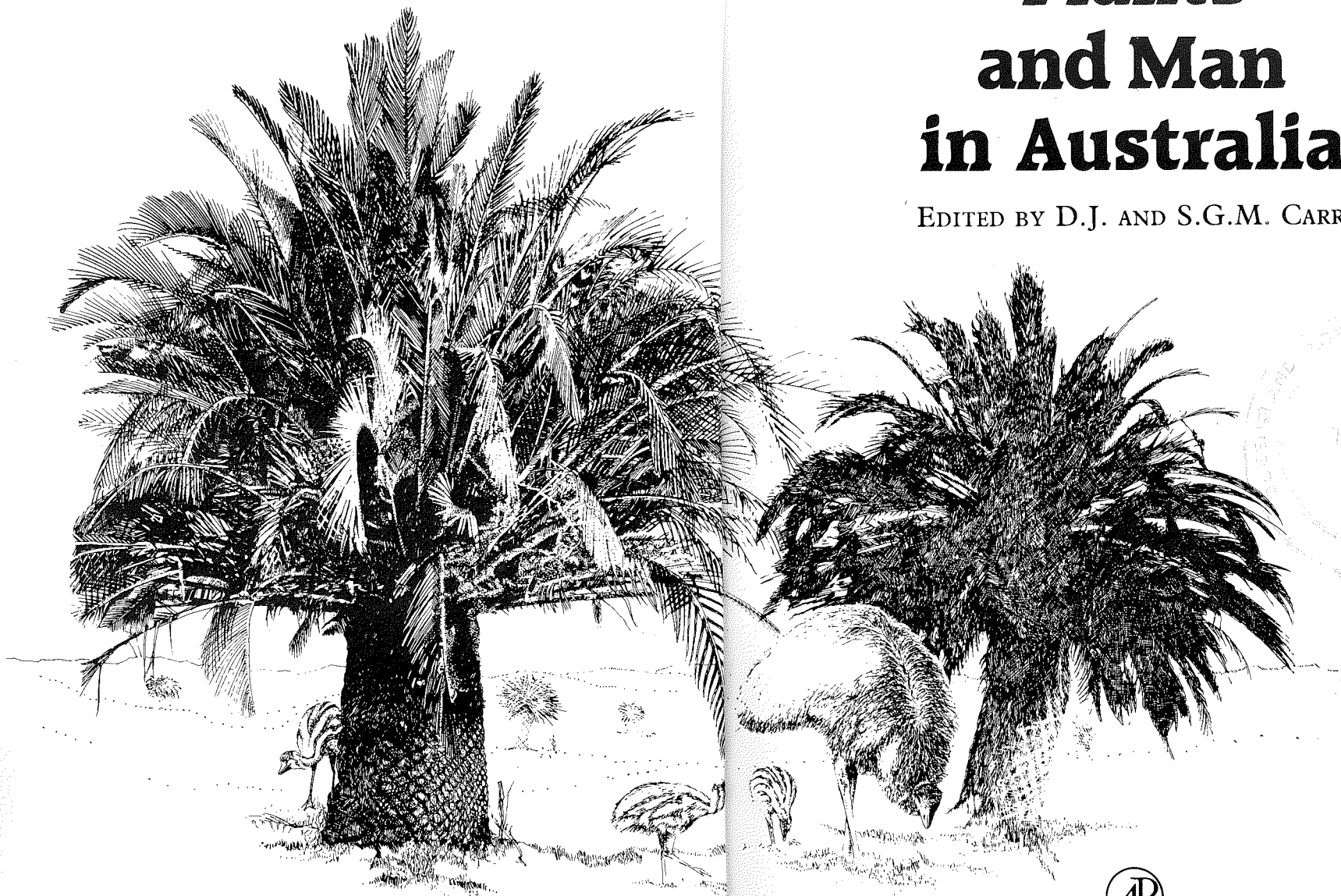


Plants and Man in Australia

EDITED BY D.J. AND S.G.M. CARR



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Contents

Preface xi

'Australia', A.D. Hope

Old homestead, Tammit Station, Euston, N.S.W., J.S. Turner

Nineteenth century plant physiology observations 3

D.J. & S.G.M. Carr

The development of plant physiology in Australia 10

J.S. Turner

Australia and trace elements 35

G.W. Leeper

A history of plant pathology in Australia 42

Neville H. White

Wheat breeding in Australia 96

C.W. Wrigley and A. Rathjen

The fuss about coal: Troubled relations between palaeobotany and geology 136

T.G. Vallance

'Pastoral', James McAuley

Baobabs, (Adansonia gregorii F. Muell.) from G. Bennett, Gatherings of a naturalist, London 1860.

The history of introduced weeds 179

W.J. Parsons

Prickly pear	194
C.B. Osmond and J. Monro	
The history of poisonous plants in Australia	223
Selwyn L. Everist	
'A-going for greens'	256
Bryan Gandevia	
Soap and society	266
D.J. & S.G.M. Carr	
Barilla production in Australia	274
Juliet F. Bird	
Barilla production and early soap making in Tasmania	281
John S. Whinray	
Index to Organisms	297
General Index	304

People and Plants in Australia

Edited by D.J. & S.G.M. Carr

A companion volume to Plants and Man in Australia

The botany of the first Australians	3
D.J. and S.G.M. Carr	
Extra-tropical Western Australia	47
L. Diels, translated by D.J. Carr	
A charmed life	
The collections of Labillardiere	79
D.J. and S.G.M. Carr	
Australian Phycology: the German influence	116
Sophie C. Ducker	
Botany of the Flinders voyage	139
Phyllis Edwards	
Karl Goebel in Australia and New Zealand	167
D.J. and S.G.M. Carr	
Botany and Society in Eastern Australia	183
Michael Hoare	
Plants, politics and personalities in Colonial New South Wales	220
Lionel A. Gilbert	
Botany and Society in the Swan River Colony	259
M.J. Hamersley	
James Drummond and the Royal Cork Institution	280
D.J. Carr and S.G.M. Carr	
'Rain Follows the Plough'	288
D.J. & S.G.M. Carr	
Goyder's Line — a Line for all Seasons?	295
R.A. Heathcote	
The contribution of women to Australian Botany	325
D.J. Carr	

Collectors and Illustrators: Women botanists of the nineteenth century 333

Ann Moyal

Augusta and Mrs Molloy 357

Marnie Bassett

The remarkable Mrs Clemens 374

R.F. Langdon

Ightham Station: on the trail of Mary S. Clemens 384

S.G.M. Carr

Jessie Louisa Hussey 388

D.N. Kraehenbuehl

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Preface

In the Preface to the companion volume, "People and Plants in Australia", we arbitrarily divided the history of the relationship between plants and people in Australia into six overlapping periods, from early discovery to the present day. In this volume we are concerned mainly with the history of the development of disciplines related to problem solving and with some aspects of plant introduction and utilisation.

Problem solving implies first, recognition of the nature of the problem, then its further study in the light of experience to find means of overcoming it. Good examples are provided by the early history of poisonous plants, dealt with by Everist in this book. Reports of mysterious stock diseases and deaths led finally to suspicion falling on plants consumed by animals. The identification of the poisonous plants themselves and their toxic principles required expert, usually professional skills. Similarly, the identification of the syndrome of symptoms of trace element deficiencies in plants required the application of chemical skills and expertise in soil science. These skills were lacking in Australia until late in the nineteenth century. The training of Australians as chemists had to wait upon the founding of the first Universities in mid century. No professional botanists were trained here until the twentieth century.

Many problems arose from the main primary industry based on plants, wheat growing. There were problems of yield, baking quality and disease resistance. They required specialised knowledge not available either to the amateur natural historians who had played so great a part in the earlier development of the plant sciences in Australia or to the professional Government botanists, whose *forte* was largely in taxonomy. It is appropriate that the history of wheat breeding and the study of the pathogens causing disease in wheat is outlined in two chapters of this book. That work continues into an indefinite future. To

A History of plant pathology in Australia

NEVILLE H. WHITE

Introduction

The history of plant pathology in Australia was greatly influenced by the fact that its unique flora does not contain any food species of the quality found in other parts of the world. This necessitated the introduction of food and pasture plants from other countries when the continent was colonised by Europeans in 1788. The history of plant pathology in Australia is therefore mainly concerned with the diseases of the introduced cultivated species brought from different countries and grown in soil never before cultivated.[1] Obviously, plant disease problems in Australia have been closely linked with agricultural pursuits since its colonisation.

The geographic isolation of the Australian continent, surrounded as it is by oceans acting as a natural barrier to the invasion of many pathogens, both before and after European settlement, also influenced the history of plant pathology in Australia. These natural barriers were breached during the first hundred years of colonisation through the prevailing ignorance about the causes of infectious plant diseases. The only restrictions on the entry of pathogens were those associated with survival of inocula during long sea voyages through the tropics, and with the survival and spread of inocula under the Australian climatic conditions. Some introduced crop species remained free from diseases which affected them in their country of origin for these reasons.[2] Seed and clones of introduced crop plants were the main vehicles for the introduction of plant pathogens which became naturalised in their new environment as the culture of the crops they affected intensified and became widespread.

In any geographic area, the presence of a plant pathogen is usually revealed as an epidemic of disease that occurs when environmental conditions are optimal for a rapid build-up and spread of inocula of the pathogen. In newly-colonised regions like Australia, epidemics of

disease in introduced plant species are caused by the naturalised pathogens brought there from other regions where they were endemic, and by indigenous pathogens present in the native flora and in the soil.

Although most of the diseases of cultivated plants in Australia are caused by introduced pathogens, there are some that result from indigenous plant pathogens moving from the native flora on to introduced plants belonging to the same taxon as the native hosts.[3] Obviously, native plants are affected with diseases caused by endemic pathogens that are frequently as unique as their native hosts.[4] These, and similar native pathogens with highly specialised host relationships have not moved on to introduced plant species. However, some of the introduced pathogens have infected native plants belonging to the same taxon as the introduced hosts.[5]

Some pathogens that are world-wide in distribution apparently are also indigenous in Australia which suggest that they have been in existence for a very long time.[6] This applies particularly to many of the soil-borne root pathogens occurring on native and introduced species in Australia. They occur in virgin land too remote from cultivation, and over distances too vast for them to have been introduced by man, and spread by natural means since Australia was settled by Europeans. They include the root-rot fungi of cereals and forest trees, the root pathogenic nematodes, some air-borne fungi like *Glomerella cingulata*[7] and some insect-borne viruses such as barley yellow dwarf.

It was imperative that the new colony became self-supporting because of the lengthy and hazardous sea voyages for the transport of food to it. Almost from the start, attempts at growing food crops in Australia were plagued with diseases referred to as 'blights' in the early records.[8] By the 1880s, epidemics of plant disease in many crops grown in Australia were resulting in serious losses in production, and causing concern amongst growers and responsible Governments in the various States. Virtually no rational attempts to control plant diseases were made in those days, except for the use of various wet steepes in the case of externally seed-borne smuts of cereals. Australia's progress towards becoming one of the wheat-producing countries of the world was threatened by the uncertainty of the harvests, mainly through the ravages of stem rust. There had been ten seasons when wheat crops had failed completely through stem rust.[9]

Public concern over the increasing epidemics of crop diseases and the development of knowledge of pathogenic causes of plant disease,

induced Government authorities to seek expert advice from scientifically trained personnel rather than from merely practical advisers, as had been the practice until then.

Recruitment and training of plant pathologists

At that time there was a scarcity everywhere in the world of scientifically trained people able to act as vegetable pathologists, as they were then called. Few had had formal biological training at colleges or universities; more often they were naturalists who could apply their general knowledge to plant disease problems. The worst epidemic of wheat stem rust ever experienced in Australia, that of the 1889 season, precipitated the first government appointments of vegetable pathologists in 1890 in the two most populous and productive States, N.S.W. and Victoria. By mere chance and good fortune, there was a dedicated and well-qualified biologist in both Sydney and Melbourne at that time. In Sydney, there was Dr Nathan Cobb (fig. 1), a 31-year-old American from Massachusetts, who had arrived early in 1889, seeking the adventures of the 'biological wonderland' of Australia. He was on his way back to America after a year's study of nematodes at Jena University. In Melbourne there was Daniel McAlpine (fig. 2), the 41-year-old Scot, who had arrived in 1884 from Edinburgh where he had been, for seven years, lecturer in Natural History at the Veterinary College and at the Heriot-Watt College. McAlpine was lecturer in biology at Ormond College in Melbourne University.

When Cobb and McAlpine were appointed as experts in plant pathology ('Vegetable Pathologists') in 1890, they faced the formidable task of pioneering plant pathology in Australia with the sole aid of self-found accommodation and equipment (including a microscope). Their respective governments paid only their salary. For the first 16 years McAlpine used rooms in his home at 22 Armadale Street, Armadale, a suburb of Melbourne (fig. 3).

For 26 years, McAlpine served the needs not only of his own State of Victoria but also the whole of Australia, especially those States without experts in plant pathology.[10] He certainly deserves the title of 'father of plant pathology in Australia'. He is commemorated in an oration of the Australian Plant Pathology Society. In a similar way, Dr Cobb gave his services to N.S.W.[10a] and other States that needed his help until 1905, when he returned to his homeland in U.S.A. and to his first love, Nematology, in founding the Division of Nematology in the US Bureau of Agriculture in Washington, D.C. McAlpine and Cobb, prolific

writers, nearly always illustrated their published works, McAlpine mainly with photographs, and Cobb with drawings, using skills at which both excelled. They published in the journals of the State Departments of Agriculture, as well as in the publications of the local Learned Societies. It is salutary to note in perusing these journals, that there are few fungal diseases in Australia today that were not reported originally by McAlpine and Cobb.[11]

Laboratory equipment associated with the culturing of plant pathogenic fungi and bacteria on artificial media came gradually into use in plant pathological laboratories during the first decade of this century. Using these techniques in 1895, Cobb was one of the first to show that bacteria can be causes of plant disease. He proved that gumming disease of sugarcane was caused by a bacterium at a time when T.J. Burrell and Erwin F. Smith were pioneering work on bacterial plant pathogens in U.S.A.[12]

The problem of recruitment of agricultural scientists, including plant pathologists, led to the establishment in the early 1900s of Faculties of Agriculture in the existing Universities of Adelaide, Melbourne, and Sydney. The first Chair of Agriculture to be filled was in Sydney in 1910. The appointee was Professor R.D. Watt, a graduate in Agricultural Science from Glasgow. Watt lectured on all aspects of agricultural science to a small group of students in 1912, and included 5 lectures on plant pathology, the first ever given at an Australian University. The textbook he prescribed to augment his lectures, B.M. Duggar's *Fungous Diseases of Plants*, published in U.S.A. in 1909, could not have been bettered at that time.[13]

The advent of the First World War in August 1914 halted the early beginnings of professional training in plant pathology, and most of the first Agricultural graduates from Sydney in 1914 and 1915 enlisted and saw active service. Following the Armistice in 1918, there was a rapid development in undergraduate training of plant pathologists by Universities in Australia. By the 1930s, universities in all States, except Tasmania, provided such training either in Schools of Agriculture, or in Departments of Botany. Because of the remoteness of Australia from centres of plant pathological progress in Northern Hemisphere countries, graduates opting to become plant pathologists sought overseas experience by visiting or studying at such centres. In fact, until the late 1940s, there was a general attitude in the profession that the training of Australian plant pathologists was not complete without overseas experience.



Figure 1. Dr N.A. Cobb.

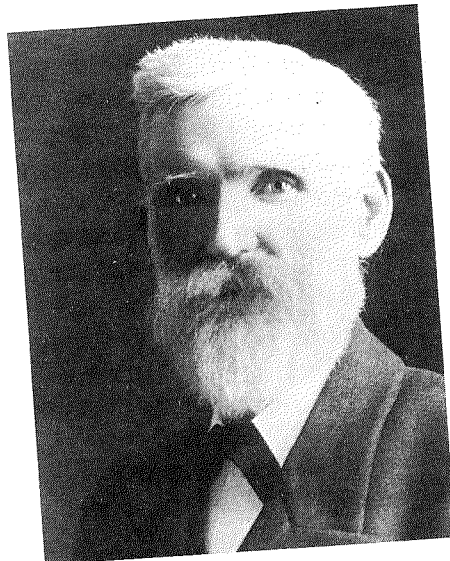


Figure 2. Daniel McAlpine.

At Melbourne University in 1920, Dr Ethel McLennan began teaching plant pathology, as part of her mycology course in the School of Botany, to undergraduates in Science and Agriculture. She had graduated in Science from Melbourne in 1915 with no formal training in mycology or plant pathology, but through contact with McAlpine and Brittlebank[120] had developed an interest in, and was self-taught in, this aspect of botany. Devising her own course, she was eminently successful as a teacher of mycology and plant pathology, inspiring many of her students to become plant pathologists, some of whom became leaders in the profession.[14]

The first professional course in plant pathology, as distinct from applied mycology, was given by W.L. Waterhouse in 1922 to undergraduates taking the degree in Agriculture at Sydney



Figure 3. The first plant pathology laboratory in Australia; MacAlpine's private home in 22 Armadale Street, Armadale, Victoria.

University.[15] Waterhouse was one of Watt's first batch of students to graduate in 1914[16]; instead of accepting the award of the 1851 Exhibition to study at Cambridge, he enlisted in the A.I.F. After recovering from war injuries in 1919, he went to Imperial College, London, on a Walter and Eliza Hall Fellowship. He studied for a year under Sir John Farmer and gained the Diploma of the College. He then visited American Universities at which plant pathology was taught, spending most of his time with E.C. Stakman at Minnesota. On his return to Sydney in 1921, Waterhouse was appointed lecturer in plant pathology, genetics and plant breeding, and agricultural botany in the Faculty of Agriculture at Sydney University.[17]

Over the next 30 years, Waterhouse established a tradition of sound undergraduate training in plant pathology at Sydney, inspiring many students to become plant pathologists, and in the early years he virtually was the Faculty. McLennan and Waterhouse were certainly pioneers in the teaching of plant pathology in Australia. Plant pathology was taught in other States of Australia either as part of a mycology course in Botany Schools, or as courses given by visiting lecturers recruited from among Government plant pathologists, like that instituted by W.M. Carne in Western Australia in 1923. The courses taught by part-time lecturers were, at best, servicing courses with certain attendant disadvantages. The 1950s and 1960s saw a proliferation of Universities in most States, as well as the establishment of the Australian National University at Canberra and a Faculty of Agriculture in Tasmania in 1961.[18]

During this era, the most noticeable change in the training of plant pathologists in Australia was the introduction of the post-graduate PhD degree with improved facilities for post-graduate training in specialised branches of the subject. This introduction was followed by the establishment of the first two chairs of plant pathology in Australia, in Adelaide in 1964, and in Sydney in 1966. A consequence of this was that graduates who had majored in plant pathology for their first degree became less inclined to seek overseas experience until after they had undertaken the post-graduate course. Many went overseas on post-doctoral fellowships, with the advantage of maturity. The implementation of training at both levels was made possible through an increase in staff and through the appointment of persons well qualified and experienced in research. In the 1960s, the Waite Institute at Adelaide University became the most favoured for post-graduate training, and Sydney's Faculty of Agriculture for undergraduate training.

The Waite Institute's premier position at that time in the post-graduate training arose partly from its total responsibility for plant pathology services in South Australia, justifying a disproportionately large staff for an academic institution in a State with a small population and a large area of land that was mostly desert. It also achieved this status through the energetic and ambitious activities of the astute head of its Plant Pathology Department from 1958 to 1970, the late Professor N.T. Flentje.[19] His persuasive tactics attracted financial support for his department and, by the mid-1960s, he had developed the best equipped and strongest research group in Australia consisting of nine specialist plant pathologists. Consequently the Waite Institute was an

ideal centre for post-graduate students, attracting many from other Australian Universities and State Departments, as well as an even greater number from South East Asian countries.

By the end of the 1960s, agricultural graduates were filling about half the positions of plant pathologists throughout the Commonwealth. This was partly due to the large intake of Agriculture students in a State with the largest population, but it was also because of the nature of the undergraduate training in plant pathology. The tradition established by Waterhouse was maintained after he retired in 1952, when plant pathology became a sub-department with an Associate Professor in charge. In 1955, the 5th honours year was abolished, and in its place, any student who had qualified could specialise in plant pathology in his or her final year. This allowed many who would not graduate with honours to take plant pathology in their final year, and so increased the pool of graduating potential plant pathologists.[19a] Since the late 1960s post-graduate students have tended to go to where there are specialists with established reputations in particular fields, as is usual in Europe and North America.

From the 1920s, plant pathologists employed in Australia have had some training in plant pathology, mostly as undergraduates in Australia and overseas Universities. The Governments of all States were providing advisory services in plant pathology by 1930, when there were 26 plant pathologists scattered throughout the Commonwealth. Until the end of the Second World War, recruitment by most of the States was from graduates of their own State University, engendering some degree of insularity, with all that that implies.

Plant quarantine was initially introduced by the States to limit the spread of introduced pests and diseases.[21] With the initiation of Federal Government in 1901, moves were made for plant quarantine to become the responsibility of that Government. In 1908 the Plant Quarantine Act was passed, incorporating the original legislation of the States; and the Federal Quarantine Service was established initially under the jurisdiction of the Department of Trade and Customs, later under the Ministry of Health. The first Director of Plant Quarantine in 1929 was E. Mackinnon, who was succeeded by Dr T.H. Harrison in 1946, when the quarantine regulations began to be vigorously implemented and upheld.

Research institutes

In the 1920s the establishment of two institutions was to exert a significant influence on the future of plant pathology in Australia. They were the Waite Agricultural Research Institute, within the University of Adelaide in 1924; and the Division of Plant Industry (Economic Botany) in 1927, within the newly formed Federal Council for Scientific and Industrial Research (CSIR). At the Waite Institute the first head of plant pathology, Geoffrey Samuel, a science graduate from Adelaide, proved to be a versatile and capable plant pathologist, ready to turn his hand to any problem. He was responsible for all plant pathology services in South Australia, including teaching it to undergraduates.[22] In 1934, he resigned from the Waite Institute to join the staff at Rothamsted in England. He was replaced at the Waite by D.B. Adam from the Victorian Department of Agriculture.[23]

The need to investigate plant disease problems of national importance was partly responsible for the establishment, in 1927, of the Division of Economic Botany (later changed to Division of Plant Industry), within the CSIR. The first Chief of the Division was Dr B.T. Dickson, an English-born Professor of plant pathology at McGill University in Canada.[24] In ten years he built up a staff of 32 research scientists, of whom eight were plant pathologists working under the direction of Dr H.R. Angell.[25] During that period, research work was undertaken on tomato spotted wilt, blue mould of tobacco, virus diseases of tobacco, flag smut and take-all of wheat, virus diseases of potatoes and bitter-pit of apples. Significant contributions to the knowledge of all these diseases were made, but the most outstanding was that made by Angell and his colleagues on the control of blue mould of tobacco in seed beds by the use of benzol vapour. In 1951, Dickson's successor as Chief of the Division of Plant Industry was Dr Otto Frankel, a geneticist and plant breeder of international repute, whose policy was for the Division to be concerned entirely with fundamental research.[26] He considered the research of plant pathologists to be 'pedestrian' and accordingly disbanded the plant pathology section, replacing it with a section labelled 'Microbiology', implying research of a fundamental nature. He appointed Dr K.O. Muller in 1953, on a five-year tenure, to pursue his researches on 'Phytoalexins' or 'warding-off substances' formed in tissues injured through the presence of pathogens. [27]

By the 1960s, the number of plant pathologists employed by Government services, in both Agriculture and Forestry, had increased

greatly.[28] Many of them were specialists, such as virologists, nematologists, mycologists, rather than general practitioners. Routine investigation work of plant diseases, was becoming the province of extension workers, including private practitioners, backed by expert advice from the specialist plant pathologists including private practitioners. During this time, well-equipped laboratories were built in the different States and, in some of them, regional laboratories were established at centres where local problems could be more effectively investigated. Some of the new laboratories in State Departments were given the dignified but well-earned appellation of 'Research Institutes', indicating their greater role in research, as well as carrying more prestige than is conveyed by the title of 'branch' or 'Laboratory'.

Communication and publication

The formation of the Australian Institute of Agricultural Science in 1935 helped very much to bring together the scattered groups of workers at annual conferences, and the Institute's journal, as an avenue for publication of scientific and technical papers, helped to bridge the communication gap. Until the end of the Second World War, the avenues for publication in Australia of plant pathological work were restricted to the journals published by the State Departments of Agriculture to inform the farmer and grower of developments; more scientific articles went into the journals of the proceedings of local Scientific Societies. In 1949, the *Australian Plant Disease Recorder* was commenced by the N.S.W. Biology Branch in an attempt to bring together the common interests of plant pathologists, hoping thereby to compensate for the great distances separating centres of work. With Dr F.C. Butler as its first editor, the *Recorder* succeeded in this aim by producing, in mimeographed form, 'articles of brief but informative nature designed to encourage the free expression of views and opinions'. The *Recorder* was distributed to all plant pathologists in the Commonwealth four times a year for the next 20 years. It was the forerunner of the Plant Pathology Society's *A.P.P.S., News Letter*, which in 1978 became the refereed journal *Australasian Plant Pathology*.

For the dissemination of research findings overseas, Australian plant pathologists depended largely on publishing in the journals of overseas Societies. Indeed, few workers overseas were aware of Australian work unless it had been published in such journals. To some extent this state of ignorance still prevails, though the establishment of various refereed

Australian scientific journals in recent years has begun to attract more attention to Australian work.[20]

After the end of the World War II there was a vast improvement in communication between plant pathologists, both within and outside Australia. Within Australia this was achieved at conferences, and by improved dissemination of research findings through an increase in the number of scientific journals published in Australia. Above all, the possibility of exchange of personnel between Australia and overseas centres was greatly improved. The most effective improvement of communication within Australia was the institution of plant pathology conferences sponsored by the National Agricultural Council, making it possible for the widely scattered community of plant pathologists to come together at five-yearly intervals. The last of these was held in 1971, when 140 delegates attended.[29] With the formation of the Australian Plant Pathology Society in 1969, these conferences were replaced by the biennial meetings of the Society, the first of which was held in Sydney in 1974.

From the published records of plant disease investigations in Australia it is apparent that progress in Australian plant pathology often reflected overseas developments in the science. Thus, during the formative years, the emphasis was on discovering causes of plant disease; the mid-1950s saw the beginning of an inevitable era of specialisation. A 'new breed' of plant pathologists came into being, specialists in plant virology, bacteriology, nematology, and so on. Through all these phases of advancement of knowledge, fashions or vogues may be perceived. One has only to look through the titles of articles on plant pathology in accredited scientific journals over a period of years to see evidence of such fashions. Australian plant pathologists not only followed these fashions, but created their own new ones.

The History of Some Plant Disease Investigations in Australia

An important part of the history of plant pathology is the history of individual plant diseases in Australia, and some of these will now be considered.

Black Stem Rust of Wheat (*Puccinia graminis* f. sp. *tritici*)

Historically and economically, black stem rust is the most important plant disease in Australia. The control of the disease is by breeding resistant cultivars, so that much of its general history belongs to plant breeding, though in many aspects of the disease in Australia, pathology

is inevitably linked with plant breeding. If the 'blight', referred to in the earliest records of growing wheat in Australia, was black stem rust, then the disease was destined to plague wheat crops from the beginning of colonisation.[30] The term 'rust' was not used until 1803, when the superintendent of Captain Cox's 'Brush Farm' in the Dundas-Eastwood district of today, reported that 265 acres of wheat grown there were completely destroyed by rust, some three weeks before harvesting.[31]

The question of the source of the earliest rust epidemics has exercised the minds of Australian cereal rust workers ever since. The possible sources are: it was on native grasses before wheat was grown in Australia; it was wind-borne from South Africa and/or India, where the rust was well-established at that time; it came from infected and introduced plants of an alternate host for the stem rust fungus, the ornamental shrub, *Berberis vulgaris*; or it was seed-borne in the Cape wheat used for sowing the first successful crops in the Sydney area. Native perennial grasses of the genus *Agropyron*, particularly *A. scabrum*, or wheat grass, are near relatives of wheat and occur throughout the wheat belt of Australia. They are hosts for stem rust which may have been present on them prior to the arrival of the first fleet in 1788. Urediniospores of the rust fungi survive aerial transport in the stratosphere (some of the few spores that do), so that viable urediniospores of stem rust of wheat could be blown from South Africa and/or India, and from Australia to New Zealand.[32] However, it is improbable that the outbreak of stem rust in wheat crops grown during the first few years near Sydney were caused by airborne urediniospores from other continents, because the chance of such spores landing on such a small target area is extremely unlikely. The common barberry, *Berberis vulgaris*, is the alternate host for the wheat stem rust fungus on which the sexual stage of the fungus occurs and produces new strains.[33] This could not have been the source of the early rust infections because this ornamental shrub was not introduced into Australia until much later than the first appearance of the rust on wheat.[31]

Rust fungi are not seed-borne in the sense that urediniospores mixed with seed do not initiate rust infections in plants grown from contaminated seed. The first wheat crops grown in Australia were raised from seed brought from Capetown, late in 1788. It is likely that this seed was contaminated with urediniospores of stem rust, since the disease was well-established in South Africa at that time. It is possible

that when the Cape wheat was being sown, urediniospores were blown onto nearby native wheat grass, *A. scabrum*, that became infected with the rust. Being a perennial grass, such infected plants would then provide inoculum for infection of the following season's wheat crop. A similar happening is believed to have occurred when snapdragon rust, *Puccinia antirrhini*, was recently introduced into Australia in snapdragon seed contaminated with urediniospores of the rust. The accidental dusting of snapdragon seedlings with urediniospores is believed to have occurred when the contaminated seed was being sown in nearby beds.[34] The most likely source of the stem rust of wheat infections in the new colony appears to have been infected *Agropyrum scabrum*. It was either host to an indigenous rust before European colonisation, or, more likely, it became a native host when dusted with urediniospores from the contaminated Cape wheat seed. This grass now serves as an important link in the infection chain of wheat stem rust in Australia.[33]

By the 1880s, the uncertainty of the wheat harvests through rust epidemics, and the nondescript nature of the wheat varieties brought to Australia by the early settlers from England and Europe, threatened the developing wheat industry. The plight of the wheat industry at that time induced William Farrer to take up the self-appointed task of improving, through selection and breeding, the suitability of wheat cultivars for the Australian climate and their resistance to stem rust. Farrer, a Cambridge graduate in mathematics, had been a surveyor with the Lands Department of N.S.W. since 1875, and had ample opportunity to observe the plight of the wheat farmer. In 1886 he resigned from his position as surveyor, and began his work on wheat on 3 acres of his property, 'Lambrigg' at Tharwa near the present Canberra.[35] He continued his monumental work until his death in 1906, during which time he succeeded in producing a number of new cultivars of wheat by hybridising the more promising wheats imported from different countries. The most popular one was 'Federation' produced in 1901, the year of the Federation of the Australian States. Widely grown throughout Australia from 1910 to 1925, the success of 'Federation' was due to its rust-escaping earliness rather than to true resistance. Its earliness permitted the extension of wheat growing areas into hotter and drier parts of Australia where stem rust was not so serious.

The challenge to know more about the rust fungus in Australia was taken up by W.L. Waterhouse at Sydney University in 1918, and

continued throughout the whole of his working life at the University. He explored the strains of stem rust present in N.S.W., and embarked on a programme of breeding rust-resistant wheat cultivars to the strains then prevalent in N.S.W. He determined the strains present by using the twelve differential wheat varieties that Stakman and Levine had established in their classic work on strain differentiation of rust in U.S.A. in 1922.[36] For the collection of rust samples for strain determination on the wheat differentials, he enlisted the help of teachers in country schools throughout the wheat belt in N.S.W. By the late 1930s Waterhouse had produced 3 new commercially viable varieties resistant to all the strains then known to be present in N.S.W. His variety 'Gabo' was the most widely grown of these, until its resistance was broken in 1948 by the appearance of a new rust strain. Breakdown of resistance to rust was to be repeated many times in the future, because the new strains were somatically induced.

In Australia, stem rust fungus maintains itself solely in the uredinial stage as continuous infection chains on self-sown wheat plants growing on roadsides and headlands out of season as well as on *Agropyron scabrum* and possibly other native grasses. Unlike the Northern Hemisphere countries, with their cold winters, the alternate barberry host does not play any significant part in the survival of the stem rust fungus in Australia, except in Tasmania where the climate resembles somewhat that of the temperate wheat growing areas of the Northern Hemisphere. In spite of the fact that barberry infections, which are normally required to give rise to new strains of the rust, do not occur on mainland Australia, there is abundant evidence that new strains arise frequently. Professor I.A. Watson at Sydney University has shown that the main source of the new strains of stem rust is through mutation and somatic hybridisation, in which hyphae of genetically different strains anastomose and exchange their nuclear material in infected wheat leaves.[37]

When Watson was appointed to help Waterhouse with the rust work in 1938, the Stakman differentials were supplemented with 8 additional wheat hosts containing single genes identified for resistance. A new system of naming the Australian rust strains was implemented, for it was essential for the breeding work to be able to identify strains accurately, not only in different localities but also in different years. Information on the occurrence of strains of rust provides evidence of movement of the rust within Australia over a period of years. Thus it was shown that there was a movement northwards in N.S.W. of strain

21.1 in the 1955-56 season; a new strain, 21.2, originated in southern Queensland in 1956 and moved southwards through N.S.W. into Victoria, South Australia and Tasmania by 1959. The virulent strain, 21.2, was somehow carried westwards across desert country into Western Australia, appearing there in 1960. Both strains, 21.1 and 21.2, of the eastern States were carried (in the wind?) across the Tasman sea to New Zealand by 1959.[38] Knowledge of strains of stem rust in Australia has made possible the study of the ecology of rust strains; an understanding of how they compete with one another on the same host in turn helps to explain why there have been 'rusty' and 'non-rusty' years, chance playing a large part in strain survival. In some years the main strain surviving after extended periods of drought has been quite different from those present beforehand.[39]

Sydney University has become a world centre for rust investigations, and now has two wheat research Institutes situated where the climate is most suitable for rust studies. One is at Castle Hill near Sydney and one at Narrabri in the northern wheat belt. Sydney is not the only place in Australia where rust work and breeding wheats is undertaken. Others are the Queensland Wheat Research Institute at Toowoomba, the Victorian Wheat Research Institute at Horsham and the N.S.W. Department of Agriculture Research Institutes at Tamworth and Wagga Wagga. In the 1960s, J.F. Brown and W.A. Shipton at Sydney University attempted to discover the nature of the gene action involved in resistance mechanisms in different wheat cultivars with specific genes for resistance. Amongst other facts it was found that tissue necrosis in hypersensitive cultivars resistant to rust infection is the result rather than the cause of resistance, as was formerly believed; the dead cells are merely 'monuments of a battle once fought' between host and pathogen.[40]

An attempt was also made by the late Dr Beverly Keed at Sydney, to assess the effect of stem rust and leaf rust infection on yield and quality of the grain. It was found that while leaf rust, *Puccinia recondita*, caused a maximum loss in yield of 26 percent, stem rust, *P. graminis tritici*, could cause a maximum loss of 55 percent. With this loss, there was a corresponding reduction of the protein of the grain resulting in inferior baking quality.[41] An interesting fact which emerged from these studies was that losses caused by stem rust differed greatly between different susceptible wheat cultivars, suggesting the operation of a factor for tolerance. More information is needed about the epidemiology of stem rust in Australia. It is under investigation by two

graduates from Sydney: Associate Professor J.F. Brown at the University of New England, and Dr R.G. Rees at the Toowoomba Wheat Research Institute. Research on this aspect of stem rust emphasises the dilemma involved in attempting to produce new cultivars of wheat resistant or tolerant to the many new strains of the rust fungus that continually arise.

Of all the splendid contributions to fundamental knowledge made over the years by Australian plant pathologists, probably the most spectacular was the culturing of the stem rust fungus on artificial medium by Drs P.G. Williams and K.J. Scott in 1965. In many ways, Sydney University was the right place for the culturing of the rust fungus. Apart from historical reasons[42], there was on hand a range of wheat host selections and rust strain isolates. At the time of their achievement they held teaching and research positions in the Department of Biochemistry at Sydney University. Both had the right kind of academic training for such an undertaking, having majored in plant pathology and biochemistry in their final year as undergraduates in the Faculty of Agriculture at Sydney, and both later pursued post-graduate training overseas, Williams at Wisconsin, and Scott at Queen's University, Ontario. Their success in culturing the rust fungus on artificial medium was largely due to their technique of producing urediniospores aseptically, from which their cultures were grown. It was also due to their choice of yeast extract additive to the Czapek-Dox nutritive medium, on which the rust was cultured.

Scepticism and incredulity were the first reactions of most scientists who heard about their success.[43] They had at this stage produced only the vegetative growth of the fungus in culture. To prove beyond doubt that it was in fact the rust fungus, Koch's Rules of Proof had to be satisfied.[44] In 1966, they were able to develop both the urediniospores and teliospores on stroma that formed on densely-seeded culture medium containing 0.1 percent Evans's peptone in addition to the yeast extract-sucrose nutrient medium. They proved the pathogenicity of the urediniospores produced in the axenic culture, thereby satisfying Koch's postulates[45] (Fig. 4). The growing of rust fungi in axenic culture is not merely a scientific novelty, but has enabled studies to be made on the nuclear cytology, infection structures and growth behaviour of different rust strains, that could only be made in axenic cultures.[46]

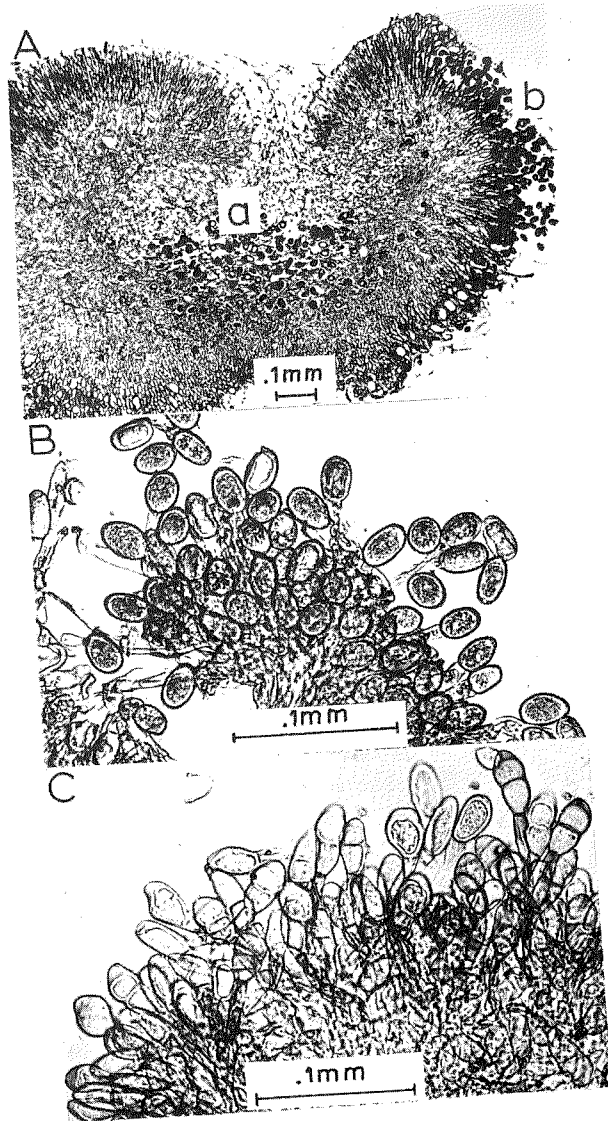


Figure 4. Wheat Stem Rust, *Puccinia graminis* var. *tritici*, producing urediniospores and teliospores in axenic culture, by Williams and Scott in 1966.

- (a) Microtome section through a uredinial stroma, cut at right angles to the agar surface, showing the original inoculum (a), and the uredinial spore-forming layer (b).
 - (b) Water mount of urediniospores, stained with crystal violet.
 - (c) Glycerine-jelly mount of teliospores formed in vitro.
- (Photos by courtesy of Dr P.G. Williams)

Blue Mould or Downy Mildew of Tobacco (*Peronospora tabacina*)
The cultivated tobacco of commerce, *Nicotiana tabacum*, originally a native of South America, was first grown in Australia about 150 years ago, but was not grown intensively until after World War I. In a short time, downy mildew appeared as epidemics, especially in the seed beds, seriously hampering the development of the tobacco industry. According to hearsay from farmers who grew tobacco as early as 1850, the disease was evident then in seed-beds but the first record of it was made in 1890 by Henry Tryon in Queensland. The next year, Dr Cobb in N.S.W. reported 'the disease, which threatens to extinction the culture of tobacco in certain parts of the colony, especially on young plants'. In 1889, McAlpine in Victoria had observed the disease on native tobacco, *Nicotiana suaveolens*, an indigenous species that occurs widely in Australia, except in Tasmania. In 1933 D.B. Adam of Victoria described and named the fungus *Peronospora tabacina* and found it on three naturalised species of *Nicotiana* (*N. glauca*, *N. rustica*, and *N. longiflora*), all introduced from South America. At that time it was believed that *P. tabacina* was introduced from South America where the disease was known to occur on cultivated tobacco. Since some of the Australian native species of *Nicotiana* (e.g. *N. debneyi* and *N. goodspeedii*) are resistant to *P. tabacina*, it is now accepted, consistent with Vavilov's rule for indigenous plant pathogens, that the fungus is indigenous to Australia.[48] The disease was reported in U.S.A. in 1922, and is believed to have been inadvertently introduced there from Australia.

In Australia in 1929 Dr H.R. Angell of the Division of Plant Industry in the CSIR began investigations on the disease; the main objective was control of the infections in the seed-bed where most trouble occurred. He found there were two sources of inoculum for seed-bed infections. One was from within the seed itself when contaminated with oospores in seed harvested from diseased crops.[49] The other was from infected native tobacco, *N. suaveolens*, and the naturalised tree tobacco, *N. glauca*, growing in the proximity of seed-beds.[50] This source of infection was confirmed by H.A. Pittman in 1930 in Western Australia, when an outbreak of the disease occurred in a newly-cultivated area at Manjimup, W.A., which he traced to diseased native tobacco, *N. suaveolens*, growing near the seed-beds[51] (another reason for supposing the fungus to be indigenous to Australia).

The disease is spread from leaf to leaf, and plant to plant by airborne conidia that germinate in water droplets on the lower leaf surface and

infect the leaf through stomata. Spore germination and penetration had therefore to be prevented in order to control the disease. Spraying and dusting with the known protectant fungicides were ineffective because of the mechanical difficulty of thoroughly covering the lower surface of leaves close to and parallel with the ground. Angell therefore sought for a vapourising chemical that could be used to prevent spore germination and penetration, without causing injury to the plant tissues.

On surveying the literature for such a chemical, Angell noted that overseas workers had used the volatile hydrocarbons, benzol and toluol, for soil sterilisation against soil fungi, as a wood preservative against fungal rots, in the control of brown rot of stored peaches and for the control of cotton root rot fungus in the soil. All reported that although fungal growth was inhibited by the volatile hydrocarbons they caused tissue injury, and in peaches, an undesirable benzol flavour in the fruit. No one had yet used volatile hydrocarbons for the control of leaf pathogens like downy mildew.

In 1934, Angell found that benzol, toluol and xylol all inhibited the germination of conidia of a number of species of *Peronospora*, including *P. tabacina*, in hanging-drop cultures in vessels exposed to the hydrocarbon vapour.[52] Assisted by J.M. Allen, and A.V. Hill, both tobacco pathologists with the CSIR, Angell experimented with seed-beds of tobacco seedlings which were first sprayed with conidia of *P. tabacina* then covered with canvas and exposed to benzol and toluol vapour. They found that infection was prevented, with no injury to seedlings, in beds treated with an area of liquid benzol or toluol equal to 2 sq. inches per sq. foot of the bed. The covers could be removed on fine days so long as the beds were exposed to the nightly fumigations. Benzol was considered to be the best vapour to use because of its availability in commercial quantities.[53]

In autumn of 1935, extensive field trials were made in the tobacco growing districts of Shepparton and Myrtleford in Victoria, at Ashford in N.S.W., and at Manjimup in Western Australia. All confirmed the efficacy of the benzol vapour treatment. From that time, benzol vapour treatment of tobacco seed-beds has become standard practice wherever susceptible varieties are grown.

In 1959, an interesting field observation was reported by W. Pont at Mareeba in Northern Queensland. Mature plants when stem-infected by *P. tabacina* did not show the foliage symptoms of sporulation by the fungus.[54] The field observations were confirmed experimentally by I.A. Cruikshank, and M. Mandryk in the Division of Plant Industry,

CSIRO, Canberra.[55] Following up these findings, J.C. Shepherd, also of the Division of Plant Industry at Canberra, found that a fungal inhibitor produced in stem-infected tobacco plants prevented sporulation by the fungus in systematically infected leaves, as well as inhibiting infection by conidia.

Meanwhile in Canberra, a programme for breeding cultivars of tobacco resistant to downy mildew was begun by D.C. Wark. He made a survey of resistant native *Nicotiana* species, and using the 'wild' strains of the mildew fungus in screening for resistance, sought for species of the *Nicotiana* host in which the growth of the fungus was arrested, or sporulation inhibited. He selected two such species, *N. debneyii* and *N. goodspeedii*, and after making the first cross in 1953, made selfings and backcrosses through 18 generations of the progeny. Two commercially viable cultivars of tobacco were produced in 1967, and named 'Sirone' and 'Sirogo'. The variety 'Sirogo' was resistant to strains present in Victoria, while 'Sirone' was resistant to the strains present in the Mareeba area. Although 'Sirogo' proved to be eminently successful in the Myrtleford area in Victoria, 'Sirone' proved to be a disaster in the Mareeba district of North Queensland because it was very susceptible to black shank disease of tobacco, ever-present in the warmer Mareeba area. The fungus causing black shank disease, *Phytophthora parasitica* var *nicotianae*, survives mainly by chlamydospores formed in diseased tissues, and remains in the soil as inoculum between crops. The newly introduced variety 'Sirone' is not only more prone to increased chlamydospore development within the host tissue, but is also more readily invaded by the fungus. The result was a dramatic upsurge of black shank disease not only in 'Sirone' but in the more tolerant varieties, through a greatly increased inoculum load in the soil, increasing the chances of infection.

Black Spot of Citrus (*Guignardia citricarpa*)

This disease was first described in 1895 in citrus orchards around the Sydney metropolitan area, but was probably present in citrus long before that. The imperfect stage of the causal fungus was named *Phoma citricarpa* by McAlpine in 1899, and by 1920 the disease was found in most of the citrus growing areas scattered throughout Australia. Since it was first described from Australia, it was thought to have originated here. It now seems certain that the disease originated in the natural home of its citrus hosts, probably in South Eastern Asia, or China. The first indication that the disease occurred outside Australia came in 1920,

when the fungus was isolated in pure culture from citrus fruits grown in many districts of China, in Hong Kong and Taiwan. Since then, the disease has been found in most citrus-growing regions of the world except U.S.A. In 1920, the Australian Plant Quarantine Authority intercepted dried orange peel imported from the Shanghai district of China, with typical black spot lesions in which the fungus *Phoma citricarpa* was found. In 1932, the United States Department of Agriculture Plant Quarantine found the disease on oranges imported from China, Japan and India.

It is difficult to say when and whence the disease was introduced into Australia, but it is not difficult to say how the black spot fungus was introduced. The work reported by Dr Temple Kiely of the N.S.W. Department of Agriculture in 1948 supplies the clue. Kiely not only discovered and described the perfect or ascigerous stage of *Phoma citricarpa*, naming it *Guignardia citricarpa*, but much more importantly found that the fungus caused latent infections in leaves and fruit of citrus.[56] The latent infections remain unnoticed until the leaves and fruit become senescent, when the black spot lesions develop, producing first the conidial stage (*Phoma citricarpa*), and later the ascigerous stage (*Guignardia citricarpa*). It is reasonable to conclude that the fungus arrived in Australia as unseen infections latent in the leaves of imported citrus stock within the early years of settlement. A similar fungus has been found on a number of hosts, members of different angiosperm families, but they are probably different species of the genus *Guignardia*. [57]

Gummosis or Die-Back of Apricot (*Eutypa armeniaca*)

The disease of apricot trees known as 'Gummosis' or 'Die-back', which occurs in South Australia, Tasmania and Victoria, is another introduced disease first reported from Australia. It was first described by Dr W.J. Dowson in Tasmania in 1931, and in 1938, D.B. Adam of the Waite Institute in Adelaide isolated a fungus from dead wood of affected limbs of apricot and identified it as *Cytosporina* sp. Wound inoculations on healthy apricot limbs, using only conidia produced in plate cultures, failed to induce infection nor could the conidia be induced to germinate by various methods and media. However, when mycelium from the plate cultures was used as inoculum for the wound inoculations, infection occurred and the characteristic symptoms developed 3 to 6 months later.[58] The experience was to be repeated by subsequent workers on the disease.[59 & 60] In 1945, in an apricot

orchard at Rokeby in Tasmania which was affected by gummosis, I exposed plates of agar culture media for a few minutes after rain in May. All the plates developed many colonies of a fungus identified by Adam as *Cytosporina*. [61] The Tasmanian isolates were shown to be pathogenic when Dr M.V. Carter at the Waite Institute used mycelium as inoculum in 1955.[62] The Tasmanian isolates clearly originated from air-borne inoculum, but air-borne conidia could not have been the source for the trapping in the plate cultures, though pycnidia of *Cytosporina* develop on dead wood of diseased limbs. Carter therefore looked for the perfect stage of the *Cytosporina* fungus on old dead wood of apricot trees and on discarded prunings in headlands of orchards in South Australia, Tasmania and Victoria. In all these localities Carter found the perithecial stage of a species of *Eutypa*. Cultures from ascospores of this fungus were identical with that of *Cytosporina* sp., and were equally pathogenic on apricot. Dr C.G. Hansford, also of the Waite Institute, described this species of *Eutypa* as *E. armeniaca* sp. nov., and it is believed to be the first record of a pathogen in the genus.

The hypothesis was advanced by Carter that *E. armeniaca* and *Cytosporina* sp. are the perfect and imperfect stages respectively in the life cycle of a single organism responsible for gummosis or die-back disease, and that air-borne ascospores of *E. armeniaca* constitute inoculum by which the disease spreads both within and between orchards.[62]

Until 1953, apricot was assumed to be the only host for *E. armeniaca*. Carter then found that 98 different varieties of peach and nectarines, and a plum, all developed gummosis symptoms when inoculated with mycelial inoculum. Subsequently, he found perithecia of the fungus on dead wood of grape vines throughout the non-irrigated vine-growing districts of South Australia, on dead stubs of limbs of apple and walnut in a garden near Crafers in the Adelaide Hills and on dead branches of the ornamental shrubs *Ceanothus* and *Tamarix* in Adelaide gardens.

When *E. armeniaca* was first described in 1957 there were no records of its occurrence as a pathogen on apricot, outside Australia. It has since been recorded on apricot from New Zealand in 1959 and from California. It was found on *Tamarix* in the Rhone Valley in France[63] in 1964 and was found to be prevalent in Switzerland on grape vines and stone fruits.[64] Since apricot trees have been cultivated in the countries of Europe far longer than in Australia, New Zealand and U.S.A., it is believed that, although the fungus has only recently been identified in Europe, it is likely that it has occurred there for a very long

time, and that it was probably introduced to the 'younger' countries of the world by importation of infected trees from Europe. Carter and his colleagues found that perithecia and ascospores of *E. armeniacae* may be produced on dead apricot wood for at least 5 years following maturation of the first stroma, and that abundant ascospores are released following rainfall exceeding 0.05 mm, except during a period in winter when there is a low ascospore frequency that coincides closely with the dormancy of the apricot.[65]

In the field, infection of apricot occurred when inocula of 10 to 100 ascospores were applied to wounds on the day of pruning, and then only by ascospores drawn into the vessels of the wood, up to a depth of 4.5 mm; those on the surface of the wound do not germinate. This explained why conventional methods of spraying newly pruned trees with a protectant fungicide did not prevent the incidence of gummosis.[65] They also found there was a rapid decline in susceptibility of pruning wounds made in early winter to infection by ascospores.[66] The seasonal decline in susceptibility of wounds was found to be related to the colonisation of the wound surfaces by other micro-organisms (bacteria and fungi), which produce diffusible metabolites toxic to both ascospores and mycelium of *E. armeniacae*. [67] It was further found that spraying pruned apricot sapwood with a suspension of spores of a common component of the wound microflora, *Fusarium lateritium*, gave a high degree of protection against infection. Such a treatment would be impractical and it was found that spraying with Benomyl after pruning gave excellent control.[68]

Grain Smuts

The cereal smuts occurring in Australia can be classified according to infection types into seedling-infecting smuts, whose inoculum is both seed-borne and soil-borne, and flower-infecting smuts, whose inoculum is only seed-borne within the embryo. The primary source of inoculum for both types is air-borne teliospores (chlamydospores). All the cereal smuts were introduced into Australia on contaminated cereal grain or cereal straw used for packing in the early days of colonisation. Most are seedling-infecting smuts, such as bunt of wheat (*Tilletia caries* and *T. foetida*), flag smut of wheat (*Urocystis tritici*), loose smut of oats (*Ustilago avenae*), covered smut of barley (*Ustilago hordei*), and head smut of maize (*Sphacelotheca reiliana*). The flower-infecting smut is 'loose smut' of wheat and barley (*Ustilago nuda*). Floral infection was

first demonstrated in 1896 in Tasmania by Frank Maddox, an experimentalist with the Tasmanian Council of Agriculture. He reported his discovery in the Tasmanian Agricultural Gazette as follows: 'I have never been able to cause infection and reproduce the disease with spores on the grain or in the ground, which I can so easily do with bunt spores to reproduce bunt. The only way I have been able to infect grain and reproduce smut is by putting the spores on the pistil of the plant at flowering time, about the same time as the pollen-grains are being shed. The grain will mature without the slightest signs of being diseased'. [69]

For the control of seedling-infecting smuts, steeping the grain in various solutions had been known since 1755 through the work of M. Tillet in France. [70] He found that steeps of urine or lye before planting the grain reduced the amount of bunt in crops. In 1807 B. Prevost, also in France, found that steeps of lime in seawater, or copper salts, gave better control of bunt. [71] The steeping of grain before sowing was a tedious process, and the necessity for the treated grain to be subsequently dried resulted in some loss in viability of the grain. In 1919, Darnell Smith in Sydney discovered that treatment with dry copper carbonate gave better results, eliminating the need to dry the seed before sowing, and was therefore less tedious. Such seed treatments were of course useless for the control of flower-infecting smuts for which the hot water treatment was devised. Seed 'pickling' with copper carbonate and hot water treatment are now superseded by breeding for resistant cultivars in both types of smuts and by seed treatment with new chemicals such as hexachlorobenzene (BHC) and the oxathiins. However, J. Kuiper at the N.S.W. Department of Agriculture Research Institute at Wagga, has found new strains of *Tilletia foetida* resistant to BHC. [72]

Of special interest, in Australia, is flag smut of wheat (*Urocystis tritici*). It was first reported from South Australia, and since then has caused serious losses in wheat crops in all wheat-growing areas in Australia. It was at first believed to be indigenous to Australia, but all evidence indicates that it was introduced and probably it first entered on contaminated wheat and/or rye seed, or straw used for packing, brought by the German settlers to South Australia in the mid-1840s. The disease is now known to occur in most wheat-growing areas of the world, but features of the climate of Australia, especially the soil temperatures, favour its survival and development here. The compound teliospores of flag smut may survive many years in the soil,

especially with the prevailing dry summers and mild winters.[73] As its name implies, flag smut infection results in covered sori of teliospores on the flag or blade of the leaf. Infected plants do not produce ears, so that when infection builds up in a crop to a high level there are serious losses in yield. The source of the inoculum is the sori of infected leaf straw, from which teliospores are released during harvesting. They become attached to the surface of the grain, or lodge in the soil, where they remain dormant until autumn; when the wheat is sown they germinate and infect the seedlings.

The teliospores do not germinate in water unless fragments of straw or wheat grains are present. Dr R.J. Noble of the N.S.W. State Department of Agriculture, when a student at Minnesota University in 1922, found that traces of benzaldehyde released from the wheat straw or wheat grains stimulate the germination of the teliospores.[74] Infection occurs in the seedling stage, as in all seedling-infecting smuts, when the coleoptile is invaded by dicaryotic hyphae, from which the infection hyphae spread into the leaf primordia, and then multiply, keeping pace with the developing leaves. Work carried out by Dr J.G. Churchward at Sydney University in 1936[75], and by H.R. Angell and Phyllis Jarrett in the Division of Plant Industry at Canberra in 1936-38[76], showed that in resistant cultivars like 'Nabawa' infection of the coleoptile occurs as in susceptible cultivars, but the infection hyphae fail to reach the leaf primordia, or if they do enter the primordia, fail to keep pace with the development of the leaves.

It was found that 'Fife' wheats provided a source of resistance to flag smut. Farrer had used them widely in his crosses, so that many of varieties bred by him were resistant to flag smut although he did not select for this resistance. The variety 'Nabawa', produced in Western Australia in 1915, owed its resistance to flag smut to its 'Fife' inheritance. The smut was not officially recorded in that State until 1922, when it was causing serious losses in susceptible varieties that were widely grown there. The resistance of 'Nabawa' was first noticed by a farmer at Belka in W.A., when he planted his 175 acres after fallow, half with 'Gluyas Early' and half with 'Nabawa'. Flag smut was found only in 'Gluyas Early'. W.M. Carne, then Government plant pathologist in W.A., was quick to confirm this observation, and 'Nabawa' became widely grown in the West as well as in other parts of Australia within 7 years.[77]

Take-all Disease of Wheat (*Gaeumannomyces graminis tritici*)

The disease is the next most important disease of wheat, after black stem rust, in Australia. It was first mentioned as occurring in South Australia as early as 1852, and since then has been found in nearly all wheat growing areas of the world. Few diseases have attracted so many workers to its investigation over such a long period of time as take-all. This applies particularly to Australia, where more than a score of workers have been investigating it since the early 1920s. Some of this work was inevitably repetitive, either denying or confirming the work of others, both within and outside Australia. A very considerable literature has resulted, but no attempt will be made here to mention all who have contributed.

As mentioned earlier, it is almost certain that the take-all fungus is indigenous in Australia, along with many other soil-borne root pathogens. The fungus occurs in grasslands in most of the temperate regions of the world. In Australia, with its varying climate and soil conditions, it occurs wherever wheat is grown in a region extending from southern Queensland to the drier parts of South Australia and Western Australia. The disease was named 'take-all' because it kills all the wheat plants it attacks, from seedling to heading stages, resulting in somewhat circular patches of dead or stunted plants scattered throughout the crop. The most obvious early sign of the disease is seen while the crop is still green, when dead or white heads appear scattered or in clumps through the crop, resulting from the premature ripening or death of culms or plants. However, the 'white-head' condition itself is not sufficient to diagnose the disease as take-all, because other agencies can cause white heads, such as other root rot fungi or even a virus.

In 1890, Prillieux and Delacroix in France were the first to establish the cause of white heads as infection by the root-rotting fungus *Ophiobolus graminis*. It was left to McAlpine of Victoria in 1902 to show that *O. graminis* was the cause of take-all in Australia, and that the patches of dead seedlings and the white-head condition were different phases of the one disease. In 1972, the generic name was changed to *Gaeumannomyces*. [78] There are two forms of *G. graminis*: one is *G. graminis* var. *tritici*, which attacks wheat but not oats or barley; the other is the oat form, *G. graminis* var. *avenae*, which attacks oats and barley as well as wheat. The wheat form is much more common and widespread, permitting the rotation of oats or barley with wheat to reduce the incidence of the disease in wheat. The fungus has a wide host range in the tribe *Hordeae* of the family *Gramineae* and, under Australian

conditions, the most important non-cereal host is the introduced barley grass, *Hordeum leporinum*, on which the fungus survives vegetatively on infected roots. This and other susceptible grasses occur throughout the temperate regions of Australia. The fungus has been isolated from some of these grasses growing in virgin land where cereals had never been grown.[79]

The fungus has poor competitive ability as a saprophyte as it is readily antagonised by soil microflora. In the absence of a suitable host it will die out.[80] Use has been made of this fact in practising bare fallow of the soil between wheat crops, resulting however in the loss of the topsoil under Australian conditions. *G. graminis* survives in the vegetative stage as 'runner hyphae' on the surface of roots, but must continually invade living root cortex tissues which serve as a food base for the continued growth of the runner hyphae. However, the cells of the invaded root cortex are killed by the fungus, and are then rapidly colonised by secondary invading bacteria and fungi that are usually soil saprophytes. The result is that the food bases of *G. graminis* are destroyed and the fungus dies out.[81] Our present knowledge of the epidemiology of the take-all disease is clearly insufficient to explain outbreaks of the disease on virgin land sown to wheat, and the appearance of new foci of infection in the field. Thus, in the Netherlands, in newly drained polders of the Zuider Zee, there were severe outbreaks of take-all in successive wheat crops after ploughing up the natural sward.[82] Nor does it explain how the fungus survives over summer in parts of Australia where there are no living susceptible grass hosts growing during the hot, dry summer periods, but only fragments of grass straw and wheat stubble on the bare earth. Other possible modes for dispersal and survival available to the fungus have to be explored.

Asexual spore development is confined to the production of microconidia that so far resist all attempts to make them germinate, and apparently plays no part in the dispersal of the fungus in the field.[83] It should be stated here that until Drs Gretna Weste and L.B. Thrower, working at Melbourne University, made their discovery, *G. graminis* was not known to produce asexual spores. Ascospores (the sexual spores) are produced in perithecia formed in dead sheathing bases just above ground level, as has been known for a long time. This may explain both the dispersal and survival of the fungus under the conditions mentioned above. However, for a long time take-all investigators have ignored this possibility on the grounds that ascospore infection of roots could not occur because of antagonism from the rhizosphere microflora.

This was deduced from experiments by many workers who found that ascospores failed to germinate in other than a sterile environment[84], and the failure to produce ascospore infection of wheat seedlings growing in unsterilised soil by the application of ascospore suspensions. In 1972, Dr Weste at Melbourne University demonstrated that ascospores will infect root hairs and epidermal cells of root meristems in sterile conditions.[85]

Perithecia are formed, both in the laboratory and in the field, in infected wheat seedlings under suitable environmental conditions: the two most important ones being sufficient moisture and light.[86] Although ascospore infection in the field has yet to be proved, there is some circumstantial evidence of its occurrence. One piece of evidence is that under laboratory conditions variations in pathogenicity occur amongst isolates of the fungus. Such variations are known to originate usually from sexually produced ascospores. By dissecting out the eight long septate ascospores from a single ascus, I was able to demonstrate that genetic segregation for pathogenicity occurred during ascospore formation.[87] This was confirmed later at the Waite Institute by S.G. Chambers and N.T. Flentje, who found variations in pathogenicity among ascospore isolates.[88] Dormant perithecia in fragments of wheat straw may enable the fungus to survive during the dry summers that prevail over a large part of the wheat growing areas in Australia. Dried leaf-sheathing bases containing perithecia, which I collected at harvest time and kept in a dry condition in jars stored at room temperature for two years, produced viable ascospores when exposed to moisture. The same thing may happen in the field with the autumn rains in fields where wheat is sown. If ascospore infection does occur in the field, then the suggestion by Samuel and Garrett in 1933 that ascospores are a means of spreading the fungus in the field is supported.[89]

Take-all is a disease of new lands and a characteristic of the disease is that it is most severe in the first crop of wheat grown on land previously under pasture or virgin grassland. In succeeding years, if the land is sown continuously to wheat the disease subsides to a low level. If wheatlands are returned to pasture again with a legume component, there is a resurgence of the disease when replanted to wheat, because the legumes build up nitrogen levels in the soil. This has a twofold effect; it stimulates the predominance of susceptible grasses, and it favours saprophytic survival of the fungus on cellulosic residues in the soil.[90] There is, therefore, a build-up of inoculum in the soil.

The decline of take-all disease in successive wheat crops may be explained by the discovery in Australia, in 1975 by P.T. Wong of the N.S.W. Department of Agriculture, and in 1978 in England by M.J. Asher, that avirulent isolates of *G. graminis* protect wheat seedlings from infection by virulent isolates. The predominance of avirulent strains in successive wheat crops would lead to a decline of the disease.[91] Stimulation of the production of large numbers of *Pseudomonas* soil bacteria which attack the runner hyphae of the take-all fungus, causing it to die out, was reported in 1976 by R.J. Cook and A.D. Rovira at the Division of Soils, CSIRO in Adelaide. The pseudomonads do not survive in large numbers in soil under pasture that allows the take-all fungus to build up on susceptible grasses.[101] Another recent discovery is that of at least 8 mycoviruses in the hyphae of hypovirulent isolates of *G. graminis*, reported from Rothamsted in England in 1978. The mycoviruses are transmitted to hyphae of virulent isolates through hyphal anastomoses, causing a decline in virulence of the fungus, and thereby reducing the incidence of the disease in wheat.[92]

Phytophthora cinnamomi, the Root Rot of Native Forests
A disease of considerable ecological importance is caused by the soil-borne fungus *Phytophthora cinnamomi*, the cause of root rot and death of a large number of species of native woody shrubs and forest trees, as well as of introduced shrubs and trees. The fungus was discovered in 1922 by R.D. Randes who found it causing root rot in wild cinnamon trees in the mountains of eastern Sumatra. Since then it has become widely known in different parts of the world. It was first identified in Australia, eight years after Randes' discovery, as the cause of a disease in pineapples in Queensland. In 1948 Dr Lilian Fraser of the N.S.W. Department of Agriculture isolated it from the roots of native shrubs that died suddenly in undisturbed bushland near Sydney.[98] Within a decade, it was found to be the cause of heavy mortality in native forests and in suburban gardens, and extended from Southern Queensland through south-eastern Australia and into South Australia and Western Australia. However, it was not until the Plant Pathology Conference held at Toowoomba in Queensland in 1966 that the attention of Australian plant pathologists was alerted to the seriousness of the disease, through a paper given by F.D. Podger of the West Australian Forest Research Institute. There followed a rapidly growing number of investigations of the disease in different parts of Australia, so much so

that at the next Plant Pathology Conference held in Hobart in 1971 there were 18 papers on the disease and *P. cinnamomi* dominated the Conference.

The dramatic appearance of the devastating disease in Australia suggests either, that it was here 'simmering' all the time until the ecology of the natural forests was disturbed by man, or that the fungus had recently invaded Australia. Amongst the many investigators of the disease opinions were divided about which was right. In 1975, Dr C.J. Shepherd, of the Division of Plant Industry at Canberra, examined the evidence for and against these two divergent views. He stressed the relative frequencies of the two mating types, A1 and A2, present in the fungal populations within Australia, and in the Celebes-New Guinea region. He observed the general absence of plant mortality in north Queensland, compared with the greater incidence of mortality the further south one went. Shepherd concluded that *P. cinnamomi* had entered northern Australia along with the Indo-Malayan element of the flora from a centre of origin in South Eastern Asia. This migration is believed to have occurred about 1 to 2 million years ago, during the Pleistocene.[99]

The movement of the fungus southwards and into the West is believed by some to be of recent occurrence and facilitated by man, spreading of chlamydospores in soil from contaminated areas. Chlamydospore formation is an unusual feature of the fungus, and is stimulated by a decrease in soil moisture. Chlamydospore survival is increased by a low organic content of the soil, and a small content of soil micro-organisms.[100] Dr Weste of Melbourne University found a relationship between microbial populations in the soil and the activity of *P. cinnamomi*; areas with severe disease and rapid disease extension had a small soil microbial population compared with areas with moderate and slow extension of the disease.[101] The spatial spread of the fungus by means of chlamydospores is associated with road-making activities, disturbance of the natural drainage and the presence of man-made drainage channels.[102] In Western Australia, the Jarrah (*Eucalyptus marginata*) forest areas could have been saved from the ravages of the disease, if it had been known in 1945 that bulldozers, low loaders, and other heavy equipment, introduced into the forest for road-making and logging operations after the end of World War II, could transport the disease. The Western Australian Forests Department has now introduced forest hygiene measures to arrest the man-made spread of the disease. Many forests in the State are prohibited areas even for

walking, to ensure that chlamydospores adhering to boots will not be brought into the forests. Recently the practice of 'controlled burning' of forest litter has come under suspicion as a contributory factor in the incidence of the disease.

For local spread the fungus depends on asexual motile zoospores, produced in sporangia, and the germination of chlamydospores which build up the population to destructive levels. The short-lived zoospores, attracted by root exudates, penetrate and rapidly destroy root tissue, seriously reducing the ability of the plant to take up water. The effects of root destruction are most severe when drought follows a period of warm wet weather. The warmth and moisture encourage rapid zoospore production causing destruction of roots on a large scale, and the drought which follows puts the plants under stress with which they are unable to cope because of their reduced root system.

Nematode Plant Diseases

Like many soil-borne pathogens, most of the soil-borne nematodes parasitic on plants in Australia existed there before the advent of Europeans in 1788, and many are world-wide in distribution. In Australia by the 1950s the importance of nematodes as causes of plant disease was beginning to attract attention. This was in part due to the uncovering of their effects on plant health as diseases with other causes came under control. It also followed from a general awakening overseas to the importance of nematodes, as is revealed in the literature. Nematodes had not been entirely ignored in the past in Australia, for as far back as 1879, Dr Joseph Bancroft, a Brisbane medical practitioner with universal interests in parasitology, recorded the occurrence of the root knot nematode (now known as *Meloidogyne*), stating: 'this flask-shaped creature lives in the centres of the knotty growths in the root of vines, and the banana also. It lays great numbers of eggs which contain a worm, coiled up like a figure of eight'. [93] Then in 1890 in Sydney, Dr N.A. Cobb (who, it will be recalled, had obtained his doctorate from Jena on nematodes), in an article on *Tylenchus* and root galls, discussed the similarity with Bancroft's root knot nematode, tentatively naming it *Tylenchus arenarius*. [94]

Root knot nematodes were uniformly named *Heterodera marioni* until 1949 when B.G. Chitwood in U.S.A. revised the genus, renaming it *Meloidogyne*. [95] About 35 species of root-knot nematodes belonging to the genus have since been described. Only three of them are widely distributed in Australia, namely; *M. javanica*, *M. hapla* and *M.*

incognita. [93] The most common of them is *M. javanica*, except in Tasmania where *M. hapla* predominates. [93] What we now know as *Meloidogyne javanica* was the first species of root-knot nematode to be clearly identified. It was discovered on sugarcane growing in Buitenzorg in Java by Dr J. van der Vecht, and was subsequently named *Heterodera javanica* by M. Treub in 1885. Thus Bancroft had found it before van Vecht, in 1879. At the turn of the century, root-knot was identified in commercial crops in different States of Australia. Dr Alan Bird, of CSIRO's Division of Horticulture in Adelaide, considers that these records were most likely of *M. javanica*. Since then, root-knot has been reported on first crops from virgin land in W.A., Gosford, Liverpool and Leppington in N.S.W., and numerous areas of virgin soil on the Atherton Tablelands in Queensland. Dr Bird has found that *M. javanica* grows on native plants better than, or at least as well as, it does on crop plants, so that it and other species of *Meloidogyne* in Australia are to be considered endemic.

Many of the species and genera of the ectoparasitic root-injury and root-stabbing nematodes are also probably endemic in Australia, as are also some endoparasitic leaf nematodes belonging to the genus *Aphelenchoides*. Thus *A. oleosistus*, although world-wide in distribution, occurs on the widely distributed and indigenous bracken fern in Australia. However, not all plant parasitic nematodes are indigenous in Australia, and some have obviously been introduced along with their hosts, e.g. *Aphelenchoides ritzema-bosi* on chrysanthemums, *A. fragariae* on strawberries and species of *Xiphinema* that are vectors for grape fan-leaf virus, also introduced with the grapevine.

Some of the cyst-forming endoparasitic root nematodes belonging to the genus *Heterodera* appear to be indigenous in Australia. *H. graminis* on the roots of native and introduced grasses and *H. schachtii* on cereals are widespread in Australia. Fortunately, the potato root eelworm, *H. rostochiensis*, has not been introduced into Australia, mainly through the intervention of plant quarantine. Unfortunately, species of seed gall nematodes of grasses belonging to the genus *Anguina* were introduced quite early, mixed with seed. They include earcockle of wheat (*A. tritici*) and the seed galls of grasses (*A. lolii*) on rye grass (*Lolium rigidum*) and *A. agrostis* on Chewing's fescue (*Festuca rubra* var. *commutata* Gaud.). Earcockle in wheat was reported in N.S.W. by Cobb in 1892 and in Western Australia by R. Helms in 1896, but was probably present in Australia long before that.

The interesting etiology of a fatal disease of sheep and cattle associated with summer grazing on rye grass infected by the nematode, *Anguina lolii*, is currently being investigated by Dr A.F. Bird of the Division of Horticulture, CSIRO in Adelaide, and Dr B.A. Stynes, plant pathologist with the W.A. Department of Agriculture in Perth. The work was begun by Dr J.M. Fisher at the Waite Institute. So far it has been found that a species of *Corynebacterium* parasitizes the developing seed of Wimmera rye grass (*Lolium rigidum*) only when the larva of the nematode *Anguina lolii* is present; the nematode is believed to act as a vector by mechanically penetrating the developing seed and carrying the bacterium with it. The *Corynebacterium* invades the tissue of the ovule, resulting in the production of a toxin by the tissue reacting to the bacterial infection. This toxin is believed to be the cause of the fatal disease in animals grazing on the nematode-infected grass when in head.[96]

The bulb nematode, *Ditylenchus dipsaci*, was almost certainly introduced into Australia in infected bulbs of lilies and hyacinths. It is probable, but by no means certain that the citrus root nematode, *Tylenchulus semipenetrans*, was also introduced in soil and roots of infected citrus stock during the early days of colonisation. The nematode occurs wherever citrus is grown, and was first observed in California in 1912. Dr Cobb, at that time back in U.S.A., first described and named it as a new genus and species in 1913.[97]

Plant Viruses in Australia

Pioneer investigations in Australia on plant viruses were undertaken during the late 1920s by G. Samuel and R. Best at the Waite Institute, by J.G. Bald and H.J. Pittman in the CSIR, and by C.J. Magee of the N.S.W. Department of Agriculture. Samuel studied the effect of environment, mainly light and temperature, on local lesion development and collaborated with Best on virus inactivation, as well as participating in the foundation work on tomato spotted wilt virus, first with Pittman and then with Bald. Rupert Best was a biological chemist at the Waite Institute who developed a life-long interest in plant viruses, and was one of the first to isolate a plant virus in purified crystalline form in 1936. Best continued to pursue fundamental work on tomato spotted wilt virus, producing the first electronmicrograph, as well as demonstrating recombination of strains of the virus. Pittman and Bald participated in the tomato spotted wilt investigations, demonstrating the unique vector relations with the virus. Magee

investigated banana bunchy top virus and successfully controlled the disease in the banana-growing area of N.S.W.

The dependence of plant viruses on vector transmission introduces another dimension into the epidemiology of plant virus diseases. When a plant virus is introduced into a new geographic region such as Australia, a suitable vector must also be present for it to become established there. It is essential that the vector should move from plant to plant, feed on the sap and so transmit the virus. Pollen produced by virus-infected plants may also transmit virus to the stigma and ovule of the plant it fertilizes. Then the virus may become seed-borne. However, with some notable exceptions, few plant viruses are seed-borne. Most vectors are insects that transmit viruses to the shoot system, while certain root-feeding nematodes and root-infecting chytrid fungi that infect the roots by zoospores serve as vectors for some plant viruses. In Australia, such vectors are either introduced naturalised species, or they are indigenous. Undoubtedly, many viruses have been introduced into Australia but have not become established here because of the absence of a suitable vector: this applies especially to those viruses which have very specific vector relationships.

Until recently, indigenous plant viruses had not been found in the Australian native plants. The first to be found was a tymovirus[103], isolated from a legume, the dusky coral-pea, *Kennedia rubicunda*, by J. Dale and A. Gibbs, and named by them Kennedia Yellow Mosaic Virus (KYMV) in 1976.[104] Though the virus has been transmitted by mechanical sap inoculation to crop legumes such as garden pea, soy bean and *Phaseolus lathyroides*, it has not been found to have moved naturally on to any crop species, probably because its flea beetle vector does not feed on crop species. Three strains of KYMV have been found in native legumes along the eastern seaboard of Australia, each being separated from one another geographically.[105] The virus has not been found in Western Australia where there are native species of legumes on which it could occur.

Passionfruit woodiness virus (PWV) may yet prove to be another indigenous plant virus in Australia. It is thought that it has moved from one or all three of the native species of *Passiflora* on to the cultivated passionfruit, *Passiflora edulis*, which was introduced from South America early last century. Woodiness or bullet disease of passionfruit was first described and so named by Dr Cobb in N.S.W. in 1901 but was present in crops long before that.[106] In 1964 R.H. Taylor and K.A. Kimble, of the Victorian Plant Research Institute, found that two

unrelated viruses, cucumber mosaic and passionfruit woodiness (PMV), separately caused the same disease symptoms in edible passionfruit throughout the length of the coastal region of eastern Australia.[107] So far as is known, passionfruit woodiness virus occurs only in eastern Australia, although woodiness disease has been reported from Western Australia, South Australia, South Africa, Royal Kew Garden in England, and in Sumatra where it is believed to be caused by cucumber mosaic virus.

Though PWV has not yet been proved indigenous to Australia, there are strong indications that it is. There are three native species of *Passiflora* with distributions that overlap geographically over the length of eastern Australia, and considered to be components of an ancient invasion of eastern Australia by elements from Indo-Malayan flora.[108] They are *P. cinnabarina*, *P. herbertiana* and *P. aurantia*, the last extending in natural distribution into New Guinea, and to some of the western Pacific Islands. It is significant that there are no indigenous species of *Passiflora* in Western Australia, where woodiness disease in *P. edulis* is caused by cucumber mosaic virus.[109] Two of the native species have been found to be symptomless carriers of PWV, which suggests the virus is endemic in conformity with Vavilov's rule. The vector is the ubiquitous aphid, *Myzus persicae*.

Barley yellow dwarf virus which affects cereals and festucoid grasses in the grassland regions of the world also appears to be endemic in Australia. First recognised as a virus disease of cereals in 1951 in U.S.A.[110], it has since been recorded from most cereal growing areas of the world. It was first recognised in Australia in 1956 in a cereal variety trial at Tamworth in N.S.W. by a visiting New Zealand plant pathologist, Dr Harvey Smith, recently returned from U.S.A. and familiar with the disease.[111] Considering its widespread occurrence, it is surprising that it had not been discovered earlier. This was probably because the field symptoms can be easily confused with other cereal diseases, such as take-all. In fact, it was claimed in 1963 by R.D. Price and L.L. Stubbs of the Victorian Plant Research Institute that the symptoms of take-all in wheat were caused by barley yellow dwarf virus rather than the take-all fungus.[112] However, other workers, before and since, have produced take-all symptoms in wheat under controlled glasshouse conditions in the absence of insects and barley yellow dwarf virus.[113]

Barley yellow dwarf virus can only be transmitted by aphid vectors. All attempts to transmit it by seed and mechanical sap inoculation have

failed. That the virus is not seed-borne is of considerable significance in relation to its widespread occurrence in Australia. Nine species of aphid vectors are known, and most of these are world-wide in distribution. In Australia, the two aphid vectors most commonly occurring on cereals and grasses are *Rhopalosiphum padi* and *R. maydis*; both were probably in Australia long before it was settled by Europeans.[114] In Australia, the incidence of the disease in cereal crops varies from season to season with the climatic conditions that influence the population and movements of the aphid vectors. The severity of the disease depends on the strains of the virus present in the area and the susceptibility of the cereal hosts. The source of the virus inoculum is in festucoid grass hosts which include many native species that are often tolerant and symptomless carriers of the virus. This suggests a long association with the virus (Vavilov's Rule) whereas the introduced cereal hosts react severely to the strains present because they are newly exposed to them.

There are certain virus-like 'yellows' diseases of plants occurring in Australia that appear to be endemic. They were at first thought to be caused by viruses because they could only be transmitted by grafting or by the leaf-hopper vector, *Orosius argentatus*. The first of these was reported on tomato in 1933 by G. Samuel, J.G. Bald and C.M. Eardley at the Waite Institute of Adelaide, and named by them 'tomato big bud'.[115] In 1935, Dr E.T. Edwards of the N.S.W. Department of Agriculture described a disease he called 'witches' broom' of lucerne.[116] Then, in 1955, E.M. Hutton and N.E. Grylls of the CSIRO, described a disease in tropical legumes they called 'legume little leaf'.[117] All three diseases were transmitted by the same vector *Orosius argentatus*.

No recognisable micro-organism was found in the diseased issues of any of these diseases until 1969, when a group of workers in the Microbiology Department of the University of Queensland found mycoplasma-like bodies in the phloem of plants affected by legume little leaf, tomato big bud, and lucerne witches' broom.[118] In 1966, Doi and co-workers in Japan had found similar mycoplasma-like bodies in ultra-thin sections of sieve elements of 'mulberry dwarf' diseased plants, viewed under the electron microscope.[119]

The association of mycoplasma-like bodies with the 'yellows group' of plant diseases is now well established, though Koch's Rules of Proof have yet to be satisfied. A common feature of these diseases is the reversion of all floral parts to green leaf-like structures, rendering the plants sterile, so that mycoplasmas could not have been introduced by

seed into Australia. It seems more likely that the mycoplasmas are endemic in Australia, especially since the leaf-hopper vector for all of them is indigenous. However, so far no natural reservoir of the mycoplasma has been found in the native flora, though fasciations similar to those occurring on cultivated plants are frequently observed on native species.

Some plant viruses have been reported for the first time from Australia and at the time of reporting were thought to have originated here. Subsequently, they have been found to have been well entrenched in other parts of the world. Such virus diseases are tomato spotted wilt, banana bunchy top, lettuce necrotic yellows, carrot motley dwarf and broad bean wilt.

Tomato spotted wilt disease was first observed in a suburban garden in Melbourne in November 1915 and was given that name by Brittlebank when he reported the disease in 1919.[102] Within a few years the disease had become by far the most serious tomato disease in all States of mainland Australia and threatened the tomato industry. In 1927, G. Samuel and H.J. Pittman began working on the disease in Adelaide. Later J.G. Bald replaced Pittman, and it was found that the disease was transmitted by thrips and could be induced artificially by sap inoculation. The conclusion was reached that the disease was caused by a virus.[121] This was later confirmed by R.J. Best when improved techniques became available for studying plant viruses.

In 1931 Bald and Samuel reported that the virus had a wide host range including members not only of the tomato family *Solanaceae*, but also of other families.[122] Since then, other workers throughout the world have found that the virus has an exceedingly wide host range including members of at least 30 families of flowering plants. Unusually for plant viruses, they include both dictyledons and monocotyledons. Since the virus was first reported in Australia in 1919, it has been reported from almost every crop-growing part of the world. It is often disguised under different names depending on the host species. In 1890, a serious disease of tobacco and tomato was reported from the Kat River district of Cape Colony in South Africa. It became known as the 'Kat River disease'. In other parts of South Africa, the same disease in tobacco was named 'Kromnek' because it produced crooked necks in tobacco plants. Later these two diseases were identified with tomato spotted wilt.

In 1946, Dr D.O. Norris of the CSIRO in Canberra found that there were many strains of the virus, each of which caused different

symptoms, not only in tomato but in other host species belonging to different families.[123] The occurrence of strains accounted for the virus not being initially identified with tomato spotted wilt in other crops in different countries. Tomato spotted wilt virus is unique in a number of ways; it is the only plant virus so far known to be transmitted by thrips vectors (a discovery originally made by Pittman in 1928), and the infective thrips adult must have fed on virus-infected sap throughout most of its larval life. Electron microscope studies reveal a morphology distinct from that of other plant viruses and it is one of the few viruses which will pass from dicotyledons to monocotyledons.

The almost instantaneous widespread occurrence of tomato spotted wilt virus in epidemic proportions in Australia soon after the end of World War I, suggests that the virus was introduced about the same time at different points of entry in Perth, Adelaide, Melbourne, Sydney and Brisbane. Once introduced at these places, the virus would have been spread rapidly by species of thrips that had been abundantly present in these parts of Australia and were recognised as garden pests as far back as 1880. Although it will probably never be known how the virus entered Australia at these points, a possible connection with the end of World War I may be that disabled Australian servicemen were repatriated in ships that called at Capetown in South Africa on their way back to Australia. Returned servicemen began arriving in Australia at the end of 1914, and were disembarked at their home ports, beginning with Fremantle in Western Australia. It is well known that the repatriates brought back plant materials, including corms and bulbs collected at Capetown, where tomato spotted wilt virus was entrenched. Apparently plant quarantine restrictions were relaxed for the repatriates as they disembarked at each of the Australian ports, taking with them their South African mementos.

Tomato spotted wilt virus may have been introduced in infected corms and bulbs of such plants as Cape lily (*Crinum* sp.) and belladonna lily (*Amaryllis belladonna*) both of which were known to have been brought in from Capetown. Both lilies have been reported to be infected with the virus in both South Africa and Australia. The wide host range which provides many reservoirs and the seasonal abundance of thrips makes control of the disease very difficult. Over the years the incidence of the disease in Australia has waxed and waned, but it is less devastating than in its early years in Australia. The disease has been alleviated by growing tolerant varieties, but planting to escape the

thrips population during the height of summer has become a general practice.

Bunchy top of banana was introduced into N.S.W. in 1913 in infected banana suckers imported from Fiji at a time when the foundations of the banana industry were laid in the Tweed River district. In Fiji the banana industry was established in 1877 and by 1889 a new disease, now recognised as bunchy top, was already spreading in the plantations. The disease is believed to have been introduced into Fiji in 1886, when suckers of banana were brought there from the New Hebrides which is now believed to be the original home of the virus.[124] Banana planting in the Tweed River district expanded rapidly towards the end of World War I. This was partly a result of disruption of the importation of Fijian bananas to Australia brought about by shipping difficulties, and partly through the encouragement given to returned servicemen to adopt banana growing as a living. As planting progressed, so did bunchy top disease through the distribution of infected planting stock. Banana production reached a peak by 1922, but two years later the industry collapsed because of the ravages of the disease. This caused intense economic stress to the highly capitalised industry in the area.

In 1924, C.J. Magee, a recent honours graduate in Agriculture from Sydney, was seconded from the N.S.W. Department of Agriculture to the Interstate Banana Bunchy Top Investigations, to investigate the disease under the auspices of the then CSIR. Within the short space of two years Magee had demonstrated that the disease was caused by a virus disseminated by the planting of infected suckers, and that the banana aphid, *Pentalonia nigronervosa*, was the chief and probably only vector for the virus. The virus could not be transmitted by artificial inoculation, but was efficiently transmitted by both winged and wingless adult aphids after they had fed on diseased sap for at least 2 hours. They remained infective for several weeks. Although the aphid was an important agent in the natural spread of the virus, the part it played in the collapse of the banana industry was a minor one. A major problem was the large number of centres of infection established through the planting of infected suckers.[125]

These facts, together with the limited range of species of the genus *Musa* capable of acting as hosts to the virus and the absence of any native *Musa* species in the area, made possible the formulation of control measures for the Tweed River district.[126] This involved the registration of plantations, destruction of diseased plants and controlled

replanting with known disease-free stock implemented by gazetting Government proclamations and regulations in 1927. The control campaign was successful in rehabilitating the banana industry in that area through the co-operation of planters and their organisations. It provides an interesting example of field control of a virus disease, as well as an example of the successful application of legislation in plant disease control. Because of the constant surveillance still practised in the area it is difficult to find examples of bunchy top disease today.

Bunchy top virus has since been reported from the Philippines, India and Ceylon where indigenous and susceptible species of *Musa* occur, rendering it difficult to control by the measures adopted in the Tweed River district. The indigenous species of *Musa* serve as natural reservoirs for the virus where the aphid vector is also present. In the Philippines, abaca disease of the indigenous Manila hemp (*Musa textilis*), introduced in 1910, is identical with banana bunchy top. On the other hand, there are important banana growing countries where the disease does not occur, such as the West Indies, Central and South America, Canary Islands, New Guinea and most parts of Africa, including Egypt.

If a virus disease which requires a specific vector occurs in a geographic region in which the vector is known to have been introduced, the virus may also have been introduced. Two such viruses reported from Australia for the first time are carrot motley dwarf and lettuce necrotic yellows, both of which have aphid vectors which overwinter as dormant eggs on host plants that were introduced into Australia from Europe. The enigma is that in neither case has the virus been shown to be transmitted through the dormant eggs of their vectors.

Carrot motley dwarf disease, reported first in 1944 in Victoria by L.L. Stubbs and B.J. Grieve, had been observed as far back as 1910 by carrot growers in older suburban market gardens in Melbourne.[127] During World War II and as a consequence of it, carrot root and seed production was greatly increased throughout Australia. The crops were mostly grown in isolated rural areas where carrots had not been grown before. Carrot motley dwarf appeared in many of these areas in Victoria, N.S.W., South Australia, Tasmania and Western Australia. It is probable that the disease was introduced into these areas by infected root stocks brought from established vegetable growing areas.

By 1943 Stubbs, in Victoria, had established that the cause of the disease was a virus that was transmitted only and specifically by the

introduced and naturalised willow aphid, *Cavariella aegopodii*. This vector overwinters as dormant eggs on willow and on weed members of the carrot family, *Apiaceae*. In spring, the winged form of the vector is most active and feeds on any member of the carrot family, but transmits the virus only to cultivated and wild carrot (*Daucus carota*) under natural conditions. The virus was transmitted experimentally by infective aphids to some member of *Apiaceae*, including two native species, the slender celery, *Apium peptophyllum*, and sea celery, *Apium prostratum*, as well as the introduced hemlock, dill and coriander. Many members of *Apiaceae* were found to be immune to the virus, e.g. celery, parsley, parsnip and fennel (*Foeniculum vulgare*[128]). Stubbs found that the virus is not seed borne, and is not artificially sap-transmissible to carrot. The incidence of the virus in carrot crops grown from seed therefore depends on the presence of the virus in reservoir hosts and the activity of the aphid vector. During the summer months the aphid is not active, so that summer-sown carrot crops escape the disease. In Australia, healthy carrot crops are produced by delaying sowing until late spring when dispersal activity of the aphid vector is over.[129]

Lettuce necrotic yellows was reported first from Australia in 1963 by L.L. Stubbs and R.G. Grogan in Victoria.[130] The virus causing the disease is rare amongst plant viruses, being bacilliform or bullet-shaped. It was classified as one of the 14 plant viruses known as rhabdoviruses by Dr R.I. Francki of the Waite Institute.[131] The virus is not transmitted by seed of lettuce or any other plant so far tested. This is a characteristic of plant rhabdoviruses.

The virus is transmitted specifically by the aphid, *Hyperomyzus lactucae* which has a wide geographical distribution in Australia and is believed to be introduced. It overwinters in the dormant egg stage on currants (*Ribes* spp.) and may have been originally introduced into Australia as dormant eggs adhering to root-stocks of currants imported from Europe. The vector feeds and breeds on the introduced and naturalised weed, sow thistle, *Sonchus oleraceus* which occurs widely in Australia and is also the main reservoir host for lettuce necrotic yellows virus. Sow thistle is an ideal host for the virus because it does not show signs of infection even in the presence of high concentrations of the virus. The virus is transmitted from sow thistle to lettuce by the aphid (*H. lactucae*) and from sow thistle to the native prickly lettuce (*Lactuca serriola*) but not from lettuce to lettuce, or from prickly lettuce to lettuce, so that all lettuce infections come from the virus reservoirs in sow thistle. Therefore the control of the disease in lettuce is relatively

easily accomplished by eliminating sow thistles from the vicinity of lettuce crops.[132] This control measure has been shown to be very successful, first by Stubbs in Victoria in 1963 and then in 1970 by Randles and Crowley at the Waite Institute.[133]

A survey of lettuce necrotic yellows virus in South Australia, reported in 1970 by Randles and Carver, has shown that three newly-discovered naturally-infected host plants occur in restricted areas. They include two endemic species, *Sonchus hydrophilus*, and *Embergeria megalocarpa*. Both species are perennial, which would favour the survival of the virus, as well as supporting colonies of the aphid vector, suggesting that the virus may have originated in Australia, if it is assumed that lettuce necrotic yellows virus is distinct from similar viruses in North and South America, and in Europe.[134]

Broad bean wilt virus, discovered and so named by L.L. Stubbs in Victoria in 1947, is almost certainly an introduced virus. It is another virus that may be controlled by the elimination of reservoir weed hosts. It is transmitted by the endemic green peach aphid (*Myzus persicae*) and other species of aphids and may also be transmitted by artificial sap inoculation.[135]

The virus has a wide host range, including introduced weeds, such as *Bidens pilosa*, or cobbler's peg, and ribwort (*Plantago lanceolata*) which serve as reservoirs for the virus between the growing seasons for the susceptible legumes, broad bean, sweet pea, garden pea and N.Z. Lupin. The overlapping of the growing seasons of the weed hosts with that of the legume hosts provides a continuous infection chain for the virus, since the aphid vectors are most active at the overlapping points in autumn and spring. In Australia, the susceptible legumes are normally sown in autumn when weed growth has ceased. The aphids move from the senescent weeds to more succulent seedling legumes, carrying with them the virus if the weeds were infected. The virus causes a devastating disease in susceptible legumes and, in the Sydney Metropolitan area, caused complete failure of broad bean and sweet pea crops. In this area, ribwort was found to be the main reservoir for the virus. In the hills district, eradication of ribwort from nearby fields, headlands and gardens resulted in the production of healthy crops of legumes.

The sugar cane industry in Australia had its beginnings on the north coast of N.S.W. In the 1820s, cane setts were brought into Australia from sugar-growing countries, and with them came the virus diseases that were to plague the future industry. Such diseases as cane dwarf, Fiji

disease, grassy shoot, mosaic and ratoon stunting, were first investigated by David North of the Colonial Sugar Refining Company in N.S.W., and later by A.F. Bell, C.G. Hughes and D.R. Steindl for the Queensland Bureau of Sugar Experiment Stations. These diseases were assumed to be caused by viruses, because they were vector transmitted. Diseased setts were freed of the disease by heat treatment. Until recently none of the diseases had been associated with virus particles. Dr Francki and his students at the Waite Institute have since described the morphology of the Fiji disease virus.[138]

The same group of workers at the Waite Institute investigated the morphology of the virus causing Chloris striate mosaic disease which was first found and described in the introduced Rhodes grass (*Chloris gayana*) by N.E. Grylls of the CSIRO in 1963.[139] In 1979, Francki and his associates reported that the virus particles are polyhedral and belongs to a newly-discovered group of plant geminiviruses, the particles of which are arranged in pairs. In this character, Chloris striate mosaic virus resembles maize streak virus, first isolated in Africa (the original home also of Rhodes grass) suggesting that both geminiviruses may have originated there. It is characteristic of the geminiviruses that the particles are confined to the nuclei of the phloem parenchyma cells in infected plants and that the virus is transmitted by cicadellid leaf-hoppers.[140]

Another geminivirus has recently been purified and identified by Dr J.W. Bowyer and his student at Sydney University from French bean plants affected with 'bean summer death' and from tobacco plants affected with 'tobacco yellow dwarf' disease. Both diseases are transmitted by the leaf-hopper *Orosius argentatus* and are believed to be caused by strains of the same geminivirus.[141] Both the purified geminiviruses produced identical symptoms when transmitted to the French bean cultivar, 'Spartan arrow'.[141]

Viruses specifically affecting introduced fruit tree crops such as citrus, stone-fruits and pome-fruits were obviously introduced into Australia because there are no related native species that could serve as reservoirs for these viruses in Australia.[136] One such virus, 'scaly butt' or 'exocortis', was not revealed until a new budding stock was used in N.S.W. to overcome a problem with a fungal root-rot disease of citrus, caused by *Phytophthora citrophthora*. Root stocks of *Poncirus trifoliata*, immune to the root-rot fungus, were used instead of the susceptible stocks commonly used. Some ten years later, trees grown on the 'trifoliata' root stocks were observed to be stunted, a symptom

associated with scaling of the butt of the stock below the bud union. Some varieties like Washington navel oranges and Marsh grapefruit were more affected than others. Work carried out from 1944 onwards, by Dr Lilian Fraser and officers of the Horticulture Division of the N.S.W. Department of Agriculture, indicated that this condition was caused by a virus latent in the budwood which affected the 'trifoliata' root stocks. They named the virus 'scaly butt'; it was later identified with the 'exocortis' virus of U.S.A.[137] A citrus certification scheme was developed in which the citrus budwood was indexed for freedom from the exocortis virus. In recent years, Troyer citrange was imported and is now used instead of the 'trifoliata' root stock. It is resistant not only to latent exocortis virus but also to the root-rot fungus.

During the last decade subviruses, known as 'viroids', were revealed as causes of virus-like diseases in plants. They are ribonucleic acids (RNA) transmissible by grafting and sometimes through seed. Their detection was made possible through electrophoresis of sap from diseased and healthy plants in polyacrylamide gels, in which a particular band develops in the infected sap gel but is absent from the gel of healthy sap. Purified preparations of the infectious RNA are then made from the band. Viroids were first discovered as the cause of potato spindle tuber by T.O. Diener in 1971 in U.S.A.[142] The next year Semancik and Weathers in the U.S.A. found that exocortis disease of citrus was also caused by a viroid.[143] In Australia, viroids have been found to be the cause of Cadang-Cadang disease of coconut palm[144] and sun blotch of avocado.[145]

Some Investigations on Biological Control

Control of plant diseases through competition between avirulent strains of a pathogen, or provided by other micro-organisms, has long been investigated by many workers throughout the world with varying degrees of success. The decline of take-all disease in wheat in Australia through competition between avirulent and virulent isolates of the fungus *Gaeumannomyces graminis*, has been suggested from the work of Dr P.T. Wong who referred to it as 'cross protection'.[91] The survival of *Phytophthora cinnamoni* in soils has been shown by Dr G. Weste to be greatly influenced by the intensity of soil microfloral activity.[101]

The successful control of bacterial crown gall, caused by *Agrobacterium radiobacter* var. *tumefaciens*, in stone fruit trees through competition from the non-pathogenic *A. radiobacter* var. *radiobacter* was reported by Dr A. Kerr of the Waite Institute in 1972.[146] The

soil-borne crown gall bacteria cause a troublesome disease of fruit trees and nursery stock, by inciting gall development on their roots and stems. It was probably introduced into Australia on nursery stock brought from Europe in the early years of settlement.[147] Crown gall bacteria live saprophytically in the soil in the absence of hosts and, once established in a nursery, are virtually impossible to eradicate. Their presence has resulted in some instances in the abandonment of rose and stonefruit nurseries.

In 1969, Kerr found that at low ratios of pathogenic (*A.r.var. tumefaciens*) to non-pathogenic (*A.r.var. radiobacter*) forms, gall induction can be completely inhibited.[148] An ecological study showed that the pathogenic form biotype 2 was present in the soil near galled almond seedlings, where it formed a significant proportion of the total biotype 2 population. The pathogenic form was not detected in soil near healthy almond seedlings; only the non-pathogenic form was present.[149] From these studies and glasshouse experiments Kerr deduced that, in the field, gall induction depends on the size of the non-pathogenic population relative to that of the pathogenic form at the wound site, as well as on the inhibitory efficiency of the non-pathogen. Significant biological control of crown gall was achieved when the ratio of pathogen to non-pathogen was 1:1, whereas when the ratio of 1:8 was used, there was a reduction of 76% of crown gall infections.[150] Much of the success of this work was due to the development of a selective medium for *A. radiobacter* (biotype 2) which permitted a quantitative estimate of the bacterium in the soil and crown gall tissue.[149]

The successful control of black spot, or scab, of apple which is caused by the fungus *Venturia inaequalis*, through stimulation of microbial activity by a pre-leaf-fall spray of 5.0% urea solution was reported in 1965 by K.E. Hutton of the N.S.W. Department of Agriculture, and his English colleagues at East Malling Kent.[151] There are two phases in the infection chain of the black spot fungus, viz:- a parasitic phase on apple foliage and fruit during the growing and fruiting season of the host in spring and summer and a saprophytic phase on the fallen apple leaves during the dormancy of the host from late autumn and to the end of winter. The parasitic phase is initiated by ascospore infection, from ascospores released from perithecia developed in stroma formed beneath the old lesions in the dead leaves on the ground at the end of the saprophytic phase.

For many years protective spray programmes were applied to the emerging foliage in early spring, in an attempt to prevent infection by

ascospores released from the overwintering leaf litter. The cost of such programmes, and the uncertainty of success because of unfavourable weather conditions, induced Hutton to seek eradicant methods that would prevent the development of the source of ascospores in spring. Overseas workers had tried unsuccessfully to eradicate ascospores by spraying leaf litter with ground sprays of various chemicals. Failure was mainly due to the impossibility of contacting all the fallen leaves with the chemicals. Hutton developed the concept of post-harvest, pre-leaf-fall sprays as an eradicant treatment, and found that phenyl mercuric chloride sprays at 0.3% concentration eliminated the saprophytic phase of the fungus and the source of ascospores in the spring.[152]

By 1963 the post-harvest, pre-leaf-fall spray was widely used by growers in N.S.W. In that year, Hutton went to East Malling Research Station in Kent, England, for a year to work with R.T. Burchill and H.E. Crosse on eradication control of black spot of apple. While there, they became interested in urea as a possible non-toxic replacement for the toxic phenyl mercuric chloride as an eradicant. Their interest in urea was aroused through two publications one of which was from Canada, where *in vitro* studies had shown that excess nitrogen suppressed perithecial formation in *Venturia inaequalis*. The other was from Norway where post-harvest pre-leaf-fall sprays of urea were used as a means of supplying nitrogen to fruit trees.

Hutton, with the East Malling group, carried out experiments with urea as an eradicant spray for black spot. They found that a maximum reduction of 97% infection was obtained when 5.0% urea was applied as a post-harvest, pre-leaf-fall spray. The urea hastened the decomposition of the leaf tissue and so prevented the formation of stroma and perithecia of the fungus.[151] The replacement of urea as a post-harvest spray for the toxic phenyl mercuric chloride was opportune because, by the 1970s, all mercury salts were banned from agricultural usage in Australia.

Concluding Remarks

The author did not intend to write a complete coverage of the history of plant pathology in Australia for such a coverage would itself need a book rather than a chapter in a book. However it is regretted that space does not permit more than the mention, to select only a few, of such fundamental studies as that on latent infection in tropical fruit by J.H. Simmonds in Queensland, the influence of the soil chemical factors of oxygen and carbon dioxide in the ecology of soil fungi by Professor

D.M. Griffin, at the time when he was Reader in plant pathology at Sydney University, the initial work on phytoalexins by Drs K.O. Muller, I.A. Cruikshank, and D. Perrin in the Division of Plant Industry, CSIRO at Canberra, and the many investigations on host-pathogen relations in plant disease which have been carried out in an attempt to elucidate disease resistance mechanisms in plants, by as many workers as there are Institutions investigating diseases of plants in Australia. Other more applied investigations deserving a place in the history of plant pathology in Australia have been omitted. Among them are: the initiation of bean seed certification for halo blight and anthracnose by the late R.D. Wilson of the N.S.W. Department of Agriculture, the tissue culture work of J. Sutton in freeing potato clones from viruses in Tasmania, and Victoria, now at the Victorian Plant Research Institute and the mineral deficiency work done by C.R. Millikan of the same Institute. There are now over 250 plant pathologists actively engaged in some aspect of plant pathology in Government institutions, at universities and in private industry in Australia.

The following abbreviations will be used:

APDR: Australian Plant Diseases Reporter
 APPS: Newsletter: Australian Plant Pathology Society Newsletter
 APP: Australian Plant Pathology
 JAAS: Journal of the Australian Institute of Agricultural Science
 JCSIR: Journal of the Council of Scientific and Industrial Research
 TBMS: Transactions of the British Mycological Society

- [1] It has been held that because of the nature of the indigenous flora the Aborigine was forced to be a hunter-gatherer with no form of settlement or cultivation such as had been developed elsewhere in the world at that time. But see the Chapter on the botany of the first Australians.
- [2] Stripe rust of wheat (*Puccinia glumarum*), a serious disease in the cooler countries of the Northern Hemisphere, has never become established in Australia, probably because it cannot survive the high temperatures of Australian summers.
- [3] The downy mildew of tobacco, *Peronospora tabacina*, is indigenous in Australia, especially on native tobacco, *N. suaveolens*, which occurs throughout Australia. The common native rust, *Puccinia lagenophorae*, which occurs on native species of Asteraceae infects introduced members of that family. Cf Wilson, Walshaw and Walker, 1975, *T.B.M.S.* 48, 501.
- [4] Obligate fungi such as *Ovulariopsis*, which causes powdery mildew of some native legumes, *Uromykladium* rusts of *Acacia*, and *Cionothrix jacksoniae* which causes rust of native wedge peas.

- [5] Grape downy mildew fungus, *Plasmopara viticola*, has moved on to native grape (*Cissus hypoglauca*) and the late blight of potato fungus (*Phytophthora infestans*) has moved on to native species of the genus *Solanum*, such as kangaroo apple (*S. aviculare*).
- [6] Before the breaking up of the ancient land mass into the fragmented and separated land masses as we know them today.
- [7] This fungus was found on native orchids, *Dendrobium* spp. on the remote and uninhabited Whitsunday Islands off the North Queensland coast.
- [8] See (30).
- [9] Waterhouse, W.L. *Proc. Linn. Soc., N.S.W.* 61, 5.
- [10] Fish S. 'Daniel McAlpine, A Pioneer Plant Pathologist in Australia'. *A.P.P.S. News Letter*, 5, 11, 1976.
- [10a] Cobb was an inventive genius who was too little understood at that time.
- [11] McAlpine published 5 monographs as well, on diseases of citrus, stone fruits, potatoes, rusts of Australia, and smuts of Australia. In all of them he described new species of fungi. Thus in his rust monograph he described 161 species of rust, and 75 of them were new.
- [12] Blanchard, F.C. *Asa Gray Soc. Bulletin* 3, 1205, 1957.
- [13] There is little doubt that Watt's students learnt more from this text, and the 25 plant diseases they collected and identified during that year, than they did from the 5 lectures he gave.
- [14] Such as: D.B. Adam, S. Fish, A. Pugsley, A.V. Hill, J.G. Bald, C.R. Millikan, G. Wade, N.T. Flentje, L.L. Stubbs, L.B. Thrower and Gretna Weste.
- [15] Waterhouse stressed the importance of cultural methods and techniques used to establish Koch's Rules of Proof.
- [16] He graduated with 1st class honours, and medal; the first of many medals he was to be awarded.
- [17] Waterhouse was so dedicated to serving the needs of plant pathology in Australia, that he declined the offer by Sir John Farmer in 1923 to become Associate Professor of Plant Pathology at Imperial College, London University.
- [18] Some training in plant pathology is undertaken by some of the new universities, but the older universities have maintained their major role in the training of plant pathologists at both under-graduate and post-graduate levels.
- [19] Flentje began his career in plant pathology when appointed to D.B. Adam's staff in 1944, when he had just graduated in Agriculture from Melbourne. Subsequently, he spent a lot of time overseas, including 2 years at the Imperial College, London, where he graduated PhD. He was a fine tenor and delighted in singing Schubert Lieder, such as the song cycle, Die schöne Müllerin. He became Deputy Vice-Chancellor of Adelaide University.

- [19a] During this period (1953-71), N.H. White was head of the sub-department. He graduated BSc from Sydney in 1938, where he was a student under T.G.B. Osborn, and W.L. Waterhouse. He had experience in research in plant pathology with the CSIR at Canberra (1938-43); later was plant pathologist in the Tasmanian Department of Agriculture (1943-46), before joining Waterhouse's staff. 'His infectious enthusiasm inspired many students at Sydney to specialise in plant pathology.' He was capably assisted by D.M. Griffin (1955-70), and C.D. Blake (1963-70).
- [20] Such journals as *Australian J. Agricultural Research*, *Aust. J. Biological Sciences*, and *Aust. J. Experimental Agriculture and Animal Husbandry*.
- [21] By the Victorian Government in 1875, N.S.W. and Queensland governments in 1887.
- [22] Samuel had transferred from the Botany School in the University of Adelaide, where he had taken over the duties of plant pathologist from Professor T.G.B. Osborn in 1920. He participated in the discovery of manganese deficiency as the cause of Grey Speck disease in oats, at a time when minor element deficiencies were beginning to be investigated. (See Chapter by G.W. Leeper)
- [23] In 1951, Dr C.G. Hansford replaced Dr Adam, who had died, as head of plant pathology at the Waite.
- [24] Dickson was first appointed Chief Mycologist and Plant Pathologist, but a year later he was made Chief of the Division. He arrived in Sydney in 1928, when he and his nucleus of staff were housed temporarily in the Botany School at Sydney University, until the new laboratories were completed at Black Mountain in Canberra in 1931.
- [25] Angell was a former student of Dickson's at McGill, and a PhD from Wisconsin.
- [26] Dr Frankel, an Austrian by birth and education, was knighted in 1966. He had been Director of Crop Research in the DSIR in New Zealand.
- [27] Muller had begun his researches on phytoalexins, so named by him, in Germany before World War II, and had continued at Cambridge during the Hitler regime in Germany.
- [28] In the 1966 census of plant pathologists, there were in each State the following; Victoria 29, N.S.W. 28, Queensland 24, South Australia 12, W.A. 10, and Tasmania 5.
- [29] The first of these conferences was held in Melbourne in 1949, the next in Sydney in 1955, in 1961 at Adelaide, and in 1966 at Toowoomba in Queensland.
- [30] David Collins: *An Account of the English Colony in N.S.W., 1798 and 1802*. Reprinted by Reid, Sydney 1975.
- [31] See [9].
- [32] Watson, I.A. & Cass Smith, W.P. *J.A.I.A.S.* 28, 279, 1962.
- [33] Waterhouse, W.L. *Proc. Linn. Soc. N.S.W.*, 54, 615, 1929.
- [34] Walker, J. *Proc. Linn. Soc. N.S.W.*, 79, 145, 1954.

- [35] 'Lambrigg' was a wedding present to Farrer from his father-in-law de Salis of Duntroon, when Farrer married his daughter in 1885. See the Chapter on Wheat Breeding.
- [36] Stakman, E.C. & M.N. Levine. *Univer. Minn. Agric. Bulletin* 8, 1922.
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- [42] It was on the site now occupied by Sydney University, that the first record of a stem rust epidemic was made by Capt. Tench in 1795.
- [43] The Editor of *Nature* returned the manuscript which was to have announced their success as a 'Letter to the Editor', on the grounds that they had not produced enough evidence that it was, in fact, the rust fungus that was growing on the medium.
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