLCuV) in all aspects *viz*.; evaluation of Gossypium species for resistance, disease severity in relation to environmental conditions, screening of cotton mutants for resistance, influence of plant age, whitefly population and cultivar resistance on infection of cotton plants, deterioration of cotton fiber characteristics caused by cotton leaf curl disease were carried out by Akhtar K.P. (2001 AD) in Pakistan.

7

Historical epidemics and important plant diseases around the world and their impact on civilisation

7.1 Irish potato famine

The potato, which was introduced in Europe from south and central America around 1570 AD, was a well-established crop in Ireland by 1800 AD and was a main source of food. Irish farmers were dependent on potatoes for their sustenance and survival. Lacking proper warehouses, the farmers used to store their potato tubers for the winter in shallow ditches in the ground. Periodically, they would open up part of the ditch and remove as many potatoes as they thought they would need for the next few weeks. The potatoes grew well for many years, free of any serious problems.

Most of the growing season of 1845 AD in Ireland was quite favourable for the growth of potato plants and for the formation of tubers. Everything looked as though there would be an excellent yield of potatoes everywhere that year. Then, the weather over northern Europe and Ireland became cloudy, wetter, and cooler and stayed that way for several weeks. The potato crop, which until then looked so promising, began to show blighted leaves and shoots, and whole potato plants became blighted and died. In just a few weeks, the potato fields in northern Europe and in Ireland became masses of blighted and rotting vegetation. The farmers were surprised and worried, especially when they noticed that many of the potatoes still in the ground were rotten and others had rotting areas on their surface. They did what they could to dig up the healthy looking potatoes from the affected fields and put them in the ditches to hold them through the winter. The farmer's worry became horror when later in the fall and winter they began opening the ditches and looking for the potatoes they had put in there at harvest. At last, instead of potatoes they found only masses of rotting tubers, totally unfit for consumption by humans or animals. The dependence of Irish farmers on potatoes alone meant that they had nothing else to eat and neither did any of their neighbours. Hunger was quickly followed by starvation, which resulted in the death of many Irish people. The famine was exacerbated by the political situation between England and Ireland. The British refused to intervene and help the starving Irish with food for several months after the blight destroyed their potatoes. Eventually,

by February of the next year (1846 AD), food, in the form of corn from U.S., began to be imported and made available to the starving poor who paid for it by working on various government construction projects.

Unfortunately, the weather in 1846 AD was again cool and wet, favouring the potato blight, which again spread widely and destroyed the potato plants and tubers. Hunger, dysentery, and typhus spread among the farmers again, and many of the survivors emigrated to North America. **It is estimated that one and a half million Irish died from hunger, and about as many left Ireland, emigrating mostly to U.S.**

The cause of the destruction of the potato plants and of the rotting of the potato tubers was, of course, unknown and a mystery to all. The farmers and other simple folk believed it to have been brought about by *the little people*, or by the devil himself whom they tried to exorcise and chase away by sprinkling holy water in the fields, by locomotives travelling the countryside at devilish speeds of up to 20 miles per hour and discharging electricity harmful to crops they went by, or to have been sent by God as punishment for some unspecified crimes they had committed. The more educated doctors and clergy were so convinced of the truth of the theory of spontaneous generation that even when they saw the mildew fungus growth on affected leaves and on some stems and tubers, they thought that this growth was produced by the dying plant as a result of the rotting rather than the cause of the death and rotting of the plant.

Some of the educated people, however, began to have second thoughts about the situation. Dr. J. Lindley, a professor of botany in London, proposed incorrectly that the plants, during the rains, over absorbed water through their roots and because they could not get rid of the excess water, their tissues became swollen and rotten. The Reverend Dr. Miles Berkeley, however, noticed that the mould covering potato plant about to rot was a fungus (oomycete) similar but not identical to a fungus he observed on a sick onion. The fungus on potato, however, was identical to a fungus recovered from sick potato plants in northern Europe. Berkeley concluded that this fungus was the cause of the potato blight, but when he proposed it in a letter to a newspaper, it was considered as an incredible and bizarre theory unsupported by facts. The puzzle of what caused blight of potato continued unanswered for 16 years after the 1845 AD destruction of potatoes by the blight.

Finally, in 1861 AD, Anton De Bary did a simple experiment that proved that the potato blight was caused by a fungus. De Bary simply planted two sets of healthy potatoes, one of which he dusted with spores of the fungus collected from blighted potato plants. When the tubers germinated and began to produce potato plants, the healthy tubers produced healthy plants, whereas the healthy tubers dusted with the spores of the fungus produced plants that became blighted and died. No matter how many times De Bary repeated the experiment, only tubers treated with the fungus became infected and produced

plants that became infected. The fungus which we know now, asoomycete was named *Phytophthora infestans* i.e. infectious plant destroyer as phyto = plant, phthora = destruction, infestans = infectious and was the cause of the potato blight.

De Bary also showed that the fungus did not just reappear from nowhere the following growing season but instead survived the winter in partially infected potato tubers in the field or during storage. In the spring, the fungus infected young plants coming out from these partially rotten tubers, produced new spores on these plants, and the spores then spread to other cultivated potato plants that were infected and ultimately getting killed. With this experiment, De Bary actually disproved the "**Theory of Spontaneous Generation**" and supported the **germ theory of disease** in plants.

7.1.1 Impact on society and government

- This devastating disease not only resulted in famine, but was also responsible for the emigration of 1.5 million people from Ireland to U.S. and Canada.
- Late blight continues to threaten potato production in many regions of the U.S. as new strains of the fungus develop.

7.2 Wheat rust epidemics

Wheat stem rust has been an on-going problem dating back to Aristotle's time (384 BC–322 BC). An early ancient practice by the Romans was one where they would sacrifice red animals such as foxes, dogs, and cows to Robigo or Robigus, the rust God. They would perform this ritual in the spring during a festival known as the Robigalia in the hope that the wheat crop will be spared from the destruction caused by the rust. Weather records from that time have been observed and it has been speculated that **the fall of the Roman Empire was due to a string of rainy seasons in which the rust would have been harsher, resulting in reduced wheat harvests.**

Laws banning barberry were established in 1660 AD in Rouen, France. This was due to the fact that European farmers noticed a correlation between barberry and stem rust epidemics in wheat. The law banned the planting of barberry near wheat fields and was the first of its kind before the parasitic nature of stem rust was discovered in the 1700 AD onwards. Two Italian scientists Fontana and Tozzetti first explained the stem rust fungus in wheat in 1767 AD. Thirty years later it received its name, *Puccinia graminis* by Persoon and in 1854 AD brothers Louis René and Charles Tulasne discovered the characteristic five-spore stage that is known to some as stem rust species. The brothers were also able to make a connection between the red (urediniospore)

and black (teliospore) spores as different stages within the same organism, but the rest of the stages remained unknown.

Due to the useful nature of both barberry and wheat plants, they were eventually brought to the northern America by European colonists. Barberry was used for many things like making wine and jams from the berries to tool handles from the wood. Ultimately, as they did in Europe, the colonists began to notice a relationship between barberry and stem rust epidemics in wheat. Laws were enacted in many New England colonies, but as the farmers moved west, the problem with the stem rust moved with them and began to spread to many areas, creating a devastating epidemic in 1916 AD. It wasn't until two years later in 1918 AD that the U.S. created a program to eliminate barberry. The program was supported by state and federal entities and was prompted by the looming fear of scarcity of food supplies during the war. The war against barberries was waged and state called upon the citizens through radio and newspaper advertisements, pamphlets, and fair booths asking for help from all in an attempt to get rid of the barberry bushes of their existence. Later, in 1975 AD–1980 AD, the program was re-established back to state jurisdiction. Once this happened, a federal quarantine was established against the sale of stem rust susceptible barberry in those states that were part of the program. A barberry testing program was created to ensure that only the species of barberry and other variations of plants that are immune to stem rust will be grown in the quarantine area.

The first note about the existence of stem rust in pre-biblical times in Israel is published by Kislev (1982 AD). He found germinating urediniospores, uredinia, and hyphae on fragments of lemmas in a storage jar from the late bronze age (ca. 3300 BC). The specimens were charred but well preserved.

Rust has been recorded on wheat in India for centuries with documented evidence from 1786 AD, 1805 AD, 1828 AD–1829 AD, 1831AD–1832 AD, 1879 AD, 1887 AD and 1907 AD (Nagarajan and Joshi, 1975 AD) and identified 17 notable appearances or epidemics of wheat rusts in India between 1786 AD and 1956 AD which were responsible for insufficient food supply to the Indian citizens and their migration towards cities.

In 1932 AD, a severe epidemic of stem rust devastated wheat crops in many east European countries. The epidemic began in Bulgaria, but spread throughout eastern and northern Europe (Zadoks, 2008 AD). Its impact was most severe in Russia. In 1977 AD and 1978 AD, a stem rust epidemic affected the northern regions of Pakistan, particularly northern Punjab and north-western Frontier Province. In 1976 AD–1977 AD, a leaf rust epidemic developed in north-western Mexico where more than 70% of the country's wheat crop was produced (Hanson et al., 1982 AD).

An epidemic of stem rust on wheat caused by race TTKSK (e.g. isolate Ug99) is currently spreading across Africa, Asia and the Middle East and is causing major concern as large number of people are dependent on wheat for sustenance. The strain was named after the country where it was identified (Uganda) and the year of its discovery (1999 AD). It spread to Kenya, then to Ethiopia, Sudan and Yemen, and is becoming more virulent as it spreads. Scientists are working on breeding strains of wheat that are resistant to Ug99. However, wheat is grown in a broad range of environments. This means that breeding programs would have extensive work left to get resistant into regionally adapted germplasm even after resistance is identified.

7.2.1 Impact on society and government

According to Jim Peterson, professor of wheat breeding and genetics at Oregon State University, "Stem rust destroyed more than 20% of U.S. wheat crops several times between 1917 AD and 1935 AD, and losses reached 9% twice in 1950 AD onwards," with the last U.S. outbreak in 1962 AD destroying 5.2% of the crop.

Losses in north Dakota, during the severe epidemics of 1935 AD and 1954 AD, were estimated as $356 million and $260 million, respectively, based on wheat prices in late 1995 AD.

The Indian epidemics during pre- and post-independent era were responsible for food shortage, hunger and immigration of people toward cities.

7.3 The great Bengal Famine (Brown Spot of Rice)

The great Bengal famine of 1943 AD was a large famine in Bengal, a state in British-ruled India, claiming the lives of at least three and a half million people. Bengal famine of 1943 AD was due to the loss of rice crop due to brown spot disease of rice caused by the fungal pathogen *Cochliobolus* (*Helminthosporium*). There was an epidemic of the disease on rice crop in Bengal. This famine was exclerated by crop failures in 1942 AD combined with continued exports of rice from Bengal to other regions of India and elsewhere in the British Empire, the destruction of most of the boats used for transportation in the province and the refusal of the government of Prime Minister Winston Churchill to allow emergency food supplies of wheat offered free by the U.S. and Canada to be shipped to the starving province.

7.3.1 Politics of famine

In the year prior to 1942 AD, when Japan seized Burma, (an important rice exporter) the British bought up massive amounts of rice but hoarded it. Dr.

Gideon Polya, an Australian biochemist, has called the Bengal famine as a man-made *holocaust*. "The British brought an unsympathetic and ruthless economic agenda to India," he wrote. Polya further noted that the "loss of rice from Burma and ineffective government controls on hoarding and profiteering led inevitably to enormous price rises." Thus, it can be estimated that the price of rice in Dacca (East Bengal) increased about four-fold in the period from March to October 1943 AD. Bengalis who purchased food (e.g. landless labourers) suffered immensely. Thus, it is estimated that about 30% of one particular labour class died in the famine. Many observers in both modern India and Great Britain blame Winston Churchill, Britain's inspiring wartime leader at the time, for the devastation wrought by the famine.

In 2010 AD, Bengali author Madhusree Mukherjee wrote a book about the famine called *Churchill's Secret War*, in which she explicitly blamed Churchill for worsening the starvation in Bengal by ordering the diversion of food away from Indians and towards British troops around the world. Mukherjee's book described how wheat from Australia (which could have been delivered to starving Indians) was instead transported to British troops in the Mediterranean and the Balkans. Even worse, British colonial authorities (again under Churchill's leadership) actually turned down offers of food from Canada and the U.S. "If it was someone else other than Churchill, I believe relief would have been sent, and, if it wasn't for the war, the famine wouldn't have occurred at all," Mukherjee told Inter Press Service. "Churchill's attitude toward India was quite extreme, and he hated Indians, mainly because he knew India couldn't be held for very long. One can't escape the really powerful, racist things that he was saying. It certainly was possible to send relief but for Churchill and the War Cabinet that were hoarding grain for use after the war." Churchill's hostility toward Indians has long been documented. Reportedly, when he first received a telegram from the British colonial authorities in New Delhi about the rising toll of famine deaths in Bengal, his reaction was simply that he regretted that nationalist leader Mahatma Gandhi was not one of the victims. Later at a War Cabinet meeting, Churchill blamed the Indians themselves for the famine, saying that they *breed like rabbits*. His attitude towards Indians was made crystal clear when he told Secretary of State for India Leopold Amery: "I hate Indians. They are a beastly people with a beastly religion."

British soldiers fighting in east Africa and the Middle East were relying on Bengali rice exports and London thought it expedient and less expensive to feed soldiers with rice than to keep Bengali civilians from starving to death. They were brown, non-Christian and colonial no doubt had something to do with this calculation. In any event, "Indians did not vote in British elections."

The government (apparently not having learned from the example of a very similar situation during the Irish Potato Famine) opposed an urgent request

from Leopold Amery and Archibald Wavell (the Indian Secretary of State and the Viceroy of India, respectively) to stop exports of food from Bengal so that it might be used for famine relief. Churchill dismissed these in a fashion that Amery regarded as "Hitler-like," by asking why, if the famine was so horrible, Gandhi had not yet died of starvation. George Orwell echoed Amery when he said: "The way the British government is now behaving in India upsets me more than a military defeat."

The British Prime Minister David Cameron expressed regret for the Jallianwala Bagh massacre of 1919 AD in Amritsar (in which at least 400 unarmed Indian men, women and children were massacred by British soldiers), but he omitted any reference to Britain's role in a far greater tragedy of colonial India: the Bengal famine of 1943 AD where 70years ago, at least 3 million people died from starvation and malnutrition during a famine in the Indian province of Bengal – a partly man-made disaster that has been largely forgotten by the world beyond north-eastern India. A complex confluence of malign factors led to the catastrophe, which occurred with the world at war, the Japanese occupation of neighbouring Burma and damage to the local rice crop due to tidal waves and a fungal disease epidemic, as Indian parliamentary member and leading agricultural scientist M.S. Swaminathan cited in the Hindu newspaper. Swaminathan also blamed panic purchase and hoarding by the rich, failure of governance, particularly in relation to the equitable distribution of the available food grains, disruption of communication due to World War II and the indifference of the then U.K. government to the plight of the starving people of undivided Bengal. But while famines were not uncommon in India throughout history, largely because of periodic droughts or monsoons, the tragedy in Bengal had the unmistakable hand of man in it, making it an even greater calamity of recent global history.

7.3.2 Impact on society and government

- India experienced the second Bengal famine in1943 AD (the first was in 1769 AD–1770 AD). The Great Bengal famine of 1943 AD killed three and half million people in Bengal (part of which is now in Bangladesh and eastern India) and the streets and highways were littered with their bodies.
- The British colonial government imposed wartime censorship on the Bengal famine of 1943 AD to avoid pressure to divert resources from the war effort. Some estimates of death put the figure above 4 million people.
- The distressing famine in Bengal in 1943 AD–1944 AD caused a gasp of astonishment as well as of horror in India and in the U.K. The worst social and economic disasters, such as the Bengal famine of 1943 AD,

or the dislocations during China's *Great Leap Forward* (1951 AD–1953 AD), have occurred in the absence of democratic safeguards.

The famine of 1943 AD and chronic food shortages after independence in 1947 AD and into the 1950 AD onwards led the Indian political leaders and government to setup India's agricultural policies. The response of the Indian government was to achieve greater security in food supply, especially food grains. The set of policy instruments employed for these efforts, and the resulting expansion in Indian agricultural production achieved the Green Revolution.

7.4 Coffee rust

Coffee is a crop of the tropics surpassed only by oil in its value as a world commodity. For centuries, it has been a significant import crop in Europe and economically important to the European countries that ruled tropical colonies. It remains an important crop to the independent nations created from those colonies.

Coffee became a popular drink in Europe in the 1600 AD onwards when contaminated drinking water limited people to fermented beverages or those made with boiled water, such as tea or coffee. Coffee houses were major social centres in England from the year 1650 AD onwards. The Dutch were the first major European coffee importers, transporting coffee.

During Napoleon's time, much of the coffee-producing area was lost by the Dutch to the English. In 1825 AD, the British began development of their property in Ceylon (now Sri Lanka), and every suitable piece of land was planted with coffee plantations. By 1870 AD, Ceylon was the world's greatest producer of coffee. In subsequent year, a disease known as coffee rust appeared on this plantation and the devastation of plant and losses started occurring.

The fungal coffee rust pathogen *Hemileia vastatrix* probably arose in southern Ethiopia, the origin of the coffee plant itself and moved with the planting material without much notice until it started appearing severely in plants. A single tiny rust pustule on a coffee tree leaf can produce 1,50,000 spores, and a single leaf can contain hundreds of pustules.

When the coffee rust fungus, reached Ceylon in 1875 AD, nearly 4,00,000 acres (1,60,000 ha) were covered with coffee trees. No effective chemical fungicides were available to protect the foliage, so the fungus was able to colonise the leaves until nearly all the trees were defoliated. The spores produced on the leaves are quite resistant to desiccation, unlike the sporangia of *P. infestans*, and are capable of long-distance movement in a viable state. They easily moved through the acres of coffee trees, feasting on the banquet

prepared by unsuspecting plantation owners. In 1870 AD, Ceylon was exporting 100 million pounds (45 million kg) of coffee a year. By 1889 AD, production was down to 5 million pounds (2.3 million kg). In <20 years, many coffee plantations were destroyed, and production had essentially ceased.

7.4.1 Impact on society and government

- The coffee rust disease caused by *Hemileia vastatrix,* appeared in coffee plantations in Ceylon (presently Sri Lanka) in the 1860 AD onwards and subsequently destroyed the coffee industry in that nation.
- As a part of the British Empire, Ceylon was one of the primary sources of coffee for England, a nation whose citizenry drank coffee as their favourite boiled beverage.
- However, as a result of the coffee rust epidemic, tea was planted in its place and coffee became very difficult to obtain. Thus, the English had to modify their drinking habits and as a result of a plant disease, England became a nation of tea drinkers.
- 40,000 workers lost wages and succumbed to poverty and hunger in region.
- US $500 million in losses in a year.

7.5 Cassava mosaic disease (CMD)

The cassava (*Manihot esculenta*) was a staple food in sub-Saharan Africa. Cassava mosaic disease (CMD) spread from its source in Tanzania during 1894 AD and occurred throughout Africa wherever cassava was grown and by 1987 AD became the most important enemy of cassava production. The disease is caused by whitefly transmitted begomo viruses, including African cassava mosaic virus (ACMV) in west of the Rift Valley and east African cassava mosaic virus (EACMV) in east of the Rift Valley.

In 1988 AD, an epidemic of a highly virulent variant of EACMV (EACMV-Ug; arising from the inter-specific recombination of EACMV and ACMV) began in Uganda and spread throughout east and central Africa, causing crop losses on a scale that required international intervention to prevent widespread famine and starvation.

7.5.1 Impact on society and government

- Across Africa, more than200 million people depend on cassava as their staple food.

- In Uganda, cassava production plummeted from a high of 3.5 million tonnes to 2.25 million tonnes during 1989 AD.
- During 1996 AD as a result of the emergence of EACMV-Ug, the losses were estimated at $60 million per year (between 1992 AD and 1997 AD).
- In the north and east of Uganda, the epidemic led to famine and food insecurity resulting from this disease and still threatens regions of east, central and west Africa.
- Girls were sold into slavery or put to early marriage due to cassava starvation and migration occurred.

7.6 Chestnut blight

In U.S., a broad band of land of several hundred miles in width and extending from the bottom of the states of Georgia and Mississippi to the top of Maine and Michigan and into Ontario, Canada – the most common trees in the forests were the majestic American chestnuts. They provided timber and chestnuts fruits serving as a source of food for humans and wildlife; while the trees served as a habitat for wildlife. Both timber and chestnuts provided a source of income for the local people. The trees had been there apparently forever and looked like they would also last forever. Then something seemingly minor happened in 1904 AD, as the leaves of a few branches of large chestnut trees and a few young trees in the New York zoo began to turn brown and die. Before anyone could figure out what was happening, many more young trees and branches of older ones died, giving the trees a blighted appearance.

From there, chestnut blight spread rapidly through eastern North America so that by the 1920 AD onwards the blight could be found in the entire natural range of the American chestnut tree. By now, scientists in general, and plant pathologists in particular, were quite adept at identifying most causes of plant disease, and chestnut blight was quite easy to diagnose.

It was soon shown that chestnut blight is caused by a fungus, *Cryphonectria parasitica*. By the late 1920 AD onwards, about three and a half billion American chestnut trees had become infected. Infected trees and branches would produce sprouts from areas below the canker and the sprouts would grow without becoming infected until they were 2–4 inches in diameter. At some point and before they produced any fruit, the fungus would attack and kill them too. That way, although the huge original chestnut trees kept producing new sprouts year after year for many years, their killing by the ever-present fungus finally exhausted the trees and they finally died in their roots. Hardly any tree escaped, making chestnuts the first tree to approach extinction in modern times because of a plant disease caused by a fungus.

7.6.1 Impact on society and government

- Three and half million chestnut trees died leaving large impact on human well-being, economy or biodiversity.
- Regional elimination of a co-dominant species that sent ripple effect throughout ecosystems.
- The American chestnut was an important component of the southern Appalachians before its removal by chestnut blight.

7.7 Dutch elm disease

The disease seems to have appeared first in north-western Europe at the end of the First World War. It spread rapidly through Europe, killing a significant proportion of the elm population in many countries. In Holland, 30% of the elms trees died between 1930 AD and 1940 AD (Went, 1954 AD).

In Britain the disease was first recorded in 1927 AD, though it may have arrived a little earlier. Initially the disease caused widespread death of elms, but after 1937 AD, it began to decline both in terms of the numbers of affected trees and the severity of disease symptoms. Gradually it came to be regarded as an endemic problem of no great importance. In 1960 AD, Peace estimated that only 10–20% of the original elm population had been killed by the disease.

The disease reached the U.S. in the late 1920 AD onwards on elm logs imported from France (Beattie, 1933 AD). Since then it spread steadily northwards and westwards causing spectacular losses in the highly susceptible American elm population. American elm grows to be a big, gracefully shaped and beautiful vase-like tree that exists naturally mixed with other hardwoods throughout eastern North American forests and extending into the Great Plains. The elm was adopted by early homeowners and town settlers in North America and beautified many a streets by being planted in rows on both sides of the street.

In 1930 AD, a few elm trees in Cleveland, Ohio, began to show wilting, yellowing and then browning of the leaves of some branches. The wilted, brown leaves later fell off and the branch appeared defoliated and dead. More branches showed similar symptoms later that year or the following year and the entire elm tree usually died within one or a few years. Trees with similar symptoms were soon observed in some east coast states. The disease became known as Dutch elm disease because, its first report was from Dutch country (Holland) in 1921 AD, received all the publicity although it had been reported from France in 1917 AD. The Dutch elm disease spread rapidly in North America, crossing the Mississippi River by 1956 AD and reaching the Pacific

coast states by 1973 AD. In its path, the disease has killed the vast majority of yard, park, and street trees, although quite a few trees in their natural forest habitat are still free from the disease.

It first appeared in Canada in the early 1940 AD onwards probably as a result of a separate introduction from Europe (McCullum and Stewart, 1958 AD).In 1967 AD the Forestry Commission received the first reports of disease in the areas now regarded as the original centres of the present epidemic.

Dutch elm disease is caused by the fungus *Ophiostoma ulmi*. The fungus is carried to healthy elm trees by two elm bark beetles that lay their eggs in weakened or dead elm trees or logs, often those killed by the Dutch elm disease. The eggs hatch and produce larvae that form tunnels, and if the trees or logs are infected with the disease, the fungus grows into and produces spores in the tunnels. The adult beetles then emerge covered with spores of the fungus and look for vigorous young elm branches to feed on. While they are feeding and causing hardly any damage to the elm trees, they deposit spores of the fungus in the feeding wound. The spores germinate and produce mycelium and more spores, both of which spread and multiply in the xylem vessels of the tree and cause the vessels to become clogged. Water and minerals cannot move from the root to the shoots and leaves beyond the point of clogging. The shoots and leaves subsequently wilt and die and eventually the entire tree die.

7.7.1 Impact on society and government

- The consequences of Dutch elm disease pandemics are profound with second epidemic (which began during the 1940 AD onwards) resulting in the loss of hundreds of millions of elms and causing closed canopy forests to become open habitat, leading to changes in bird community composition.
- The deaths of mature elms resulted in increased mortality rates of sympatric trees such as paper birch (*Betula papyrifera*), as they became more exposed to storm damage.
- This disease induced effective extinction, had serious consequences for the ecology of the region, aggregations of dead wood from affected trees have environmental repercussions even decades after the initial epidemic such as influencing the structure and function of Appalachian streams.

7.8 Lethal yellowing of coconut palms

Lethal yellowing like symptoms on dying palm trees had been included in brief reports from the Cayman Islands, Cuba, and Jamaica even during the 19th century.

In 1955 AD, coconut palm trees in the key west islands of Florida were noticed to drop their coconuts prematurely. Then, the next inflorescence had blackened tips and set no fruit. Soon, first the lower older leaves and then the next younger leaves turned yellow and then brown and died. Finally all the leaves and the vegetative bud died and the entire top of the tree fell off leaving the tall palm trunk looking like a telephone pole.

The lethal yellowing disease was first found in mainland Florida in 1971 AD and killed 15,000 coconut palm trees by1973 AD; 40,000 by 1974 AD, and, by 1975 AD, 75% of the coconut palm trees in Dade County were dead or dying from the disease.

The lethal yellowing disease is caused by a phytoplasma. The phytoplasma lives and multiplies in the phloem sieve elements of palm trees and causes the lethal yellowing symptoms by clogging some of the sieve tubes and interfering with the transportation of organic foodstuffs out of the leaves and also by producing biologically active substances that are toxic and cause the yellowing and death of the leaves, inflorescence, and vegetative bud of coconut trees. The phytoplasma is spread from diseased to healthy trees by a small plant hopper. The plant hopper sucks up juice from the phloem of infected palm trees acquires the deadly phytoplasma and when the plant hopper lands and feeds on a healthy palm tree, it transmits some of the phytoplasmas to the phloem sieve element of healthy tree. Once in the phloem cells, the phytoplasmas multiply and move throughout much of the phloem of the tree and cause the tree to develop the symptoms of lethal yellowing and tree dies.

7.8.1 Impact on society and government

- Tremendous losses of palm trees occurred in many countries. For example, in Jamaica, of six million trees counted in 1961 AD, 90% had been killed by lethal yellowing by 1981 AD. Thousands of hectares of palm trees were killed in Mexico and also in Tanzania; more than a million coconut palm trees were killed in Ghana and more than 60,000, (about 50% of the palm) trees in Togo, were killed by lethal yellowing by 1964 AD.
- The coconut being a portable source of food, water, fuel, and construction materials, played a fundamental role in human migrations and the development of civilisation across the humid tropics.

7.9 Oak wilts and sudden death

Oaks have been killed for decades by oak wilt caused by the fungus *Ceratocystis fagacearum*, but its spread and development are slower than the Dutch elm

disease of elm. At the same time, the oak population is larger and distributed more widely compared to elm. It was first recognised as an important disease in 1944 AD in Wisconsin where, in localised areas (<100 acres (40.4 ha)), over half the oaks have been killed. Surveys in eight Wisconsin counties showed that about 11% of the annual growth increase of oak forests was off-set by mortality caused by oak wilt.

The causal agent, *Ceratocystis fagacearum*, is found only within the borders of U.S. Recent evidence suggests that the pathogen was introduced into the U.S., possibly from Central or South America, or Mexico. The disease currently affects much of the eastern and central U.S., from Virginia to Minnesota to Arkansas, with pockets of infection as far south-westward as Austin, Texas. One area of infection has recently been discovered as far north-eastward as Glenville, New York. It is particularly common in the mid-west.

The disease occurs in the north-eastern U.S. but extends as far south as Texas in Dallas in 1961 AD. The progression of these epidemics is hard to predict, but the loss of thousands of oak trees is certain.

Symptoms vary by tree species but generally consist of leaf discoloration, wilt, defoliation, and death. The fungus is spread from diseased to healthy trees by insect vectors or via connections between tree roots. Oak wilt is an important disease of oak for reduced timber production.

7.9.1 Impact on society and government

- Led to the death of tens of thousands of oak trees.
- In urban areas where susceptible oaks are abundant, the impact on property or other social values has been significant.
- In central Texas, for instance, oak wilt has caused considerable decline in urban and rural property values through landscape degradation, shade privation and resulting decline in property values.
- The Texas Forest Service estimated in 1990 AD that over 10,000 trees worth millions of dollars were lost in the City of Austin alone (Davies, 1992 AD).

7.10 Karnal bunt of wheat and rye

Karnal bunt is a fungal disease of wheat, rye and triticale caused by *Tilletia (Neovossia) indica*.

The first report of a new bunt disease in wheat came from the region of Faizalabad (Pakistan) in 1909 AD. Karnal bunt was first discovered in 1930 AD near the town Karnal in north-west India (Mitra, 1931 AD). Since then,

it has been identified in all the major wheat producing regions of India, Iraq, Nepal, Pakistan and Afghanistan. It has also been found in seed exported from Syria and Lebanon, and in the late 1960 AD onwards it was introduced into Mexico. The first report of Karnal bunt from a non-Asian country came from Mexico in 1972 AD, where the disease has been reported from localised areas (5,00,000 ha) within the state of Sonora (EPPO, 1991 AD) and from the states of Sinaloa and Baja California Sur.

In U.S., the disease was first detected in 1996 AD in Arizona, and thereafter in southern California and in Texas, where it has spread to additional areas in 2001 AD. It was found in South Africa during December 2000 AD in the Herbert district near Douglas in the Northern Cape.

Within India the pathogen spread and can now be considered widespread in northern and central India (in regions where low winter temperatures and high humidity prevail, viz; Delhi, Uttar Pradesh, Haryana, Punjab, Himachal Pradesh, Rajasthan, Madhya Pradesh, Jammu and Kashmir, West Bengal and Gujarat (Singh et al., 1985 AD).

Karnal bunt is usually noticed first when broken and/or partially smutted kernels are seen in threshed grain. However, symptoms are present at the soft-dough stage in the form of blackened areas surrounding the base of the grain, which extend upward along the suture to varying degrees. Unless the disease is severe, only a few florets per spike are affected and diseased spikes are not conspicuous because the glumes are not distorted by infected kernels. In severely infected spikes, however, the glumes may spread apart near maturity, exposing the bunted grains. Infected grains are irregularly distributed in the spike; some are completely infected, but most are partially infected (hence the alternate common name of the disease, partial bunt). The weight of infected grains is directly related to the severity of infection; as the degree of infection increases, the weight of the grains correspondingly decreases. Karnal bunt is not toxic to humans, but wheat grain containing more than 3% bunted kernels is generally considered unfit for human consumption. Infection by *T. indica* can affect the appearance and smell of grain products. Bunted grain smells like rotting fish due to the production of trimethylamine.

While diseased seeds often retain a partial seed coat, the embryo and part of the endosperm can be converted to masses of small black spores, which emit a fishy odour (due to the presence of trimethylamine) similar to common bunt. Partially bunted seeds often retain their capacity to germinate, and grains with trace-to-moderately-low infections may produce healthy plants. Recent interest in the disease has surrounded the potential for its use as a biological weapon and exports of wheat from many regions with Karnal bunt have been banned, leading to severe economic loss for affected countries.

7.10.1 Impact on society and government

- India yielded total losses of ~0.5% but up to 89% of kernels infected in certain areas, with yield losses from 20% to 40% in highly susceptible varieties with poor quality of wheat unsuitable for human consumption.
- Most importantly, Karnal bunt has potentially serious phyto sanitary implications for wheat production and agricultural trade in South Africa. Wheat consignments having Karnal bunt disease is reported to return to the exporting countries causing great losses in foreign revenue.

7.11 Citrus canker

It is a bacterial disease, caused by plant pathogenic bacterium *Xanthomonas axonopodis* pathovar. *citri* which causes necrotic cankerous lesions on fruit, leaves, and twigs. Losses mainly caused are due to reduced fruit quality, due to cankerous lesions on fruit and quantity and premature fruit drop. The lesions that first appear are small, slightly raised, round, and with light green spots. Later, they become grayish white, rupture, and appear corky with brown, sunken centers. The margins of the lesions are often surrounded by a yellowish halo. The size of the lesions varies from 1 to 9 mm in diameter on leaves and up to 1 cm in diameter or length on fruits and twigs. Severe infections of leaves, twigs, and branches debilitate the tree, while severely infected fruit appear scabbed and deformed.

In U.S., citrus canker was introduced into Florida in 1912 AD with infected nursery seedlings from Japan. The disease is endemic in Japan and south-east Asia from where it has spread to all other citrus-producing continents except Europe. It took 20 years for eradication of citrus canker in Florida by burning of more than a quarter million fruit bearing trees and more than three million nursery trees, many millions of dollars in expenses, and untold inconvenience and heartaches before citrus canker was eradicated. It took more than20 more years (until 1949 AD) to eliminate it entirely from U.S. Unfortunately, a bacterial leaf spot resembling that caused by the citrus canker bacterium appeared in Florida in August 1984 AD and was assumed to be citrus canker. A new series of eradicating measures went into effect immediately, resulting in the destruction of at least 20 million nurseries and young orchard trees through 1990 AD. However, in 1986 AD, the real citrus canker (Asiatic canker or canker A) was also found in Florida, and the eradications continued in areas where canker was present until 1992 AD. After no citrus canker was found for 2 years, Florida was declared free of citrus canker in early 1994 AD, and all regulations to control the eradication were suspended. Citrus canker, however,

was again found in residential trees in the Miami area in October 1995 AD, and the tree removal regulations were reinstated.

Citrus canker has been eradicated from South Africa, Australia, and New Zealand. The latest outbreak and eradication of citrus canker in Australia occurred in 1991 AD. In South America, citrus canker was found in Brazil in 1957 AD. It subsequently spread to Uruguay, Paraguay, and Argentina and despite attempts to eradicate it, the disease has got permanently established there. Eradication efforts in Brazil, however, have kept the large citrus-producing areas of that country free of the disease. All citrus-producing countries without canker maintain a strict prohibition on import of citrus plants and fruit from non-canker free countries.

7.11.1 Impact on society and government

- In the largest individual program to eradicate a plant disease, the U.S. Government conducted a $200 million canker eradication program during the mid-1990 AD onwards, consisting of clear cutting of more than1.8 million infected trees or ones within a 32-m radius of an infected tree.
- Citrus canker is an extremely costly disease. Worldwide millions of dollars are spent annually on prevention, quarantines, eradication programs, and disease control.
- Undoubtedly, the most serious consequence of citrus canker infestations is the impact on commerce resulting from restrictions to interstate and international transport and sale of fruit originating from infested areas.
- The USDA, Animal and Plant Health Inspection Service in collaboration with the Florida Department of Agriculture and Consumer Services, Division of Plant Industry have formed a joint state/federal eradication campaign to eliminate the disease.
- An average of over 50 million dollars per year and over 600 personnel are presently dedicated to this program. In Florida alone, costs of running an eradication program from 1995 AD–2005 AD plus compensation to commercial growers and homeowners for residential citrus destroyed is approaching $1 billion dollars.

7.12 Butternut canker

Butternut is found in mixed hardwood forests throughout central North America from New Brunswick to North Carolina and their wood has been used for furniture and for carving. Butternut canker caused by *Sirococcus*

clavigignenti was identified as an invasive species in 1967 AD. It was first discovered in Wisconsin, but has since spread to other states and into Canada. In 1967 AD, butternut trees in Iowa were observed to have multiple cankers on branches and stems and subsequently died from the disease. Broad dead areas known as cankers form on the main stems, branches, young twigs, and exposed roots. Most cankers are covered with bark cracks. The fungus forms a dark mat of branching mycelium below the bark, from which arise peg-like hypha that lift and rupture the bark. In the later stages of infection, the bark above the canker is shredded.

The U.S. Forest Service found that 84% of all butternuts in Michigan as well as 58% of all trees from Wisconsin have been affected; later surveys by the Wisconsin Department of Natural Resources revealed that 91% of all living trees in Wisconsin were diseased or cankered. In Virginia and North Carolina, the butternut tree population has been reduced from 7.5 million to 2.5 million.

7.12.1 Impact on society and government

- The disease has spread so rapidly that the U.S. Forest Service estimated that about 80% of the butternut trees in the south-east had been killed by the mid-1990 AD onwards. The remaining survivors were mostly along the banks of streams and rivers, but most of them were also heavily infected and were not reproducing.
- USDA Forest Service Inventory and Analysis forest inventory data showed a dramatic decrease in the number of live butternut trees in U.S.
- Live butternut trees decreased by 58% in Wisconsin and 84% in Michigan in the last 15 years.
- A recent Wisconsin Department of Natural Resources survey revealed that 91% of the live butternut trees throughout Wisconsin were diseased.
- Surveys in the south-east U.S. revealed that 77% of the butternut trees have been killed in North Carolina and Virginia, and infected trees continues to be found in new counties in most of U.S.

7.13 Cypress canker

Cypress trees and other species grow in Mediterranean climates, including California, the Mediterranean, and Persia. For more than three millennia they have been valued as ornamentals for their tall, statuesque, columnar shape,

as well as for their wood, which is resistant to woodworms, rots, and decays. Cypress trees are extremely long living, some of them possibly living for more than 2000 years. Many of the world's centres of civilisation, such as the Acropolis of Athens, Olympia, Delphi, Florence, and others, and many of the paintings over the centuries derive much of their classic beauty from the real or painted cypress trees in them.

The first cypress canker outbreak was described in California in the mid-1920 AD onwards, but the disease apparently existed there earlier for more than 10 years. The disease then spread inland across the U.S. and into South America and, apparently, was transported from there across the oceans into the Mediterranean countries, New Zealand and South Africa so that by now it is believed to be present in most parts of the world where cypress trees grow.

Cypress canker or cypress blight is caused by the fungus *Seridium cardinale*. The fungus produces spores (conidia) that infect twigs and small branches through wounds and cause cankers that kill the twigs and branches. Resin flows out of the cracks of cankers while the foliage of infected twigs and branches turns yellowish to red at first, becoming reddish brown as the twigs die. A noticeable die-back of twigs, branches, and tree tops becomes visible from a distance. Heavily infected trees die. Large numbers and large percentages of cypress trees have been killed by the cypress canker fungus in the last few decades. Spread of the disease among the remaining trees continues, possibly at an accelerated rate.

7.13.1 Impact on society and government

- As many as one million cypress trees have been killed in central Italy, including Florence, with some groves showing more than 45% tree mortality from cypress canker infections.
- In some of the Greek islands and in parts of the mainland, 70–98% of the cypress trees have been killed by this disease.

7.14 The xylella outbreak

The European grape, *Vitis vinifera*, which provides all high-quality tables and wine grapes throughout the world, cannot be grown in the south-eastern U.S. because it is devastated by the indigenous xylem-inhabiting bacterium *Xylella fastidiosa*, the cause of Pierce's disease of grape.

The disease was reported in California as back as in the 1880 AD onwards, but lack of appropriate vectors, appropriate alternate hosts, and timing of unfavourable weather conditions kept the disease under control. As a result, grapes in California and Texas were free of that disease, but in 1990 AD, the

disease was found in Texas and from there it has spread widely among the vineyards and has caused heavy losses.

In 1998 AD, one of its plant hopper vectors and the bacterium causing Pierce's disease were found in vineyards of southern California, threatening not only the grape industry, but also many of the ornamental crops of California. *Xylella* bacteria were expected to do well in the California climate, but the absence of an effective vector of the bacteria provided protection and comfort to its agricultural industry. Now that the bacteria and one of their vectors have been brought together in that state, the California grape industry, and possibly its ornamentals, will probably never be the same again.

7.14.1 Losses and impact

It is present in some California vineyards every year, with the most dramatic losses occurring in the Napa Valley and in parts of the San Joaquin Valley. During severe epidemics, losses due to pierce disease may require major replanting. In Florida and other south-eastern states, pierce disease has precluded commercial production of European varieties, but some muscadine grapes and hybrids of American wild grape species with European grapes (*Vitis vinifera*) are tolerant or resistant to pierce disease.

7.15 Panama wilt of banana

Bananas (*Musa* species) are an example of a crop where the spread of soil borne pathogens has caused tremendous losses worldwide and necessitated the abandonments of many acres of contaminated land. Panama disease, also known as Fusarium wilt of banana (*Musa* spp.), is one of the most notorious of all plant diseases.

Although the pathogen probably originated in south-east Asia, the pathogen and the disease due to it was first reported in Australia in 1876 AD. The same disease was reported in Panama in 1890 AD with severe intensity and gained a name of Panama wilt. By 1950 AD, few banana-producing regions remained free of the disease. Panama disease is now found in all banana-producing regions except islands in the South Pacific, the Mediterranean, Melanesia, and Somalia. Panama disease impacts the production of a wide range of banana cultivars, however, it is most widely known for damage it caused on a single cultivar in the early export plantations. Prior to 1960 AD, the export trade was based almost entirely on the susceptible cultivar *Gros Michel*. This reliance on *Gros Michel* and the common practice of using infected rhizomes to establish new plantations resulted in widespread and severe losses, especially in the western tropics.

The first internal symptoms develop in feeder roots, the initial sites of infection. They progress to the rhizome and are most prominent where the stele joins the cortex. As the pseudo stem is colonised, faint brown streaks or flecks become evident on and within older leaf sheaths. Eventually, large portions of the xylem turn brick red to brown colour. The first external symptoms of Panama disease are a yellowing of the oldest leaves or a longitudinal splitting of the lower portion of the outer leaf sheaths on the pseudo stem. This is followed by a wilt and buckling of leaves at the petiole base. In some cases, these leaves remain green. As the disease progresses, younger and younger leaves collapse until the entire canopy left consists of dead or dying leaves.

7.15.1 Geographical distribution

Although the disease probably originated in south-east Asia, the first recording of the disease was made in 1874 AD in Australia, where it was observed at Eagle Farm near Brisbane. It was then reported from Panama in 1890 AD. Within a decade, the disease had spread to Costa Rica and subsequent outbreaks occurred in Surinam (1906 AD), Cuba (1908 AD), Trinidad (1909 AD), Jamaica (1911 AD), Honduras (1916 AD) and Guatemala (1919 AD). The disease has since been reported from most banana-producing countries.

The fungal strain tropical Race 4 (TR4) is used to distinguish this strain that readily causes Fusarium wilt on Cavendish bananas from the other strain that requires predisposing factors such as low temperatures and waterlogging to cause disease and is thus known as subtropical Race 4 (STR4). The strain associated with TR4 was identified in samples from Taiwan in 1990 AD. The vulnerability of Cavendish cultivars was highlighted when newly established plantations were decimated in Malaysia and Indonesia in the early 1990 AD onwards.

Since then TR4 has been found in the island of Borneo (in both the Malaysian and Indonesian parts of the island), other Indonesian islands (Papua province, Kalimantan, Halmahera, Java, Sulawesi and Sumatra), mainland China (Guangdong, Hainan, Guangxi, Fujian and Yunnan), the Philippines and Australia (Northern Territory in 1997 AD and Queensland in 2015 AD). It was reported to be in Jordan and Mozambique in 2013 AD, and in Pakistan and Lebanon in 2015 AD. There are reports that it is also present in Oman.

In India, symptoms of Fusarium wilt have also been observed in the Cavendish cultivar *Grande Naine* in the absence of predisposing factors, with the difference that the vegetative compatibility group of the isolate (VCG0124) is normally associated with Race 1.

7.15.2 Impact on society and government

- In the Ulua Valley of Honduras alone 30,000 ha were lost between 1940 AD and 1960 AD.
- Damage occurred more rapidly in areas such as Surinam, where an entire operation of 4,000 ha was out of production within 8 years, and the Quepos area in Costa Rica, where it took 12 years for 6,000 ha to be destroyed. It costs between $2,000 and $5,000 to establish a hectare of banana plantation at the time, direct losses due to the disease and replanting during the "Gros Michel Era" reached many millions of dollars.

7.16 Southern corn leaf blight

Southern corn leaf blight (SCLB) is a fungal disease of maize caused by the pathogen *Bipolaris maydis* (also known as *Cochliobolus heterostrophus* in its teleomorph state). The fungus is an *Ascomycetes* and can use conidia or ascospores to infect. There are three races of *B. maydis*: Race O, Race C, and Race T; and the symptoms of southern corn leaf blight vary depending on the infectious pathogen's race. Race T is infectious to corn plants with the Texas male sterile cytoplasm (cms-T cytoplasm maize) and this vulnerability was the cause of the U.S. southern corn leaf blight epidemic of 1969 AD–1970 AD. For this reason, Race T is of particular interest for U.S. maize growers. Although the disease thrives in warm, damp climates, it can be noticed in many of the world's maize-growing areas.

In 1970 AD, almost 85% of U.S. corn fields were planted with one type of corn, called Texas cytoplasmic male sterile (Tcms) corn and that was the main reason for the epidemic of the disease in U.S.

The three races of southern corn leaf blight pathogen attack the maize lines of various cytoplasmic background to cause disease symptoms.

Race and Toxin Produced	Susceptible Maize Lines
Race O/O toxin	Maize with normal cytoplasm (N)–most maize plants
Race T/T-toxin	Maize with Texas male sterile cytoplasm (Tcms)–these plants have gene *T-urf 13*, which encodes for T-toxin's site of action
Race C/C-toxin	Maize with cytoplasm male sterile C (Ccms)–currently found only in China

The prevalence of farming practices and optimal environmental conditions required for the propagation of *B. maydis* in the U.S. led to an epidemic of southern corn leaf blight in 1970 AD. In the early 1960 AD onwards, seed corn companies began to use male sterile cytoplasm so that they could eliminate the previous need for hand detassling to save both money and time. This seed was eventually breed into hybrid crops until there was an estimated 90% prevalence of Texas male sterile cytoplasm (Tcms) maize, vulnerable to the newly generated Race T. The disease, which first appeared in the U.S. in 1968 AD, reached epidemic status in 1970 AD and destroyed about 15% of the corn belt's crop production that year. In 1970 AD the disease began in the southern U.S. and by mid-August had spread north to Minnesota and Maine. It is estimated that Illinois alone suffered a loss of 250 million bushels of corn due to southern corn leaf blight. The monetary value of the lost corn crop is estimated at one billion US dollars. In 1971 AD, the losses due to this disease had disappeared mainly due to the return usage of normal cytoplasm corn, as weather being not so conducive, residues being buried, and early plantation. The disease epidemic highlighted the issue of genetic uniformity in monoculture crops, which allows for a greater likelihood of new pathogen races and host vulnerability.

In the present day, there are many management methods and better education practices but the disease can still be an issue in tropical climates, causing devastating yield losses up to 70%.

7.16.1 Impact on society and government

- The losses of corn were catastrophic, reaching as high as 50–100% in some areas of the U.S.
- The actual food energy losses were considered to be greater than those caused by the potato late blight epidemic of the 1840AD's.
- The economic losses from southern corn leaf blight disease totalled about 1 billion dollars.

7.17 Bacterial leaf blight of rice

Rice *(Oryza sativa)* is one of the most important food crops in the world, feeding about half of humanity. The disease bacterial leaf blight (BLB) of rice caused by bacterial pathogen *Xanthomonas oryzae* pv. *oryzae* is said to have been observed first by farmers in Japan during 1884 AD–1885 AD.

Its occurrence has been reported from Australia, Bangladesh, Cambodia, Indonesia, India, Korea, Mainland China, Malaysia, Srilanka, Thailand, Philippines, U.S., west Africa and Vietnam (Ezuka and Kaku, 2000 AD). It

has been observed during recent years that BLB incidence is increasing in Pakistan especially in *Kaller* belt which is famous for rice cultivation (Khan et al., 2000 AD).

BLB occurs at all the growth stages of rice and is manifested by either leaf blight or *kresek* symptoms. The causal bacterium invades plants through water pores and wounds (Mizukami, 1956 AD; Tabei and Mukoo, 1960 AD). Since the water pores are located at the margins of upper parts of the leaf, the lesion usually starts from the leaf margin near its tip. As the disease progresses, the tiny water soaked lesion turns yellow, enlarges in size progressively and develops into an elongated irregular lesion with wavy margins. Bacterial ooze, which consists of small, yellowish, spherical masses, may sometimes be seen on the margins or veins of the freshly infected leaf under moist conditions. With the passage of time, the lesion may cover the entire blade, which turns white and later grayish owing to saprophytic growth (Tagami and Mizukami, 1962AD; Octa, 1970 AD; Ou, 1985 AD). If plant ever produces panicles, it results in sterile immature grains, which are easily broken during milling. There may be 50% reduction in yield in case of severe infection (Mew et al., 1993 AD) whereas 10–12% yield reduction has been recorded in case of mild infection (Ou, 1985 AD).

Bacterial blight has the potential to become a destructive disease of rice in Pakistan and can cause huge losses mainly because the information regarding the pathogen and its effective control measures is lacking among the farmers. Resistance to BLB is known to be widely different with rice cultivars. This is due to the fact that the presences of different pathogenic races subsequently break the resistance of rice cultivars. So evaluating rice cultivars for BLB resistance is a routine practice to overcome yield losses.

In the early 1960 AD a dwarf rice cultivar IR8, which had high yields, was non-lodging and had good response to nitrogen was planted throughout south-east and south Asia. Unfortunately, this cultivar was also very susceptible to BLB. Since the pathogen was endemic in the region amongst the moderately resistant native cultivars, an epidemic of the disease occurred. In fields planted with high yield varieties, bacterial blight often cut yields by 20–50% throughout the 1960 AD onwards.

All over south-east Asia, epidemics broke out, with loss of yields as high as 80% in some areas. The problems were compounded by the fact that the pathogen changes quickly to overcome resistance, especially under monoculture conditions. Uniform crops exert selection pressure on the pathogen, and those pathogen strains for which the rice has no defence end up quickly dominating the populations. By collecting farmer varieties and *wild* material throughout the world, the International Rice Research Institute (IRRI) in the Philippines has now identified around 2,200 lines resistant to bacterial

blight. The bulk of these varieties come from three geographic centres: one in the area comprising Bangladesh, Nepal and north-east India, a second in Southern India and Sri Lanka, and a third in Java and the surrounding islands.

7.17.1 Losses and economic impact

Bacterial leaf blight is the most serious disease of rice in south-east Asia, particularly with the widespread cultivation of dwarf high-yielding cultivars (Ray and Sengupta, 1970 AD; Feakin, 1971 AD). In 1954 AD, in Japan, 90–150 thousand ha were affected and annual losses put at 22–110 thousand tonnes. The disease was first reported in India in 1951 AD, but it was not until 1963 AD that an epiphytotic occurred. In the Philippines, present losses are of the order of 22.5% in wet season to 7.2% in dry seasons in susceptible crops and 9.5–1.8%, in resistant crops (Exconde, 1973 AD). Nitrogen fertilisation considerably increases susceptibility. Losses are generally less important in the less fertile soils and in summer-grown crops (December–April). Transplanted autumn (May–September) and winter (July–December) crops, however, suffer considerable losses. Diseased crops contain a high proportion of chaffy grains. The world situation has been reviewed by an International Workshop held at Manila, Philippines (Banta, 1989 AD).

7.18 Downy mildew of grapes

Downy mildew of grapevine is present to a greater or less extent in nearly all parts of the world where grapes are cultivated (in Greece it is less intense in southern region than the moist centre and north; in Northern Africa i.e. Algeria, the dry inland region suffers little while in coastal belt the disease causes trouble; in U.S. it prevails in East of Rocky mountains and reaches its greater severity in the northern Mississippi valley and states to the eastwards especially the middle Atlantic States; in Australia; South Africa; South America particularly Brazil; New Zealand, Britain and India).

The disease was first observed in America on wild grapes in 1934 AD and the fungal origin of trouble was recognised by Schweintz in the same year; however, the parasite was first described by Berkeley and Curtis in 1848 AD. De Bary studied the fungus very carefully in 1863 AD, Farlow in 1876 AD and Berlese and De Toni in 1888 AD, when it was assigned to the present genera. The disease may be considered endemic in the eastern U.S. from where it spread to France, perhaps some time earlier than 1874 AD.

The first severe infestation in France was recorded in 1879 AD. Severe epiphytotic have been recorded thereafter in 1900 AD, 1910 AD, 1915 AD and 1927 AD in France. Subsequently, the spread of the disease to other parts of Europe followed. It reached South Africa in 1907 AD. The first severe

outbreak in Australia occurred in north-east Victoria in 1917 AD (De Castella and Brittlebank, 1923 AD) and reached New Zealand in 1926 AD. The disease was first reported in India by Burns in 1910 AD on leaves of *Vitis vinifera* near Pune and Chennai. In India the disease is common in Maharashtra State.

7.18.1 Losses and economic impact

The disease caused heavy losses in U.S. and Europe during 1870 AD–1880 AD and is noted as **European epidemic of downy mildew of grapevine in the history of plant diseases**. A loss of 70% of potential yield was reported during epidemic of 1915 AD in France alone.

It is also known to have caused serious losses in some years in northern Africa, in South Africa, in parts of Asia, in Australia and South America. Fortunately epidemics of downy mildew have not so far been reported in India. However, when weather is favourable and no proper protection against the disease is provided downy mildew can easily destroy 50–70% of the crop in one season leaving the farmers in miserable conditions and bankruptcy.

7.19 Apple scab

Apple orchards suffer from a serious disease on apple trees known as Apple scab caused by the fungus *Venturia inaequalis*. The disease manifests as dull black or grey-brown lesions on the surface of leaves, petioles, blossoms, fruits, pedicles and less frequently on young shoots and bud scales. Lesions may also appear less frequently on the woody tissues of the tree. Fruits and the undersides of leaves are especially susceptible. Young lesions on the leaves are velvety brown to olive green and have feathery in distinct margins. Later the margin becomes distinct, but they also become obscured as several lesions coalsce. As the infected leaf ages, the tissue adjacent to lesion thickens resulting in deformed leaves. The disease rarely kills its host, but can significantly reduce fruit yields and fruit quality. Affected fruits are less marketable due to the presence of the black fungal lesions.

The disease is important in the Indian apple growing states of Jammu & Kashmir and Himachal Pradesh.

In India it was first detected in 1930 AD on the native cultivated Ambri in Kashmir valley. In 1973 AD, this disease appeared in epidemic form in the valley where by about 70,000 acres orchard got infected. This epidemic resulted in a loss of Rs.54 lakhs in a season. In July 1977 AD, scab was detected in Himachal Pradesh in few localised pockets. Subsequently, the disease spread was so quick that the entire apple growing area of the state was affected resulting in severe epidemic in 1983. Realising the magnitude of the

problem and finances required the Himachal Pradesh government prepared a special scheme under endemic disease control programme during the year 1978 AD to which Union Government agreed to finance to the extent of 50% with the state government. The orchard area affected due to this disease is nearly 1,18,000 ha out of total 1,82,000 ha area under apple cultivation in India.

7.19.1 Impact on society and government

- The losses of Rs. 5.4 million occurred in a single season in Kashmir valley in 1973 AD at the time of first epidemic. Due to diseased apples ultimately beverage industry suffered loss.
- In Himachal Pradesh during 1983 AD, epidemic, 10% of apple crop (30,000 out 3,00,000 metric tonnes) were made unfit for market and had to be destroyed on the spot resulting in a loss of Rs. 15 millions to state government which paid compensations to farmers. The accumulated losses by the disease were around 50 millions i.e. 10% of the total income.

7.20 Soybean rust

Soybean rust *(Phakopsora pachyrhizi*) has been known to occur in eastern Asia and Australia for decades. In recent years, the disease entered Africa and South America and has spread rapidly in these continents. It has become a cause of concern to the U.S. soybean industry. Soybean rust was indeed discovered in the states of Louisiana, Mississippi, and Florida in the middle of November of 2004 AD. Since the U.S. annually produces approximately 70 million metric tonnes of soybeans, or slightly more than half the world production of soybeans, introduction of this pathogen into the U.S. is a major financial catastrophe to producers and worldwide consumers of soybeans alike. It has been estimated that much of the soybean-producing area in the U.S. would suffer a loss of 10% or more, in the south-eastern U.S., which have more favourable climatic conditions for the disease to flourish.

The main regions where rust has not been reported (but might over winter) are located in the western hemisphere, including northern South America, central America, the Caribbean, Mexico, southern Texas, and Florida. South-eastern China and neighbouring areas are suggested as the primary regions where initial spores for soybean rust epidemics in central China are produced. If the disease is to establish in the U.S., it is likely to be restricted to parts of Florida and southern Texas during the winter in the frost-free areas or areas where the fungus could overcome short periods of below-freezing

temperatures. Occurrence of rust epidemics within the U.S. soybean belt would depend on south-to-north dispersal of uredospores.

Soybean rust has been known to occur in the far east from Japan to Australia, in India, parts of central Africa, in central and south America, and the islands of the Caribbean Basin. Wherever it occurs, it causes severe losses in yield ranging from 10% to 50%, with even higher losses, up to 80%, occurring in the more humid tropical and sub-tropical regions. The soybean rust fungus has not yet been found in the continental U.S., but in 1995 AD it was found in Hawaii. Considering that the fungus produces hardy, windborne uredospores, it is considered quite likely that the pathogen will be introduced into the mainland of the U.S. in the next few years.

Its life cycle appears to be microcyclic (producing only uredia and telia) and is completed on the same host. Uredia produce uredospores that are spread by wind and can cause infection, while the telia produce teliospores, which however, have never been shown to germinate.

In the U.S., the pathogen has been quarantined vigorously in the past and its quarantine will continue.

7.20.1 Impact on society and government

- U.S. would suffer a loss of about 50%, bringing the cost of the disease to producers and consumers to ~$7.2 billion per year.

7.21 Red rot of sugarcane

Red rot is the most dreadful disease of sugarcane. It is of great economic importance and cause of failure of many popular varieties in different countries. The disease first came to notice in Java (now Indonesia) by Went (1893 AD). He called the fungus as ***Colletotrichum falcatum*** and named the disease as *netrood sno* meaning *red smut*.

In India, large scale mortalities of sugarcane were noticed in the Godavari delta of Andhra Pradesh due to an unknown disease in early nineties of the 19th century. Barber (1901 AD) studied the disease and reported that it was caused by ***Colletotrichum falcatum***. Red rot is widely distributed and present in many countries of the world (Sevanesan and Waller, 1986 AD). However, it is regarded as a disease of major importance in India, southern U.S., Queensland, Pakistan, Bangaladesh and Myanmar. In India, the disease is most destructive in sub-tropical parts of the country and coastal areas of Andhra Pradesh and Tamil Nadu. The occurrence of the disease was reported for the first time from northern Karnataka and southern Maharashtra during 1997 AD–1998 AD (Anonymous, 1998 AD). The disease is fast becoming a limiting factor

in sugarcane production, particularly in Uttar Pradesh, Uttaranchal, Bihar, Punjab and Haryana in sub tropical and Andhra Pradesh, Tamil Nadu and Gujarat in tropical India.

In India, the first documented epidemic occurred in 1895 AD–1901 AD and in subsequent years a number of major outbreaks have been recorded as a regular event in the sub-tropical and tropical regions of the country. The most widespread epidemic of the disease the country experienced was in eastern U.P. and northern Bihar during 1938 AD–1940 AD, resulting in failure of variety Co 213. A reduction of about 70,000 tonnes of sugar production of the country during 1939 AD–1940 AD was due to red rot epidemic (Chona, 1980 AD). These epidemics have resulted in the elimination of several commercial varieties. There have been reports of red rot epidemics in many other countries viz; U.S., Mauritius, Australia, Hawaii and Myanmar (Abbott and Hughes, 1961 AD).

Expression of the disease may vary depending on the environmental conditions. If setts are infected and sufficient inoculum is present, it causes both pre- and post-emergence death of the sprouts during April–June. Early symptoms during pre-monsoon period are expressed as dark brown coloured continuous lesions with heavy sporulation on both the surfaces of the mid-rib of unfolded leaf spindle and old leaf sheaths. Typical symptoms in standing crops are seen during monsoon season. In addition to the stalk symptoms, the disease appears on the mid-rib of the leaf as red elongated lesions with ashy grey centre.

7.21.1 Impact on society and government

- A reduction of 70,000 tonnes of sugar production in India during 1939 AD–1940 AD.
- Elimination of several commercial varieties of sugarcane.
- Affect the sugar recovery and thereby less pricing of sugarcane to the farmers.

7.22 Rice tungro disease

Rice tungro virus disease appears if a susceptible variety, virus inoculum and the vector green leafhopper (*Nephotettix virescens*) that carries the virus are available in a rice field. Plants infected with rice tungro virus showed marked stunting, yellow to yellow orange leaf discolouration and reduced ear-bearing tillers. Two viral particles, namely spherical (RTSV: an RNA virus) and bacilliform (RTBV: a DNA para retrovirus) are known to be associated with rice tungro virus disease.

Tungro disease is a serious threat to rice production in many parts of the world. In India the disease was first observed in the mid-60s in the rice growing areas of West Bengal and Orissa. Since then the disease has also been reported from Andhra Pradesh, Assam, Bihar, Karnataka, Kerala, Manipur, Tamil Nadu, Tripura and Uttar Pradesh. Tungro disease, which appeared in north India during 1967 AD, moved to peninsular India in 1977 AD.

Tungro outbreaks were reported to be discontinuous within a district, state and country over the years. For example: TN-1 was found infected at the experimental farm of the Indian Agricultural Research Institute (IARI), but not in the farmers' field in Delhi and the surrounding areas. After 1995 AD, the disease also did not occur at the IARI experimental farm. However, in August 1998 AD, a widespread yellowing and stunting of rice crop in the Gurdaspur and Amritsar districts of Punjab was observed. Almost all the cultivars of rice grown in these districts in an area of about 0.45 million ha were severely affected. The outbreaks of this disease were restricted to irrigated and rain fed shallow lowlands. The loss from tungro epidemics steadily increased during 1979 AD–1980 AD.

Three major epidemics in farmers' fields during 1984 AD, 1988 AD and 1990 AD, caused severe quantitative and monetary losses. Each of the other two epidemics during 1987 AD and 1998 AD led to a similar loss of about a million tonnes (mt) in rice production, but showed a steady increase in loss in terms of real value.

7.22.1 Impact on society and government

An epidemic outbreak of tungro during 2001 AD in three districts of West Bengal caused an unmilled rice production loss of 0.5 mt valued at Rs. 2911 millions at current prices. The studies demonstrated that tungro epidemics could cause a maximum production loss of 53% in a district, 23% in a state and 2% in the country.

7.23 Bacterial blight of tomato

The bacterium *Xanthomonas campestris* pv. *vesicatoria* causes leaf spot and blight on tomato and capsicum and is observed to be a major disease on tomato in Nashik region of Maharashtra, India. Tomato cultivation in Nasik district occupies over 14,000 ha of land around the year with a net profit of Rs. 0.1 million/ha as 60% of tomatoes are transported to other parts of Indian states and abroad. Infection of the bacterium *X.c.*pv. *vesicatoria* was consistently appearing on tomato cultivation in the area since 1990 AD, but during kharif 1992 AD, tomato crop succumbed heavily to the disease throughout the cultivated area due to favourable epiphytotic condition (26–30°C temperature,

93–97% RH coupled with either rain/downpour and cloudiness) and within 10–15 days whole tomato crop was devastated forcing the farmers to have only one picking as against 4–5 normal pickings. Most of the tomato hybrids under cultivation were susceptible to the disease. Market surveys revealed that there was 40% decline in the tomato supply to the market yard due to the disease and the losses estimated were around Rs.190 million, thus assuming the disease an epidemic.

Estimating the gravity of this bacterial disease, the pathogen *Xanthomoas campestris* pv. *vesicatoria* was isolated and various agrochemicals were tested against the bacterial pathogen for their effectiveness at Agriculture Research Station, Niphad, district Nasik by Professor S.G. Borkar. These effective agrochemicals were recommended for the management of this disease. A documentary film was made on this disease and its management which was the first documentary film on bacterial plant disease in India and probably in the world. The Indian phytopathological society awarded a commendation certificate to this film under teaching aid during its annual conference held at Coimbatore in 1993 AD. The film was shown at different locations in the districts to create awareness about this disease among the tomato cultivators which subsequently helped them to control this disease. Since then, the region did not have any other epidemic of this disease.

7.23.1 Impact on society and government

A loss of Indian Rs.190 million to the tomato growers in Nasik region has occurred during 1990 AD–1991 AD. Shortage of tomatoes in the local market affecting the supply to other states in India and increase in the tomato prices.

7.24 Bacterial blight of pomegranate

The bacterial pathogen *Xanthomonas axonopodis* pv. *punicae* causes a serious disease on pomegranate known as oily spot or bacterial blight. Bacterial blight is a devastating disease of this crop reported from major pomegranate growing areas of Maharashtra, Karnataka and Andhra Pradesh in India from 2003 AD onwards. The infection of the bacterium *Xanthomonas axonopodis* pv. *punicae* was so severe that most of the pomegranate orchards in Jat and Sangola areas of Sholapur district have been destroyed and uprooted by the farmers incurring loss of 100% yield and financial losses. The economic losses due to this disease were estimated to the tune of Rs. 23,183 million in the diseased areas and the contribution of the Indian government to combat the disease was Rs. 10,000 million during 2003 AD–2008 AD.

Based on the laboratory and field study conducted at Department of Plant Pathology, Mahatma Phule Agriculture University, Rahuri under leadership of

Professor S.G. Borkar, a protocol was formulated and validated on farmers' field with 100% success in the management of this disease. Those contract farmers who followed the protocol step by step without missing any component of the protocol could control the disease in their field and are still managing their crop without bacterial incidence. The recommendations/protocol for the control of the disease should be applied in totality to keep the pathogen under check.

7.24.1 Impact on society and government

A loss of Rs. 23,183million to pomegranate growers has occurred in peninsular India with several agitations of the farmers for compensation of the losses of their crops. This led to anxiety and fear among the farmers about the disease and its occurrence.

7.25 Fire blight of apple and pear

The bacterium *Erwinia amylovora* causes fire blight on apple and pear, the most important fruit crops of temperate region. It is generally believed that fire blight was originated on wild hosts presumably Crataegus in the north-eastern U,S., and has been described after the import and cultivation of European apple and pear varieties in U.S. (van der Zwet and Keil, 1979 AD). The first description of the disease outside the U.S. was in New Zealand (1919 AD). In Europe, fire blight was first described in the U.K. (Kent) in 1957 AD and in subsequent years the spread of the disease was recorded in northern, western and central Europe. In 1998 AD, all countries belonging to the European Union (except Portugal) had fire blight on pears, apples or ornamentals, either widespread (England, Belgium, Germany), localised (France, Switzerland) or in restricted spots, under control and local eradication (Spain, Italy, Austria). It is said that western Europe has been invaded by fire blight in the second half of the 20th century. However, even today, wide areas of Europe (Italy, Spain and the south-east of France) remain free of fire blight. Fire blight also invaded a large area around the Mediterranean Sea and most probably spread from an initial outbreak detected in the Nile delta region of Egypt in 1964 AD. The disease was later found in Greece (Crete), Israel, Turkey, Lebanon, Iran and countries of Central Europe. The introduction and infection of *E. amylovora* in Egypt and England has resulted in one continuous zone of fire blight area, which encompasses most of western Europe and most of the Mediterranean region. Only countries in North Africa seem to be free of fire blight; although the disease has recently been described in Morocco.

A number of unconfirmed reports of fire blight (China, India, Korea, Saudi Arabia, Vietnam, Colombia) may rely on misdiagnosis, or insufficient description of the causal agent (confusion of fire blight with pear-blast symptoms caused by *Pseudomonas syringae* pv. *syringae* or with other *Erwinia* species reported on Asian pear). It must also be remembered that *E. amylovora* is a quarantine organism (list A2 OEPP), the economic consequences of a declaration of the presence of fire blight in a country may have costly consequences for the international trade of that country: it cannot be ruled out that the list of actually *infected* countries is slightly longer than the list of officially declared areas.

In most cases attempts to eradicate the pathogen in newly infected countries only slows down the spread of the disease. Until fire blight is again detected in Australia, this country might be the only case where eradication has been successful. Fire blight-like symptoms were detected on cotoneaster in the Royal Botanic Gardens, Melbourne, Victoria, in April 1997 AD, and diagnostic tests confirmed that the causal organism was *E. amylovora* (Rodoni et al., 1999 AD). An intensive eradication programme was undertaken and national surveys conducted for 3 years following the detection of *E. amylovora* have confirmed the absence of the disease in all states of Australia (Rodoni et al., 2002 AD). There has been no positive detection of *E. amylovora* in New South Wales.

Large areas of the world are still free of fire blight (South America, most of Africa and Asia) in spite of the fact that susceptible cultivars of European and American origin are grown in these areas, and that potentially susceptible host plants may be common in the environment (EPPO/CABI, 1998 AD).

7.25.1 Impact on society and government

The disease causes immediate losses to the crop as it spreads like a fire. Anxiety and fear of the appearance of the disease in the fields at any time among the farmers. Due to its quarantine importance the planting material from infected areas are banned from export to other regions affecting the nursery business.

8

Historical crop yield losses due to plant diseases

Plant diseases, by their presence, prevent the cultivation and growth of food plants in some areas. Food plants may be cultivated and grown but plant diseases may attack them, destroy parts or all of the plants, and reduce much of their produce, i.e., food, before they can be harvested or consumed or reduce the quality of produce due to disease symptoms on them.

It is conservatively estimated that diseases, insects, and weeds together annually interfere with the production of, or destroy, between 31% and 42% of all crops produced worldwide (Table 1). The losses are usually lower in the more developed countries and higher in the developing countries, i.e., countries that need food the most. It has been estimated that out of the 36.5% average of total losses, 14.1% are caused by diseases, 10.2% by insects, and 12.2% by weeds. Considering that 14.1% of the crops are lost to plant diseases alone, the total annual worldwide crop loss from plant diseases is about $14 billion. To these should be added 6–12% losses of crops after harvest, which are particularly high in developing tropical countries where training and resources such as refrigeration and storage are generally lacking. Also, these losses do not include losses caused by environmental factors such as frosts, droughts, air pollutants, nutrient deficiencies and toxicities.

Table 1 Estimated Annual Crop Losses Worldwide.

1	Attainable crop production	$1.5 trillion
2	Losses in crop production (36.5%) due to pest and weeds.	$540 billion
3	Losses caused by diseases only (14.1%)	$14 billion

Crop losses caused by plant pathogens, worldwide, have obstructed efforts to increase crop productivity as well as total production. In spite of significant progress achieved in plant breeding, chemotherapy and other disease management techniques, losses due to diseases remain a major factor in limiting agriculture in many parts of the world, including many developed nations. According to the Cramer, global pre-harvest losses due to plant pathogens are 9–15% of annual production. Crops like potatoes, grapes and

coco incur even higher losses due to disease, 20% or more on a worldwide basis. The USDA handbook provides estimates of disease losses in the U.S. of 11.5%, resembling the estimates of 13.1%, 12.9% and 11.3%, respectively for Europe, Africa and Asia (Teng and Shane, 1984 AD).

The losses in crop production do not include the innumerable stories of large populations in many poor countries suffering from malnutrition, hunger, and starvation caused by plant diseases; or of loss of income and loss of jobs resulting from crops destroyed by plant diseases, forcing people to leave their farms and villages to go to over crowded cities in search of jobs that would help them survive. Moreover, the recurrence of plant diseases in an area limits the amount of land available for cultivation of that particular crop each year. It also limits the kinds of crops that can be grown in fields already contaminated with certain microorganisms and annually necessitates the use of millions of kilograms of pesticides for treating seeds, fumigating soils, spraying plants, or the post-harvest treatment of fruits. Such control measures not only add to the cost of food production, some of them, e.g., crop rotation, necessarily limit the amount of food that can be produced, whereas others add toxic chemicals to the environment.

8.1. The major diseases which caused worldwide yield loss (Tripathi, 2010 AD)

8.1.1 In the West (Europe and America)

8.1.1.1 Late blight of potato

The devastating outbreak of **late blight of potato** (*Phytophthora infestans*) that began in Europe in 1845 AD and brought about the **Irish famine** caused starvation, death, and mass migration of the Irish population. Out of a population of 8 million, approximately 1 million (about 12.5%) died of starvation and 1.5 million (almost 19%) emigrated, mostly to the U.S., as refugees from the destructive blight. This fungus thus had a tremendous influence on the economic, political, and cultural development in Europe and the U.S. During World War I, late blight damage to the potato crop in Germany may have helped in ending the war.

8.1.1.2 Southern corn light

In 1970 AD, epidemic of corn blight by new race specific for plants with Texas male sterile cytoplasm occurred in southern U.S., and was responsible for 15% of U.S. corn supply loss (enough to feed sufficient cattle to make 30 billion Big Macs, USDA projections were for 90–100% loss in 1971 AD).

8.1.1.3 Stem rust of cereals

Widespread epidemics have also been documented for Australia. Losses in north Dakota, during the severe epidemics of 1935 AD and 1954 AD, were estimated at $356 million and $260 million, respectively, based on wheat prices in late 1995 AD.

8.1.1.4 Bacterial soft rot

Erwinia carotovora subsp. *carotovora* affects a wide range of plants causing soft rot of potatoes and many fruits and vegetables. The mean incidence of soft rot ranged from 10–100%.

8.1.1.5 Bacterial wilt of solanaceous crops

Serious obstacle to the cultivation of many solanaceous plants like potato, tomato and brinjal in both tropical and temperate regions, are the *Ralstonia solanacearum* causing wilting in these crops. When seed potatoes are cut during planting, the disease incidence was found to increase by 250% and yield was reduced by 40%. In India, tomato yield loss study with one cultivar showed 10–100% mortality of plants and 10–91% yield loss, respectively, at different stages of infection (0–90 days).

8.1 1.6 Potato cyst nematodes

Potato cyst nematodes cause extensive damage and the crop losses can be as high as 80%. *G. rostochiensis* reduced the potato yield by 76%; the marketable yield of 13.75 t/ha on treated areas was reduced (by 85%) to 1.96 t/ha in non-treated areas. In Germany, the populations of 500 larvae/100 cm^2 recorded a loss on affected areas at 10–15%.

8.1.1.7 Root knot nematode

In the field, crops including lucerne, groundnut, potato, carrot, sugarbeet, strawberry, pyrethrum and onion may be severely affected. Losses may range from 30–60% in severely affected areas.

8.1.2 In Georgia

Woodward (2010 AD) reported losses in various crops due to different diseases.

(1) In apple: 5.0% and 1.0% of yield losses are due to bitter rot and fire blight, respectively.

(2) **In blackberry:** 5.0% of yield losses are due to viral diseases.

(3) **In bunch grapes:** 9.5% of yield losses are due to downy mildew disease caused by *Plasmopara viticola.*

(4) **In corn:** 11.1% of yield losses are due to southern corn rust.

(5) **In peanut:** 13.5% of yield losses are due to various diseases like white mould, leaf spots, pod rot and tomato spotted wilt virus.

8.1.3 Indian continent and far east

8.1.3.1 In India

Dhaliwal and Arora (1993 AD) reported the pre-harvest loss up to 30% in cereals and pulses and it can be up to 50% in cotton and oil seeds crops due to various diseases in India.

(1) **In rice**

Padmanabhan (1965) reported that 2,66,000 tonnes (0.8% of total yield) of rice were lost in India due to blast disease.

Reddy (1980 AD) reported 65–93% and 86% yield losses in rice due to bacterial leaf blight disease caused by *Xanthomonas oryzae* in varieties like Jaya and PR 106, respectively in Punjab state.

Raina et al., (1981 AD) reported grain losses ranging from 60–70% in rice due to bacterial blight.

Nagarajan (1989 AD) reported 4.5–43.0% yield losses in rice due to rice blast disease caused by *Pyricularia oryzae* from major growing states like West Bengal, Tamil Nadu, Andhra Pradesh, Maharashtra, Uttar Pradesh, Madhya Pradesh, Kerala, Tripura, Karnataka, Gujrat, etc.

Naidu (1992 AD) reported sheath blight of rice caused by *Rhizoctonia solani* as a potential threat to rice cultivation, causing severe yield losses up to 69% to the rice crop.

Muralidharan et al., (2003 AD) reported that the tungro epidemics could cause a maximum production loss of 53% in a district, 23% in a state and 2% in the country.

(2) **In wheat**

Singh et al., (1985 AD) reported 1.2–9.6% yield losses in HD 2009, Sonalika, WL 711 and WL 1562 varieties of wheat due to Karnal bunt disease caused by *Neovossia indica* from north-western states.

(3) In pearl millet

Thakur (1986 AD) reported 2.3–30.1% crop losses due to Downy mildew disease caused by *Sclerospora graminicola* from various high yielding varieties like HB 3, NHB 5, PHB 15, BJ 104 and BS 1.

(4) In barley

Pant and Bisht (1983 AD) reported stripe disease of barley which caused 70–72% yield loss.

(5) In greengram and blackgram

Jain et al., (1995 AD) reported 67% yield losses caused by yellow mosaic virus in greengram and blackgram.

(6) In groundnut

David (1995 AD) estimated the yield losses caused by collar rot in groundnut, which were ranging from 28% to 47%**.**

(7) In maize

Rajput and Harlapur (2014 AD) reported 51.10% yield losses due to banded leaf and sheath blight (BLSB) caused by *Rhizoctonia solani* f. sp. *sasakii* Exne.

Dey et al. (2012 AD) reported the yield losses due to sorghum rust caused by *Puccinia sorghi* Schw in the range of 11.75–60.53%.

(8) In tomato/chilli

Mathur and Shekhawat (1986 AD) reported that the early blight disease causes loss from 50–86% in fruit yield.

Anonymus (2009 AD) reported that the tomato suffers with many diseases among which damping off caused by *Pythium debaryanum* and *Pythium aphanidermatum* is the most prevailing disease of tomato and is often responsible for as much as 90%mortality of seedling in susceptible plant species.

Prathibha et al., (2013 AD) reported anthracnose disease caused by *Colletotrichum* spp. is one of the major economic constraints to chilli production worldwide, especially in tropical and sub-tropical regions and is mainly a problem on mature fruits resulting in yield losses up to 50% and substantial deterioration in quality parameters.

(9) In grapes

Anonymous (2006 AD) reported yield losses in grape due to downy mildew up to 100% in major grape growing areas in Maharashtra.

Thakur and Saharan (2008 AD) estimated that post-harvest losses in grapes are about 39% of yield and 30% of value.

Rawal (2013 AD) reported the damage caused by powdery mildew is serious with 25–31% crop yield loss.

(10) In bell pepper

Anonymous (2004 AD) reported losses due to *Phytophthora* fruit rot and blight was up to 50%.

(11) In sunflower

Reddy and Usha (2001 AD) reported that some parts of India have seen epidemic proportion incidence by Tobacco Streak Virus (TSV) resulting in 6–100% loss due to sunflower necrosis.

(12) In cotton

Singh and Verma (1988 AD) reported 5–90% yield losses due to root rot disease caused by *Rhizoctonia* spp.

Mukewar et al., (1998 AD) estimated 25.49% and 13–14% yield losses due to *Alternaria macrospora* and *Colletotrichum capsici,* respectively, causing alternaria leaf spot and anthracnose disease in cotton.

Meyer et al., (2006 AD) reported 60% yield losses due to *Myrothecium roridum* pathogen causing myrothecium leaf spot in cotton.

Rakholiya and Sabalpara (2013 AD) recorded 29.1% yield losses due to *Myrothecium roridum.*

(13) In linseed

Singh et al., (2014 AD) maximum yield loss of 58.44% was recorded in cultivar Neelum followed by Parvati (55.56%), Meera (55.56%) and Chambal (51.72%), respectively while minimum loss was recorded in Kiran (19.99%) and Jeevan (22.22%) due to Alternaria blight.

(14) Red rot of sugarcane (*Glomerella tucumanensis*)

It is an important disease, particularly in sub-tropical countries. Reductions of production of over 30% have been recorded in India, but may reach upto 50% or more. In Bangladesh, 10–15% and in Pakistan, 29–75% reduction in cane weight, 30–87% loss in cane juice yield and 30–74% loss in recoverable sugar is reported.

(15) In strawberry

Wilson and Pusey (1985 AD) reported 30% yield losses due to post-harvest diseases caused by various fungal pathogens.

(16) In mango

Prabakar et al., (2005 AD) reported 25–40% yield losses due to post-harvest diseases. Among them anthracnose caused by *Colletotrichum gleosporoides* is the major one.

(17) In pomegranate

Raghuwanshi et al., (2013 AD) Bacterial blight disease of pomegranate caused by *Xanthomonas axonopodis* pv. *punicae* is reported to cause 30–50% losses on an average. However, under favourable environmental conditions for disease 80–100% losses are also reported.

8.1.3.2 In Pakistan

(1) In potato

Turkensteen (1985 AD) estimated more than 30% yield losses in potato due to bacterial wilt caused by *Pseudomonas solanacearum*.

(2) In wheat

Hussain et al., (1980 AD) stated that the national wheat yield was reduced by 10% due to the epidemic of wheat leaf rust caused by *Puccinia recondita.*

(3) In chickpea

Malik (1986 AD) estimated 46–48% yield losses due to ascochyta blight disease in chickpea.

Sattar et al., (1985 AD) estimated 10% yield losses due to *Fusarium* spp., which causes Fusarium wilt in gram.

8.1.3.3 In Bangaldesh

(1) In vegetables and fruit crops

Hossain et al., (2010 AD) estimated the average yield loss due to early blight of potato and soft rot of potato were 37% and 39%, respectively, also 43% due to early blight and 29% due to Phomopsis blight in tomato crop were reported.

8.1.3.4 In China

(1) In rice

Wang (1986 AD) reported yield losses in severely infected fields of rice crop by bacterial leaf blight disease, ranging from 10–20% caused due to *Xanthomonas oryzae.*

Wang (1986 AD) reported 40–50% yield losses in case of severe occurrence of rice blast disease caused due to *Pyricularia oryzae.*

Shen and Lin (1994 AD) reported annual losses ranging from 0.1–2.2% due to rice blast disease.

Teng et al., (1989 AD) reported 12–90% yield losses due to RTSV and also 1–40% losses associated with RTBV and 22–100% with rice ragged stunt virus.

(2) In wheat

Chen and Wang (1982 AD) studied the pathogenic species of wheat in Zhejiang Province and indicated that *Fusarium graminearum* predominated in distribution, by infecting 93.9% of the total samples and affected the production. *Fusarium graminearum* causes scab disease in wheat.

Wu and Le (1983 AD) reported 80–90% infection of common rot causing fungi such as *Bipolaris sorokiniana, Curvularia lunata, C. oryzae, Alternaria alternata, Fusarium graminearum, F. semitectum* and *Rhizoctonia* spp., which led to more yield losses.

(3) In cotton

Lu et al., (1987 AD) reported that the verticillium wilt of cotton caused due to *Verticillium dahlia* resulting in up to 15.7% yield losses.

8.1.3.5 In Philippines

(1) In rice

Olivares et al., (1980 AD) reported 30% yield losses in rice crop due to rice tungro *Bacilliform* virus, which caused tungro disease.

Hasanuddin and Hibino (1989 AD) reported grain yield reduction in the BW 272-6B, FK-135 and TN-1 plants, infected with both RTBV and RTSV or RTBV alone was >85% and also 40% in IR 36 and 20% in IR 54 due to RTSV.

(2) In coconut

Bigornia et al., (1980 AD) estimated $2.5 million yield losses due to cadang cadang disease, which is caused by viroid like pathogen.

Zelazny and Pacumbaba (1982 AD) annually estimated $ 3.5 million yield losses due to cadang cadang disease, which is caused by viroid like pathogen.

San Juan (1984 AD) constitutes an annual loss of 14.3 tonnes of copra, valued at $ 5,107 due to lethal wilt disease in coconut.

(3) In sugarcane

San Pedro and Latiza (1974 AD) recorded yield losses due to smut on varieties Phil 5333, 56226, 6019 and 56260 were 34.27%, 66.70%, 59.59% and 71.80%, respectively, caused by *Ustilago scitaminea* Syd.

TDC (1988 AD) reported yield losses i.e. 28,500 tonnes of cane due to smut disease caused by *Ustilago scitaminea* Syd. in sugarcane.

(4) In Abaca

Reinking (1955 AD) recorded 60% yield reduction i.e. fiber production due to abaca mosaic disease caused by abaca mosaic and it is the destructive disease of abaca in Philippines.

FIDA (1988 AD) also reported 3,45, 990 tonnes of fiber yield loss caused due to abaca mosaic disease because of abaca mosaic.

8.1.3.6 In Malaysia

(1) In rice

Saito et al., (1975 AD) reported that the rice blast caused by *Pyricularia oryzae*, leaf blight caused by *Xanthomonas oryzae* and sheath blight caused by *Thanatephorus oryzae* are devastating resulting in substantial yield losses.

Supaad (1989 AD) reported the highest loss in rice yield amounting to 15,482 MT of rice valued at $ 3.7 million due to PMV (Penyakit Merah Virus) or tungro caused by complex of bacilliform and spherical particle types.

8.1.3.7 In Srilanka

(1) In rice

Mithrasena et al., (1987 AD) stated that in severe cases the symptoms of sheath blight caused due to *Rhizoctonia soloni* reaching the flag leaf which may even become completely desiccated and destroyed, thereby affecting the yield considerably.

8.1.3.8 In Japan

(1) In rice

Kushibuchi (1987 AD) reported average annual losses of 2.5% due to blast disease. Otomo (1989 AD) reported about 20.6% and 54.7% yield losses in rice due to blast and sheath blight diseases, respectively.

(2) In wheat and barley

Otomo (1989 AD) recorded 1–5% yield losses due to four kinds of rusts i.e. stripe rust (*Puccinia striformis*), stem rust (*P. graminis*), leaf rust (*P. recondita*) and dwarf leaf rust (*P. hordei*) in wheat and barley. He also reported about 2% loss due to powdery mildew and Fusarium blight.

(3) In citrus

Otomo (1989 AD) recorded 10–20% yield losses in citrus due to melanose, scab and canker diseases.

(4) In apple

Otomo (1989 AD) reported yield losses due to blossom blight and alternaria leaf blotch diseases.

(5) In vegetables

Otomo (1989 AD) recorded yield losses in fruits and vegetables due to downy mildew, powdery mildew, bacterial spot and CMV, while in foliage vegetables yield loss is due to bacterial soft rot, club rot and viruses.

8.1.3.9 In Africa

(1) In cassava

CIAT (1974 AD) reported yield losses due to different wind blown pathogens, which caused leaf blight, brown leaf spot and phoma leaf spot ranging between 18.8% and 92%.

Wheatley et al., (1984 AD) reported 70% and more losses due to post-harvest root rot disease in cassava.

Lozano (1988 AD) recorded yield losses in cassava due to soil borne pathogens, which reached upto 100%.

Hahn et al., (1989 AD) reported 20–100% yield losses in cassava due to cassava bacterial blight caused by *Xanthomonas campestris,* depending on the variety, the weather conditions and the geographical location.

Lozano (1989 AD) reported more than 30% yield losses due to root rot induced by *Phytophthora drechsleri.*

(2) In starchy bananas

Hahn et al., (1989 AD) recorded losses due to black sigatoka disease that have been put between 30% and 50%.

(3) In wheat

Saadaoui (1987 AD) reported 40–50% yield losses in wheat due to leaf rust disease in wheat, which is caused by *Puccinia recondita.*

8.1.3.10 In Kenya

(1) In sugarcane

Nzioki and Jamoza (2006 AD) assessed yield loss caused by sugar cane smut infection. Results indicated yield losses of 38% on the susceptible, 17% on intermediate tolerant and 20–33% on resistant cultivars.

8.1.3.11 In Australia

(1) In sugarcane

Roach (1987 AD) reported 6.7–55.4% yield losses due to ratoon stunt diseases caused by ratoon stunt virus.

Pande (2010 AD) reported the cumulative yield losses in three legume crops viz.; chickpea, pigeonpea and groundnut to the tune of $3,038 million (Table 2) in Asia and Africa.

Tripathi (2010 AD) enumerated the yield losses due to some new emerging diseases (Table 3).

Table 2 Cummulative Yield Losses in Legume Crop in Asia and Africa.

Crops	Diseases	Causal organisms	Distributions	Yield losses ($ million)
Chickpea	Wilt	*Fusarium oxysporum* f.sp. *cicero*	Asia, Africa	218
	Ascochyta blight	*Ascochyta rabiei* (Pass.) Labr.	Asia, Africa	248
	Botrytis graymold	*Botrytis cinerea* Pres. Ex Fr.	Asia	33
Pigonpea	Fusarium wilt	Fusarium udum	Asia, Africa	193
	Sterility mosaic	Sterility mosaic virus	Asia	290
Groundnut	Foliar diseases Early leaf spot Late leaf spot Rust	Cercospora arachidicola Hori Phaeoisariopsis personata Puccinia arachnids	Asia, Africa	1392

	Aflatoxin	*Aspergillus* species	Asia, Africa	371
	Rosette/clump virus		Africa	194
	Bud necrosis virus		Asia	89

Table 3 Losses due to NewEmerging Diseases.

Diseases	Distribution and crop losses
Wheat Rust – "Ug99" Threat	Most serious threat to wheat and barley crops in 50 years and most of the world wheat is vulnerable
Scab of wheat and barley	Major threat in America. Yield losses were estimated at 95 million bushels in north Dakota
Sudden death syndrome of soybean	In the north-central U.S., estimated average annual yield losses of 175,619 tonnes occurred during 1989 AD–1991 AD
Potato early dying	In North America, yield reduction in moderately diseased fields can easily be 10–15%, and in severely diseased fields it can be as high as 30–50%
Gray leaf spot of maize	One of the most significant yield limiting diseases of maize (corn) worldwide. Yield losses due to gray leaf spot are as high as 50% in some U.S. maize fields
Soybean rust	Disease that causes serious crop losses in many parts of the world, long known to occur in Asia. Yield losses in other parts of the world due to soybean rust have been reported to range from 10–90%.
Karnal bunt of wheat	The disease is endemic in Gurdaspur, Hoshiarpur, Jalandhar and Ropar districts (the sub-mountainous tracts) of Punjab. Brennan et al., (1990 AD) estimated the economic losses from Karnal bunt of wheat in Mexico, was $7.02 million per year

9

Plant pathological societies and their development

Phytopathological societies were established in different parts of the world as a regional organisation for plant pathologists as the scientists working in this subject and the contributions made by them increased many fold, so as to discuss, deliberate and find out solutions to pathological problems at concerned regional level.

Some of the plant pathological societies or associations are as under in respective country/region.

Argentina:	Argentine Association for Crop Protection (CASAFE), Buenos Aires, Argentina.
Australia:	The Australasian Plant Pathological Society (APPS).
Brazil:	Brazilian Phytopathological Society (SBF), Sociedade Brasileira de Fitopatologia.
Canada:	Canadian Phytopathological Society (SCP), Societe Canadienne de Phytopathologie (SCP).
China:	China Society for Plant Pathology (CSPP).
Columbia:	Association for Phytopathology and related Sciences in Columbia (ASCOLFI), Association Colombiana de Fitopatologica y Ciencias Afines (ASCOLFI).
Finland:	Plant Protection Society in Finland, Kasvinsuojeluseura Finland.
France:	French Phytopathological Society (SFP), Societe Francaise de Phytopathologie (SFP).
Germany:	The German Phytomedical Society (DPG), Deutsche Phytomedizinische Gesellschaft (DPG).
India:	Indian Phytopathological Society, New Delhi.
Israel:	Israeli Phytopathological Society (IPS).
Italy:	Italian Phytopathological Society, Societe Italiana di Patologia Vegetale.

Japan:	Phytopathological Society of Japan (PPSJ).
Korea:	Korean Society of Plant Pathology (KSPP).
Latin America:	Latin American Association for Plant Pathology (ALF), Association Latinoamericana de Fitopatologia (ALF).
Malaysia:	Malaysian Plant Protection Society (MAPPS).
Mexico:	Mexican Society for Plant Pathology (SMF), Sociedad Mexicana de Fitopatologia (SMF).
Netherlands:	Royal Netherlands Society of Plant Pathology (KNPV), Koninklijke Nederlandse Planteziektenkundige Vereniging (KNPV).
Norway:	Norwegian Society for Plant Pathology (NPPF), Norsk Plantepatologisk Forening, (NPPF).
Portugal:	The Portuguese Society for Phytopathology, Sociedade Portuguesa de Fitopatologia (SPF).
South Africa:	Southern Africa Society for Plant Pathology (SASPP).
Spain:	Spanish Society of Plant Pathology (SEF), Sociedad Española de Fitopatología.
Switzerland:	Swiss Society for Phytiatry (SGP), Schweizerische Gesellschaft fur Phytomedizin (SGP).
United Kingdom:	The British Society for Plant Pathology (BSPP).
United States of America:	The American Phytopathological Society (APS).

9.1 Pathological associations and related organizations

Besides these important plant pathological societies as above several associations and organisations were founded to deal with issues of plant protection in various regions of the world. These organisations or associations with their activities are as follows:

9.1.1 American Association of Pesticide Safety Educators (AAPSE)

An association of environmental and pesticide safety educators providing science-based education programs to the public through the co-operative extension and the Land-Grant University System. **Available information**: General information on AAPSE, directories (members, working groups), contact, meetings, position papers, Journal of Pesticide Safety Education, and more.

9.1.2 Association of American Pesticide Control Officials (AAPCO) (USA)

A primary goal of AAPCO is to encourage uniformity among the states in their pesticide regulatory programs. **Available details**: mission, officers, officials, meeting details, by-laws, uniform policies, surveys, links, State's FIFRA Issues Research and Evaluation Group (SFIREG) and American Association of Pesticide Safety Educators (AAPSE).

9.1.3 Association Nationale de Protection des Plantes (ANPP)

Available details: calendar, general information, commissions, Groupe des Experts Environnement et Protection des Plantes (GEEPP), database, news, publications, methods, Phytoma (journal) and links. Site is only available in French.

9.1.4 Biologische Bundesanstalt fur Land- und Forstwirtschaft, (BBA) (Federal Biological Research Centre for Agriculture and Forestry) (Berlin and Braunschweig, Germany)

It is a research and administrative organisation attached to the Federal Ministry of Food, Agriculture and Forestry. It is engaged in plant pathology, entomology, plant protection and related fields. The BBA concerns itself with the effect of pesticides on humans, animals and the environment. The German Plant Protection Act defines the majority of its duties. **Available details**: overview, addresses, institutes, staff, laws, authorised index of plant protection products, European Commission Co-ordination (ECCO), publications, diagnosis, databases (e.g. Phytomed, BioSearch), events, flower checklist and more. Most information is available in English but there are additional or specific pages only available in German like EPPO guidelines, application forms, news among others.

9.1.5 British Crop Protection Council (BCPC)

It is a U.K. charity whose objective is to promote and encourage the science and practice of crop protection for the benefit of all. **Available information**: objectives, Board of Management, committees, BCPC Enterprises, Commercial Ventures, BCPC Corporate Members, The Brighton Conferences, addresses and contacts.

9.1.6 CAB International (CABI)

An inter-governmental organisation providing information, publishing and scientific services worldwide to agriculture, forestry, human health and the

management of natural resources. **Scientific services**: International Institutes of Entomology, Mycology, Biological Control and Parasitology.

9.1.7 Consortium for International Crop Protection (CICP)

A non-profit organisation, formed in 1978 AD by a group of U.S. universities. Its principal purpose was to assist the developing nations to reduce food crop losses caused by pests while safe-guarding the environment.

9.1.8 European Foundation for Plant Pathology (EFPP)

The EFPP promotes scientific and technical co-operation in the arena of plant health in Europe and facilitates the exchange of scientific information between plant pathologists who are members of national or regional societies in the field of plant pathology or related fields. **Available details**: events, committees, statutes, associated societies, *European Journal of Plant Pathology* and links.

9.1.9 European and Mediterranean Plant Protection Organisation (EPPO/OEPP)

An inter-governmental organisation responsible for international co-operation in plant protection in the European and Mediterranean region. **Available details**: about EPPO, EPPO meetings, EPPO news, publications and software. Publications include books (e.g. quarantine pests for Europe), the EPPO electronic documentation service (for EPPO standards on plant protection products, standards on plant quarantine, summaries of phytosanitary regulations and original texts and more). Available software: PQR – EPPO Plant Quarantine Data Retrieval System, EPPO Plant Protection Thesaurus (including the Bayer Code System). Especially the electronic documentation services is of high value for those interested in the latest developments/ documents and are recommend to use it as a resource for plant protection products, plant quarantine standards or the regular reporting services.

9.1.10 Food and Agriculture Organisation (FAO)

It is an organisation of the United Nations. A specific priority of the organisation is encouraging sustainable agriculture and rural development, a long-term strategy for the conservation and management of natural resources. **FAO webserver topics**: What is FAO?, statistical databases, agriculture, economics, fisheries, forestry, nutrition, sustainable development, programs, co-operations, publications, documentations, search the FAO website, news, global watch.

9.1.11 International Association for the Plant Protection Sciences (IAPPS)

IAPPS mission is to provide a global forum for the purpose of identifying, evaluating, integrating, and promoting plant protection concepts, technologies, and policies which are economically, environmentally, and socially acceptable. **Available details**: Synopsis of the International Plant Protection Congresses, Rationale and Objectives for IAPPS, organisational structure, IAPPS Journal, Governing Board, membership, statutes and more.

9.1.12 International Organisation for Biological and Integrated Control of noxious animals and plants (IOBC)

The following sections of IOBC provide **web services**: IOBC/NRS, Neartic Regional Section, IOBC/NRS, Biological Control of Weeds Working Group, members, announcements, projects, IOBC/NRS application, links IOBC/SEARS, South and East Asian Regional Section, working group on IPM in greenhouse crops.

9.1.13 West Palaeartic Regional Section

It is located at Eidgen assische Technische Hochscule Zurich (ETHZ), Zurich, Switzerland. **Available details**: general information, working groups, publications, addressess.

IOBC/WPRS, West Palaeartic Regional Section, working group on integrated control in cereal crops. Located at the Institute for Plant Diseases and Plant Protection, Department of Horticulture, University of Hanover, Germany. General information: contacts, meetings and abstracts of meetings.

9.1.14 International Organisation for Mycoplasmology (IOM)

IOM exists to promote the co-operative international study of mycoplasmas (Mollicutes) and mycoplasmal diseases and to disseminate knowledge about their characteristics, effects, transmission, and control. **Available details**: about IOM, history, news, newsletters, membership, jobs, IOM directory, meetings, research progammes and more.

9.1.15 The International Society for Molecular Plant–Microbe Interactions (The ISMP-MI (ISMPMInet))

Available information: Molecular Plant–Microbe Interactions Journal, directory (password access), job placement service, meetings, new books, IS-MPMI reporter, and common names for plant diseases.

9.1.16 International Society for Plant Pathology (ISPP)

The ISPP promotes the world-wide development of plant pathology and the dissemination of knowledge about plant diseases and plant health management. **Available details**: general information, news, committees, task forces, names of plant pathogenic bacteria, reports, associated societies and their contact addresses, find the committees for extension, common names of plant diseases or *Fusarium*. The task force on Global Food Security is publishing their reports here. Also available: ISPP World Directory of Plant Pathologists.

9.1.17 International Society of Regulatory Toxicology and Pharmacology (ISRTP) (Belleview, Columbia, USA)

ISRTP is dedicated to promoting education and regulatory consensus based upon sound science and open information exchange among scientists, governmental regulators, industry and the general public. **Available details**: about ISRTP, member's room (password required), international achievement award, upcoming events, the journal information and subscription (The Regulatory Toxicology and Pharmacology Journal), comments. In the editorial comments find topics like pesticides and the FQPA.

9.1.18 Mediterranean Phytopathological Union (MPU)

The Mediterranean Phytopathological Union (MPU) is a non-profit society open to organisations and individuals involved in plant pathology with a specific interest in the aspects related to the Mediterranean area considered as an ecological region. **Available details**: about MPU, join MPU, discussion page, news, links and more. Also find a link to journal of the union (Phytopathologia Mediterranea).

9.1.19 New Jersey Pest Control Association (NJPCA)

Available information: executive board, Pest-O-Gram newsletter, members, pesticide licensing exams, choosing a pest control operator, history and links.

9.1.20 New Zealand Plant Protection Society (Inc.)

NZPPS's main objective is to pool and exchange information on the biology of weeds, invertebrate and vertebrate pests, pathogens and beneficial organisms and methods for modifying their effects. **Available details**: contact, objectives, membership, scholarships, conferences, publications, online proceedings and more.

9.1.21 North American Plant Protection Organisation (NAPPO)

NAPPO is a regional plant protection organisation of FAO comprised of the National Plant Protection Organisations of Canada, the U.S., and Mexico. **Available information**: announcements (e.g. meetings), archives (see the older documents of the NAPPO), activities, associated websites, documentation (e.g. position papers), newsletters, organisation, people, list of regulated pests, standards (e.g. Pest Risk Analysis).

9.1.22 Plant Protection Society of Western Australia (Victory Park, WA, Australia)

This society is an organisation which brings together research scientists, extension officers, consultants, company representatives, farmers and other practitioners who have an interest in all aspects of plant protection. **Details available**: about the society, publications, weed science links, contact and members.

9.1.23 Welsh Pest Management Forum (WPMF)

A consortium of Welsh Companies, University research groups, users and support organisations dedicated to the development and profitability of the pest management sector in Wales and internationally. **Information you will find on this server**: Ecotoxicology Conference, forthcoming events, newsletters, staff, members, directory of pest management expertise. Links to other sites: Welsh Development Agency, Entomological Sites and more.

9.1.24 American Crop Protection Association (ACPA)

It represents the companies that produce, sell and distribute virtually all the active compounds used in crop protection chemicals registered in the U.S. **Details available**: about association, join and contact ACPA, issues, publications (e.g. safe pesticide use), links, educational material and more. The issue section includes Food Quality Protection Act, Federal Insecticide, Fungicide and Rodenticide Act or pesticide testing.

9.1.25 Arizona Crop Protection Association (AZCPA)

It is non-profit trade association committed to the continued central role of synthetic chemistry in agriculture through programs of technical information and training, issue management and environmentally sound pest control practices. **Available information**: mission, staff, pesticide training schedule, newsletters, pesticide information.

9.1.26 Asia-Pacific Crop Protection Association (APCPA)

The APCPA site is designed to provide information about APCPA and other items of interest to the Asia-Pacific agricultural community. **Available information**: background, rationale, aims and objectives, regional associations, FAO Code of Conduct, safe use product, founding members, contact, and events.

9.1.27 Association of Natural Bio-Control Producers (ANBP)

It is a non-profit corporation made up of producers, distributors and users of natural enemies of agricultural pests, as well as industry supporters, researchers and government representatives. **Available information**: membership information, newsletter, contact, meetings, links, bulletin board, find beneficial, bio control resources and more.

9.1.28 British Agrochemicals Association (BAA)

It is the U.K. trade association of companies engaged in the manufacture, formulation and national distribution of pesticide products for agriculture, forestry, horticulture, home gardening, industrial and local authority outlets. **Available information**: press releases, member companies, publications, education, grapevine and news.

9.1.29 European Crop Protection Association (ECPA) (Brussels, Belgium)

ECPA is the pan-European voice of the crop protection industry. One of ECPA's key objectives is to raise awareness of the crop protection industry's contribution to sustainable agriculture. **Details available**: ECPA (with details on the association), news, products, topics, library, site map, road map. Under topics find general issues like integrated crop management or biotechnology. The library gives access to publications and position papers. In products, get details on plant protection products (use of, production of).

9.1.30 Global Crop Protection Federation (GCPF)

It is the recognised worldwide representative of the crop protection industry. Its network comprises six regional associations in Africa/Middle-East, the Asia-Pacific region, Europe, Japan, Latin America and the U.S. One Working Group section are the Resistance Action Committees. **Available information**: general, network, publications, working groups, industry positions, safe use initiatives, links of interest. Here find under heading "The Network" information of regional branches which do not have their own homepage.

Listed are: The Africa/Middle-East Working Group, Asia-Pacific Crop Protection Association, European Crop Protection Association, Latin America Crop Protection Association and Japan Crop Protection Association.

9.1.31 Latin American Crop Protection Association (LACPA)

It is located in Zapote, San Jose, Costa Rica. LACPA represents the crop protection industry in Latin America. Actions are conducted through 17 Industry national associations and 12 research and development companies. **Details available**: general information, news, events, national associations and companies, projects, publications, toxicology centres, links and more. In product find the safe use product and the Fungicide Resistance Action Committee. Site is available in Spanish and English.

9.1.32 Mid America Crop Protection Association (MACPA)

MACPA States are: Colorado, Illinois, Indiana, Iowa, Kansas, Michigan, Minnesota, Missouri, Nebraska, North and South Dakota, Ohio and Wisconsin. **Available details**: education, news, membership national groups, state offices and more.

9.1.33 National Pest Control Association (NPCA)

It is representing pest management firms worldwide. **Get information about NCPA**: Contact, mission, news, academy, NCPA services, buyers guide, pest gazette, spotlight on a pest, pest management firms and more.

9.1.34 Southern Crop Production Association (SCPA)

SCPA is a regional trade association which provides their members with needed information and speaks for the group at state and federal levels. SCPA states are: Alabama, Arkansas, Delaware, Florida, Georgia, Kentucky, Louisiana, Maryland, Mississippi, North and South Carolina, Oklahoma, Tennessee, Texas and Virginia. **Available details**: general information, publications (e.g. Spray Drift Library), organisation, events, links, hazardous materials shipping descriptions, editorial and more.

9.1.35 UIPP French Agrochemical Association

Union des Industries de la Protection des Plantes (UIPP) main objectives are to defend industry interests, coordinate activities of their company members, inform and represent them as often as necessary. **Details available**: general

information, news, publications, links and more. Information is in French, some general information also in English.

9.1.36 United Producers Formulators & Distributors Association (UPF&DA) (Duluth, Georgia, USA)

UPF&DA represented more than 90% of all chemical distribution companies and formulators in the pest control industry. The purpose of this unity is to establish an industry position and recommend solutions to common problems. **Details available**: About UPF&DA, officers, committees, constitution, meetings, membership (addresses of companies and individuals including e-mail), events.

9.1.37 Western Crop Protection Association (WCPA)

WCPA is a non-profit trade association representing manufacturers, distributors, formulators and retailers of crop protection products and services. WCPA members are located throughout ten western states, including Arizona, California, Hawaii, Idaho, Montana, Nevada, Oregon, Utah, Washington and Wyoming. **Available details**: links, legislation, regulators issues, education, stewardship, news, FQPA, IPM, general information, membership and more. An update section features the latest news. The legislation section is sorted by state.

9.2 Phytopathological societies and their contributions

The information on some of the important phytopathological societies and their contributions are given below.

9.2.1 The American Phytopathological Society (APS)

The American Phytopathological Society (APS) is the premier society dedicated to high-quality, innovative plant pathology research. For more than a century, members of APS have been making and sharing significant breakthroughs, both for the science and society. APS is driven by a distinctive community of scientists, who ensure the global advancement of science of plant pathology.

They were the most energetic 54 people who researched much on this subject, met in December of 1908 AD to create *The American Phytopathological Society*. These scientists envisioned a society that would be of invaluable aid in promoting the future development of this important and rapidly growing

subject. Today, APS is a vibrant, flourishing society whose members have made significant and far-reaching advances in plant pathology.

APS members come from academia, government, industry, and private practice. The diversity of the members and science makes the society pertinent to a multitude of research areas, while the international involvement ensures that the latest innovations from around the world are available to all.

The American Phytopathological Society (APS) is arguably among the most robust, service-oriented, and successful scientific societies in the world, whether measured by the quality and number of its journals and non-journal publications, public service and outreach, leadership among its scientific-society peer groups, success of its annual meetings, international leadership and service, member services, member volunteerism, or financial health. With now more than 5,000 members, it may well be the most successful of all professional scientific societies of similar size in the U.S., if not the world.

The American Association for the Advancement of Science (AAAS), formed in 1848 AD, is to be credited with fostering the development of plant pathology as a science in America and for providing the leadership that led to the formation of APS. A committee of AAAS members consisting of J.C. Arthur, C.E. Bessey, W.G. Farlow, T.J. Burrill, and C.H. Peck was appointed in 1884 AD for the encouragement of research on the health and disease of plants. This AAAS committee wrote in 1885 AD to the Commissioner (now Secretary) of Agriculture urging that plant diseases be included as part of the studies of the Department's botanist. In response, a section of mycology was established within the USDA Division of Botany, with F. Lamson-Scribner as the head, starting in 1885 AD. In 1890 AD, the Commissioner of Agriculture established the Division of Vegetable Pathology with Beverly Galloway as chief. The *Journal of Mycology*, devoted at first entirely to taxonomy of fungi by the section of mycology, was taken over by the Division of Vegetable Pathology as an outlet for information on plant diseases, and as such became the first American journal for plant pathology. It was also under the auspices of AAAS that USDA plant pathologist C.L. Shear organised a meeting, during the annual meeting of AAAS held on December 30, 1908 AD in Baltimore, to discuss the formation of an independent American scientific society for plant pathologists. The first officers of APS were elected during 1908 AD organisational meeting: were President L.R. Jones, (University of Wisconsin); Vice-President A.D. Selby, (Ohio Agricultural Experiment Station); Secretary-Treasurer C.L. Shear, (USDA); and Councilors J.B.S. Norton, (University of Maryland) and B.M. Duggar, (Cornell University). The officers wrote a constitution and bylaws for the new organisation, and 130 people responded to the invitation to join APS as charter members. Membership fee were set at ¢50. That led to formation of American phytopathological society.

The first meeting of APS was held jointly with AAAS the following year, on December 30–31, 1909 AD, in Boston. Fifty members attended the meeting and 45 papers were presented. Membership fee was raised to $1. The new officers elected at the first official meeting were President: F.L. Stevens, (North Carolina State); Vice-President A.F. Woods, (University of Minnesota); Secretary–Treasurer C.L. Shear; and Councilors L.R. Jones, A.D. Selby, and H.H. Whetzel, (Cornell University); APS was incorporated under the laws of the district of Columbia on October 25, 1915 AD.

9.2.1.1 Journals of the APS

(1) *Phytopathology*

It was also decided at the first meeting of APS to start a new journal, to be named *Phytopathology*. The journal was launched as a bimonthly journal with Volume 1, Number 1, issued in February 1911 AD, subsequently *Phytopathology* became a monthly publication with the January issue of Volume 8 in 1918 AD. The first editorial board was chaired by L.R. Jones and supported by editors C.L. Shear and H.H. Whetzel, with 12 associate editors and Donald Reddick as business manager. The title of editor-in-chief was first used in 1921 AD, and the subtitle, *An International Journal,* was added in 1925 AD. A European editor was added in 1924 AD, starting with H.M. Quanjer of Wageningen, but this arrangement was discontinued in 1943 AD. The decision to charge for reprints was made at the 1950 AD annual meeting in Memphis.

(2) *Plant Disease Reporter*

The need for a second journal was felt as large number of complaints were received from members doing applied research that *Phytopathology* was not meeting their needs. President James Tammen in 1975 AD appointed a committee to study the feasibility of publishing a second journal. Coincidentally the same year USDA–ARS made an announcement that *Plant Disease Reporter* (PDR), started in 1917 AD, would be discontinued as part of a budget cut. This quickly gave way to the idea that APS might take on PDR as the long-desired second journal of the society for publication of applied research. The key element was money and Ed Kendrick was in a position to do something about this. Ed Kendrick organised a grant [from USDA–ARS] to provide APS with starting money. Also, staff was asked if they could edit and publish another monthly journal – the answer was yes, the editorial staff could beef up. This was accomplished along with a major design change orchestrated by an APS committee and staff. That grant turned out to be $2,36,000 provided in two instalments.

President Jack Schafer appointed a committee in November 1978 AD at the annual meeting in Tucson. Three new sections – an Editorial page, Features, and a Focus page on the latest in plant pathology published in other journals – were added in addition to the research articles. The name was changed to *Plant Disease* but the PDR volume numbers were continued, starting with Volume 64 in January 1980 AD. In fact, the first issue dated January 1980 AD was actually published in July 1979 AD and this allowed the society six months to market a journal that would no longer be free. Twenty thousand copies of the premier issue were mailed in July 1979 AD to members, libraries, and other potential subscribers to promote the revamped publication. At the 1979 AD annual meeting, *Plant Disease* was approved by the APS members as an official journal of the society, with Malcolm Shurtleff as the first editor-in-chief.

(3) *Molecular Plant-Microbe Interactions*

The next (third) major journal taken on by the society was *Molecular Plant-Microbe Interactions* (MPMI). MPMI was launched in 1987 AD as a new journal shared with the International Society for Molecular Plant-Microbe Interactions (IS-MPMI) and was sponsored initially by APS PRESS. George Bruening was appointed as the first editor-in-chief. Two years later, at the annual meeting in Richmond, Council approved offering MPMI as a journal option for members, appointed Luis Sequeira to succeed Bruening as editor-in-chief, and added the MPMI editor-in-chief as a member of Council. Sequeira as president of APS in 1986 AD had appointed an ad hoc committee to review the need and opportunity for a new journal. He was also a member of IS-MPMI and took the lead in obtaining the agreement that MPMI would serve as an official journal of both IS-MPMI and APS.

As a relatively new scientific society, IS-MPMI had few resources to start a journal. APS, with its publication infrastructure and healthy financial status, thus underwrote essentially all of the start-up costs for MPMI, including the design, production, promotion, marketing, and distribution. This was a strategic investment on the forefront of molecular plant pathology as an emerging field of science, and it addressed the need for APS to provide young scientists with another reason to join the society. Sharing this journal also headed-off the possibility that IS-MPMI might find another way to start a competing journal. However, while editorial and financial responsibilities for MPMI were transferred to and assumed by a joint committee of APS and IS-MPMI in 1990 AD, it was not until 1992 AD that an agreement on sharing of revenue was reached, when the IS-MPMI Executive Committee accepted an offer of royalty payments from APS based on the number of personal and library subscriptions to the journal.

9.2.1.2 Electronic and online publications

In early 1990 AD, it was clear that to remain competitive as a publishing house, APS would need to make a successful transition from providing exclusively hard-copy journals and books sent through the mail and stored on shelves to embrace entirely electronic methods of journal and book production, dissemination, and storage. In 1992 AD, council appointed an ad hoc committee, called the Electronic Technology Committee, chaired by Larry Moore at Oregon State University to aid staff in identifying the best technology for use at headquarters. This led, in 1994 AD, to the launch of the highly successful APSnet which was introduced for the membership at the 86th annual meeting in Albuquerque. One outgrowth of this effort was a decision to convert to all-electronic handling of *Plant Disease Notes*. In 1993 AD, J.D. MacDonald was appointed the first assigning editor of the *Notes* and charged with developing the procedures for e-mail-based submission, review, editing and final acceptance——a mission that was a challenge when e-mail was still in its infancy. As the number of electronic initiatives grew, President Sue Tolin, in 1995 AD, repositioned the ad hoc committee, forming instead a standing committee of the society, called the Electronic Technology Advisory Committee (ETAC) and chaired by MacDonald. The ETAC was charged to advise Council, journal editors and APS staff of opportunities to better serve members through electronic communications/publications. Again, in 1997 AD, as the number of initiatives and policy complexities continued to expand, council approved a further reorganisation that created the Office of Electronic Communications (OEC) with MacDonald as the first director, and ETAC became a committee under OEC.

9.2.1.3 Introduction of online journals

APS launched two online peer-reviewed publications in 2000 AD: *Plant Health Progress* (PHP), with Tim Murray as its first editor-in-chief, and *The Plant Health Instructor* (PHI), focusing on instructional materials and scholarship in teaching, with editor-in-chief Gail Schumann. PHP also then became the founding publication for the online Plant Management Network (PMN), developed in partnership with the American Society of Agronomy and the Crop Science Society of America, and in co-operation with the Entomological Society of America, the Society of Nematologists, the Weed Science Society of America, and other related societies. This is another example of APS working with its peer group of professional scientific societies.

9.2.2 Indian phytopathological society

The increasing importance of plant pathology led to the foundation of the Indian Phytopathological Society in 1947 AD by B.B. Mundkur under the

chairmanship of S.R. Bose with 20 members and started the Journal *Indian Phytopathology*. The Indian Phytopathological Society (IPS) is a professional forum for promoting the cause of science of Phytopathology. It is the third largest society of plant pathologists in the world devoted to the study of plant diseases and their control. The society provides information on the latest developments and research advances in plant health science through its journals and participates in the exchange of plant health information with public policy-makers, and the larger scientific community; and provides opportunities for scientific communication, collaboration, and professional development. The society focuses on the field of Mycology, Fungal Pathology, Bacteriology, Virology, Phytoplasmology and Nematology. It provides a forum to the scientists to interact on important issues of Plant Pathological research, education and extension. The society organises National and International conferences, symposia and seminars on major topics of Plant Pathology. It has nearly 2,000 members who are located in more than 50 countries. These include research scientists, teachers, extension professionals, students, private consultants, administrators, technicians, agricultural field representatives, and pest management personnel. Their professions vary, but they have one common goal – to promote knowledge of plant diseases and their control.

Following awards are presented by Indian phytopathological society on the occasion of their annual meeting and National symposium.

- Mundkar memorial lecture award, (annual)
- Jeersannidhi award lecture (one in three years)
- Professor S.N. Dasgupta memorial lecture (once in 3 years)
- Professor M.S. Pavgi award lecture (once in 3 years)
- K.C. Mehta and Manoranjan Mitra Award (annual)
- A.P. Mishra life time achievement award (Perennial)
- Professor M.K. Patel memorial young scientist award (annual)
- Professor A.K. Sarbhoy memorial lecture award (Priennial)
- Professor J.P. Verma memorial lecture award (Priennial)
- Sharda Lele memorial award (annual)
- S.P. Raychaudhari memorial lecture award (annual)
- M.J. Narasmhan academic merit award contest
- M.J. Narasimhan medal award for best research paper
- IPS travel sponsorship for young scientist

The journal, *Indian Phytopathology* is published quarterly in March, June, September and December.

ISSN : 0367-973(print version);

ISSN : 2248-9800 (electronic version).

9.2.3 Indian society of mycology and plant pathology

Later on *Indian Society of Mycology and Plant Pathology* was established in 1970 AD in Udaipur under the guidance of Dr. N. Prasad and Dr. R. Prasada. The first issue of the *Indian Journal of Mycology and Plant Pathology* was released in December 1971 AD. The society has more than 1,000 members which include annual members, life members and foreign members, institutions and libraries.

Following awards are presented by Indian Society of Mycology and Plant Pathology yearly on the occasion of their annual meeting and national symposium.

- Professor H.C. Dube outstanding young scientist award
- Professor K.S. Bilgrami award for best poster in annual symposium
- P.P. Singhal memorial pesticide India award
- Smt. Guman Devi Verma memorial best woman scientist award
- P.R. Verma M.Sc., and Ph.D. awards
- Y.L. Nene outstanding plant pathology teacher award
- Professor V.P. Bhide memorial award lecture

The journal, Indian *Journal of Mycology and Plant Pathology* is published quarterly in March, June, September and December.

9.2.4 The Chinese Society for Plant Pathology (CSPP)

The Chinese Society for Plant Pathology (CSPP) was established in China in 1929 AD to promote the development of plant pathology. Over the years, the organisation has grown into a national first-class society, with 14 professional committees, five working committees, 26 local committees and more than 6,500 members from China and abroad.

The CSPP became a member of the International Society for Plant Pathology in 1983 AD and is one of the fundamental members of the Asian Association of Societies for Plant Pathology. It's headquarter is located in the campus of China Agricultural University. The CSPP journal *Acta Phytopathologica Sinica* was initiated in 1955 AD. The journal is published bimonthly in Chinese or

English covering fundamental and application aspects of plant pathology. As an indicator of the academic level of CSPP, *Acta Phytopathologica Sinica* is one of the most highly rated academic journals in China. CSPP has carried out a great deal of application research related to the prevention of plant diseases. The scientific development of plant pathology is the main concern of CSPP. The society sponsored the First Asian Plant Pathology Conference in Beijing in 2000 AD and co-organised the 15th International Plant Protection Congress in 2004 AD. It also organised 10th International Congress of Plant Pathology during 25–30 August 2013 AD at Beijing, China.

9.2.5 The Bristish Society for Plant Pathology (BSPP)

The British Society for Plant Pathology (BSPP) was founded in 1981 AD for the study and advancement of plant pathology. The BSPP welcomes members from all over the world and from all branches of plant pathology. It supports the professional interests of plant pathologists worldwide and provides information and communicates with its members via a newsletter, website and annual meeting. The society organises regular scientific meetings, edits three international pathology journals and makes funds available to members for both travel expenses and short-term undergraduate and masters level studentships.

The society organises regular scientific meetings, often with other societies, to cover topics relevant to plant pathology. Members pay reduced conference fees. Meetings include paper readings and poster sessions, discussion forums, workshops on specialist topics and visits to research establishments. Young members are particularly encouraged to contribute. The P.H. Gregory Prize is awarded annually for the best presentation made by a young scientist who has not previously presented a paper at a BSPP meeting. The J Colhoun Prize is awarded annually for the best poster presentation made by a young scientist who has not previously presented a paper at a BSPP meeting. The Garrett Lecture was inaugurated in 1993 AD, and is given at a meeting each year by a plant pathologist from overseas.

There are reduced membership fees for students and the unsalaried members. One can have online access to both BSPP journals, receive a regular newsletter with updates on society's activities, information on meetings, etc. One can attend BSPP scientific meetings and other functions at members rates, apply for travel and study grants and have access to the members.

9.2.5.1 BSPP funds and studentships

- Travel funds provide financial assistance to members wishing to attend a plant pathology conference in the U.K. or overseas or make short study visits abroad.

- Visiting fellowships are for members to undertake a longer period of study at another institute.
- Student vacation and M.Sc. bursaries allow undergraduate or M.Sc. students to undertake research projects in appropriate plant pathology laboratories.
- The plant pathology promotion fund supports new initiatives with potential to further the promotion of plant pathology in the U.K. and elsewhere.

BSPP Newsletters are sent to all members three times a year providing information on society's activities.

A searchable online database is available to members.

BSPP Presidents and Honorary members whose contribution to the society and the discipline of plant pathology has been recognised are listed on the BSPP website.

9.2.5.2 BSPP journals

- *Plant Pathology* is an international journal that publishes research papers and critical reviews on all aspects of plant pathology.
- *Molecular Plant Pathology* is a an international journal that has a special interest in publishing papers that emphasise molecular analysis of pathogens, determinants affecting host response to plant pathogens or their interaction.
- *New Disease Reports* is an on-line global reporting service for rapid and cost-free new publication of new and significant plant disease situations. Papers are published rapidly on the BSPP web site and biannually in the journal Plant Pathology.

9.2.6 The Mediterranean Phytopathological Union (MPU)

It is a non-profit society open to organisations and individuals involved in plant pathology with a specific interest in the aspects related to the Mediterranean area considered as an ecological region. The Union was created with the aim of stimulating contacts among plant pathologists and facilitating the spread of information, news and scientific material on plant diseases occurring in the area. The Union also intends to facilitate and promote studies and research on diseases of Mediterranean crops and their control. The Union hosts scientific papers dealing with problems related to diseases of Mediterranean crops in a journal *Phytopathologia Mediterranea*, specifically devoted to these aspects of plant pathology. The Union is affiliated to the International Society for Plant Pathology (ISPP).

9.2.7 The Canadian Phytopathological Society (CPS)

CPS was formed in 1929 AD as a federal, non-profit organisation and is now more than 85-years-old. Prior to this, the society had functioned as a division of the American Phytopathological Society. CPS has more than 400 members in Canada and abroad. Its membership has expertise in all facets of plant pathology.

The officers are the President, President-Elect, Vice-President, Secretary, Treasurer, and Membership Secretary. The affairs of the society are managed by a board which consists of the officers, the immediate past President and two Directors. In addition, there are standing, subject matter and ad hoc committees for program, awards, nominations, membership, resolutions, future meetings, and those dealing with other society matters. The society has nine regional and society associations.

The society publishes a journal known as *Canadian Journal of Plant Pathology*.

9.2.8 European Foundation for Plant Pathology (EFPP)

The European Foundation for Plant Pathology is located at Wageningen, The Netherlands.

The foundation promote scientific and technical co-operation in the field of plant pathology in Europe and for such purposes to facilitate the exchange of scientific information between plant pathologists, especially European plant pathologists, who are members of national or regional scientific organisations in the field of plant pathology or related fields.

The foundation organises meetings, symposia and conferences; collects and disseminates information; advises, collaborates and maintains liaison with relevant national and international organisations ; encourages formation of national societies of plant pathologists within Europe and of international groups of plant pathologists specialised in certain fields, undertakes any lawful activity in pursuance of the object of the foundation and remains a scientific organisation not engaged in any form of political activity and without the objective of financial gain.

The foundation publishes *European Journal of Plant Pathology*:

The affairs of the foundation are conducted by a board. Board members are nominated by phytopathological associations and societies and other societies with a predominantly phytopathological interest in the European area (hereafter referred to as member societies). Board members must be residents of Europe. Admissions of societies to the foundation shall be decided by the board provided that the society making the application has submitted

sufficient documentation of its activities to the General Secretary at least three months before the board meeting at which a decision on the application is to be made. Any member society can withdraw from the federation, provided that notice of such an intention has been communicated to the Secretary General at least three months before the board meeting at which a decision on the withdrawal application is to be made. Each member society, which is contributing annually an agreed sum of money to the foundation, has the right to nominate one or more delegates to the board, as defined in the internal regulations. If a delegate, nominated by a society, cannot attend a meeting of the board, that society may appoint a deputy. The board has the right to co-opt to the board one additional member for a term of three years. The co-opted member has the right to vote and shall be eligible for a position in the executive committee. The procedures for meetings of the board and for voting are defined in the internal regulations.

The board shall elect an executive committee (EXCOM), comprising at least a president, two vice-presidents, a secretary general, a treasurer and a meeting secretary. The procedures for meetings, voting and reelection, and the term of office of EXCOM officers are defined in the internal regulations. The EXCOM controls the finances and administration, and carries into effect the decisions of the board. It can take and carry out its own decisions within the limits of the policy of the foundation and of the budget approved by the board. The EXCOM shall report on the activities and on the finances to the board. The EXCOM can take decisions provided that the president and at least two members are present. The president together with the secretary general represents the foundation in legal and other matters. If for any reason one of them is unable to act, he shall be represented by the treasurer.

The funds of the foundation come from member societies supporting organisations and government's gifts, legacies and grants and other sources. Portion of the funds of the foundation can be transferred to, and reversely obtained from, the treasurer of a committee for the organisation of a scientific meeting, symposium or conference, or for any other activity, provided that these activities are organised under the auspices of the foundation. No portion of the funds shall be transferred, directly or indirectly, to member of the board or to organisations which they represent, except in good faith as reasonable and proper remuneration to any board member, officer or servant of the foundation in return for any services actually rendered to the federation.

The financial year begins on January 1st and ends on December 31st. All expenditure on behalf of the foundation shall be allowed only in accordance with the annual budget approved by the EXCOM. Other supplementary expenditure shall only be possible by permission, in writing, of the treasurer and the president and shall be explained at the following meeting of the EXCOM and board. The treasurer shall produce an annual financial statement and budget which

will be offered to the EXCOM and board. The audit of the finances of the foundation shall be conducted annually by two auditors, elected by the board, who may ask the assistance of a chartered accountant. The authors shall report to the EXCOM and to the board.

Scientific meetings of a general character (conferences) shall be held once every two years (not in the years that congresses of the International Society of Plant Pathology takes place) at places chosen by the board. Such a conference shall be organised by the member society (societies) of the country in which the chosen place is located. The general plan of the conference shall be submitted to the EXCOM for approval. Financial responsibility for the conference shall rest with the member society of the host country and not with the foundation. Any surplus assets derived from a conference will be made available to the foundation as a start for the organisation of the following conference. Meetings of a more specialised character (symposia, discussion groups, working groups) may be organised, when and where regarded to be useful, under the auspices of the foundation, provided approval of the EXCOM is obtained.

Member societies and affiliated non-member organisations will submit to the secretary general fully detailed documentation of national and local scientific meetings in the field of plant pathology in a broad sense. The secretary general shall collect this and other relevant information in a quarterly information bulletin and send it to member societies, affiliated non-member organisations and other supporting organisations. The foundation may also issue other publications, the financing and distribution shall be decided by the board.

The European Foundation for Plant Pathology offers:

- Easy access to the national Societies of plant pathology (in Europe)
- Subscription at a reduced membership rate to the European Journal of Plant Pathology
- An EFPP Conference every three years

9.2.9 Australasian Plant Pathology Society (APPS)

The Australasian Plant Pathology Society is dedicated to the advancement and dissemination of knowledge of plant pathology and its practice in Australasia. Australasia is interpreted in the broadest sense to include not only Australia, New Zealand and Papua New Guinea, but also the Indian, Pacific and Asian regions. Although the society's activities are mainly focused on the Australasian region, many of the activities of their members are of international importance and significance.

The society was founded in 1969 AD. The members of APPS represent a broad range of scientific interests, including research scientists, teachers, students, extension professionals, administrators, industry and pest management personnel.

Each member of APPS is an associate member of the International Society for Plant Pathology and is included in the ISPP mailing list. Through the International Society, APPS is a member of the International Union of Biological Sciences (IUBS), the International Union of Microbiological Societies (IUMS), in liaison with the UN Food and Agriculture Organisation (FAO), and the International Council for Science.

APPS is also a member of the Asian Association of Societies for Plant Pathology and has a formal linkage with the Phytopathological Society of Japan.

9.2.10 New Zealand Plant Protection Society (Inc.) (NZPPS)

The New Zealand Plant Protection Society is open to all groups and individuals with an interest in all aspects of biology, ecology and control of weeds, vertebrate and invertebrate pests, and pathogens and beneficial micro-organisms in agriculture, horticulture, forestry and natural eco-systems. The scope of relevant activity includes everything from pre-border evaluation of biosecurity risks, through eradication of newly-established invaders, to the management and control of well-established weeds, pests and diseases of pasture, arable and horticultural crops, and conservation.

Members include scientists, commercial company representatives, growers, foresters, and regional and local government. All are interested in sharing information and promoting plant protection in New Zealand.

The objectives of the society are:

1. To pool and exchange information on the biology of weeds, invertebrate and vertebrate pests, pathogens and beneficial organisms and methods for modifying their effects.
2. To hold an annual conference and publish a scientific journal.
3. To administer trust funds for the furtherance of plant protection science, education and extension in New Zealand.
4. To affiliate with similar societies throughout the world.

New Zealand Plant Protection is the journal of the New Zealand Plant Protection Society. It publishes original research papers on all aspects of biology, ecology and control of weeds, vertebrate and invertebrate pests, and pathogens and beneficial micro-organisms in agriculture, horticulture,

forestry and natural ecosystems. A summary or a selected aspect of every paper published in the journal is presented orally at the Society's Conference.

9.2.11 Phytopathological Society of Japan

Following the revolution of 1866 AD, the Japanese government in its efforts to introduce western practices, invited European and American professors in all branches of science and the arts to Japan. Plant pathology was mainly introduced through Tokyo and Sapporo in Hokkaido, the northernmost island of Japan. The first agricultural experiment station was founded at Nishigahara, Tokyo in 1893 AD and this, became the present National Institute of Agricultural Sciences in 1950 AD. The Hokkaido Agricultural Experiment Station was established in 1901 AD at Sapporo where there had been the agricultural college (the By Sapporo Nagakko) since 1876 AD organised by President W.S. Clark and Professor W.P. Brooks of the Massachusetts Agricultural College and where the latter lectured on plant pathology for 12 years. Both these stations have divisions for plant pathology and entomology.

During 1902 AD–1924 AD, the Ministry of Education promoted a plan to establish colleges for agriculture and forestry or horticulture in the cities of Morioka (Iwate prefecture), Tottori, Tsu (Mie prefecture), Utsunomiya (Tochigi prefecture), Gifu, Miyazaki, and Matsudo (Chiba prefecture). All these colleges were raised to the status of university after the Second World War.

At the turn of the century additional research stations were established for tea (1896 AD) and horticulture (1902 AD) and other regional stations have been started since, all of which focused attention to disease in plants. There has been a Forest Experiment Station at Tokyo since 1905 AD (with nine substations in 1966 AD).

The Phytopathological Society (Nihon Shokubutsu Byorigaku-Kai) was established in February 1916 AD, and Mitsutaro Shirai was recommended as the first president.

A chair for plant pathology was provided in Kyoto Imperial University in 1924 AD, with **Takewo Hemmi** as professor.

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