PRESIDENTIAL ADDRESS

On the Pathology of Arborescent Plants

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I count it a great honour that I should be called upon to give my Presidential Address at the first conference of the Australasian Plant Pathology Society to be held in New Zealand. When our Society was formed in 1969 as the Australian Plant Pathology Society, it immediately attracted members from outside that one country — and most significantly from New Zealand. The links rapidly strengthened further and in 1980 it was decided to rename the Society so that New Zealanders became, as it were, first-class members. The Society now owes a great debt to those in New Zealand who have organized this Conference, to Dr. Sanderson who has undertaken editorial responsibilities for a section of our journal in recent years and, of course, to an earlier President, Dr. R. Close.

The links between our two countries are numerous and have existed for many years. As long ago as 1909, an eighteen year-old New Zealander left his home in Otago to widen his experience in Australia. In about eighteen months, he picked apples and felled trees in Tasmania, cut cane in Queensland, helped on an orchard in New South Wales, took charge of a camel-train and worked in a vineyard in South Australia. As though that were not enough, he also tried prize fighting in Sydney. This young man was no other than G.H. Cunningham, who was to become a great pioneer in plant pathology and taxonomic mycology. Virtually self-taught, he applied himself with distinction to both basic and applied research the quality of which was recognized by the award of high academic honours and the fellowships of the Royal Societies of both New Zealand and London. His applied research was mainly addressed to the problems of fruit-growers in his native land but his taxonomic research focussed on both Australian and New Zealand members of the Gasteromycetes, Polyporaceae and Thelephoraceae.

Cunningham gave some attention to the *Diplodia* disease of pines and was thus an early contributor to the forestry industry that is now of such significance to the New Zealand economy. It is therefore perhaps appropriate if in this Presidential Address I focus on matters relating to the pathology of trees.

Neither 'tree pathology' nor 'forest pathology' clearly denotes my topic, because the former gives undue emphasis to the individual (probably unintentionally), the latter conjures up childhood visions of a 'forest'. In the past, it is true that ecosystems with a closed tree canopy have been the forester's particular concern but the roles of trees in open or modified formation in rural, range or urban settings is assuming long-overdue prominence. The forestry profession is thus concerned with the management of all ecosystems in which trees are significant components. Similarly my current concern relates to the pathology of arborescent plants, by which I mean to include both trees and shrubs, wherever and however grown, singly, in plantation or natural communities. Surprisingly, I have not found any published consideration of the fundamental factors which differentiate the pathology of arborescent perennials from those of the annuals or herbaceous perennials which are the main focus of agricultural plant pathology. I shall therefore try and identify factors which distinguish the sub-discipline of what I shall be forced to call 'forest' pathology, for want of a better term, from its general parent discipline. This is a topic that my colleague Dr. W.A. Heather and I have discussed on so many occasions in varied contexts that I can no longer remember with whom a given thought originated. I pay tribute to him: in fairness he ought to be joint author but that is not permissible in a Presidential Address. He has been a sustained and penetrating thinker on matters of forest pathology and I greatly regret that my Department will lose his services when he retires in a few weeks time.

The overwhelming features of arborescent plants are the size of the secondary body with its associated great spatial separation of the sites of photosynthesis and nutrient uptake and their extended perenniality. To leaves and roots as major organs susceptible to disease must now be added the above and below-ground portions of the secondary body whose integrity is essential for the good health of the plant. Within the secondary body, there are both living and dead tissues of great diversity and all must be protected from being overwhelmed by microbial attack, parasitic or saprophytic, for many, even hundreds, of years. Only in arborescent perennials is the resistance of dead tissue to microbial attack of great significance to the health of the living organism.

The first line of defence of the secondary body, whether it be stem or root, is the periderm and rhytidome. Anatomically, these tissues are complex, consisting principally of phellum, phellogen, phelloderm and phloem, living, dying or dead. An even more complex structure is proposed by recent Canadian work. Much of the literature suggests that the response of the phellogen and other living tissues to damage of many origins is of the nature of 'healing'. This view is too restricted, however, and the response may be far more active and antagonistic to the invader. Unless I am much mistaken, remarkably little research has been done on resistance mechanisms, *sensu lato*, located in the periderm and rhytidome and certainly no comparative studies on genera representative of the differences to be found in these layers. Even within the single genus *Eucalyptus*, great and obvious differences exist between species characterized as 'gums', ironbarks, stringybarks and so on, often with more than one bark type on a single specimen. Active responses by the living cells are known to exist but the resistance to biodegradation of the phellem and rhytidome is also of great importance. This resistance lies in both the presence of toxic chemicals and the difficulty experienced by most microorganisms in splitting and utilizing many of the complex biopolymers. The unavailability of suberin as a substrate to all but a few fungi is particularly noteworthy, no doubt to the relief of both the wine industry and the palaeobotanist, for whom cork is one of the most frequently fossilized tissues. Few microorganisms appear to be able to penetrate the rhytidome unaided. Most species attack through natural or artificial wounds and even such as *Fomitopsis an-*

nosa, which can penetrate intact cork after enzymatic weakening, infects far more rapidly if wounds are present. *Armillaria* spp. are able to split the suberin molecule itself and this, combined with the presence of rhizomorphs, permits regular penetration of the intact rhytidome by members of the genus.

If the periderm is breached by a pathogen, it is faced by the phloem, cambium and outer parts of the secondary xylem. Species of *Armillaria* have a marked predilliction for initial growth in the cambium, as does *Endothia parasitica*. From there, deeper invasion takes place. Any organism invading the sapwood meets a range of active host responses which, at their most effective, lead to physical and chemical compartmentalization so that the invader is walled-off and restricted to wood present at the time of initial invasion. Although recent work has demonstrated the great importance of compartmentalization, much yet remains to be learnt of the intricacies of host-pathogen relations within the massive and complex secondary body.

The secondary body differs between tree species in many characteristics, of which the presence or absence of resin duct systems and of heartwood are of particular note. Enhanced resin production and exudation is so frequent a sign of, and response to, injury that its involvement in resistance is readily assumed. Detailed knowledge of the significance of resin in pathology is, however, surprisingly scant. Far more attention has been paid to the role of phenolic extractives in heartwood. Much is now known about the relation between age of heartwood and concentration of extractives and of the range of sensitivity *in vitro* of wood-destroying fungi to these often potent chemicals. In standing trees, however, decay of the old central heartwood still remains an intracticable problem of great economic consequence and is frequently a forerunner of termite damage. Tree species which have high resistance to decay when grown in a native forest, often show a much reduced resistance when grown in plantations. This is related to the generally faster growth of trees when grown in managed plantations. With increasing age, the extractive content of heartwood increases, reaches a plateau and then decreases again. Wood in which the increase is occurring is said to be juvenile wood in distinction to mature wood. Size for size, fast-grown trees therefore have a higher proportion of juvenile wood than slow-grown trees. As trees are generally felled at a given merchantable size rather than a particular age, the wood of fast-grown trees tends to have a markedly increased susceptibility to decay both in the standing tree and after conversion.

The secondary body, of course, provides not only the structural scaffold for the plant but also the means for transfer of solutes and water between the various plant components. Given the presence of an extensive and persistent vascular system, it is not surprising that wilt diseases, induced by vascular colonizers are important in trees. Often, the main sites of invasion are at root or twig tips where the xylem is least protected by overlying tissues, although root grafting is also important. Oak wilt and dutch elm disease are well-known examples of tree wilts and have in recent years been joined by various diseases caused by *Verticicladiella* and *Chalara*. Lying within the xylem, the pathogens are well-protected from attack by man and cause amongst the most serious of diseases.

A comprehensive understanding of tree pathology is complicated by the succession of pathogens which may invade a single tree during its long life. To think of extremes, susceptibility to damping-off is soon lost but invasion by some heart-rot fungi will occur only after physical damage and then be limited to old heartwood where the content of antifungal extractives is low. Changes in susceptibility as a tree ages can be attributed both to factors intrinsic to the host itself and to those involving the environment. Thus severity of some leaf diseases change as a tree becomes an emergent and breaks free from the higher humidity and lower light intensity below the general canopy. The individual is thus subjected to many waves of pathogens some at least of which are characteristic of only one growth stage of the host. Even for these, the tree is usually exposed to attack for many years as it goes through its annual cycle of ontogenetic development. Here the difference between determinate and indeterminate branches is of importance. In the former, characteristic of most orchard species, the number of leaves to be produced by a branch in a season is determined by the number of primordia laid down in the bud. In such species, there is a pronounced seasonal rhythm of leaf production, maturation and abscission which is also reflected in pathogen activity. Susceptible host tissue is present only at certain seasons and a break in the disease cycle occurs automatically. Indeterminate branches, however, have no such preordained number of leaves in each growth period but continue growing whilst conditions are favourable. *Eucalyptus* provides a good example of a genus with indeterminate branches whilst *Populus* possesses both determinate and indeterminate. In such genera growing in clement climates, new highly susceptible leaves are always present and the disease situation is much aggravated. Thus 'evergreen' cultivars of *Populus* in low latitudes suffer severely from rusts of the genus *Melampsora*.

The size of a tree makes infection by a number of different organisms at the one time a likely occurrence. Often, the invaders will have no significant effect but sometimes two major pathogens will occur simultaneously. Then an evaluation of their interactions in affecting plant growth is a complex, biometrical task. Deleterious effects on trees can also arise from the associated activities of insects and a pathogen to an extent equalled in agricultural pathology only in the case of virus diseases. Thus a number of tree wilt diseases involve insects which, during their maturation feeding, carry the pathogen from previously infected sapwood in which the larvae have developed to new young shoots. Pine wilt disease is transmitted in a similar way, although here the pathogen is a nematode, *Bursaphalenchus xylophilus*, with a predilliction for resin ducts. The combined effects of *Sirex noctilio* and *Amylostereum areolatum* on pines is also well known in New Zealand and Australia.

Most natural forests exhibit great complexity of structure and species composition. In agriculture, this complexity is approached only in the village gardens of Indonesia and similar wet tropical regions. Although when studying epidemiology, the cereal pathologist, for instance, may find that he must take into account nearby wild grasses, it is only with heterocyclic rusts in agriculture that it is usual for a non-economic host to be of major significance. With arborescent plants, however, infection of non-economic species in a forest is often of great significance when considering disease

in a valued species. This is particularly so with diseases of roots where, for instance, species of *Armillaria, Rigidiporus* and *Phellinus* are often present on both economic and non-economic trees. Even plantations do not escape this problem as citrus growers and rubber planters know to their cost. The situation is aggravated by the very long period of saprophytic survival of pathogens in the massive residues of dead trees. Periods in excess of fifty years are not uncommon for a number of basidiomycetes.

The vastly differing time scales of the life-cycles of trees and their pathogens have profound implications. At first sight, it is a wonder that forests of limited species diversity, whose individuals have a genome determining resistance to attack fixed for tens or hundreds of years, survive at all against the onslaught of pathogens of enormous variability, short life-cycle and high potential rate of change. Clearly, trees must be well-served by the relative impenetrability of periderm and cuticle and by generalized physiological responses to invasion such as compartmentalization. In many cases, however, the stability of the ecosystem probably depends more upon the great range of host genomes present, even in forests of but a few species, rather than on the resistance of each individual.

Monoculture plantations are obviously much less diverse in structure and species composition than natural forests yet frequently they have escaped serious damage from pathogens for long periods. Most existing plantations have originated from many seed parents, however, and thus continue to contain a wide range of host genomes relating to resistance. In the past, clonal populations of trees were largely limited to horticulture, with notable exceptions in *Salix, Populus* and *Hevea*. Casual observation suggests that such populations are more susceptible to disease than are populations of mixed genotype. It is therefore not surprising that some forest pathologists, myself included, worry at the implications of clonal forestry which has become a reality with the ready ability to produce clonal material, from cuttings or tissue culture, of many forest species. Fortunately, the potential for disaster is also being appreciated by some forest managers and I am told that in Sweden it is now a rule that no more than 250,000 members of a clone may be planted and that each plantation must involve at least 120 clones.

Many plants exhibit an increased susceptibility to infection when grown in environments of higher humidity than that found within the native range. Trees are no exception, and the pink disease of eucalypts, caused by *Corticium salmonicolor*, is a good example. Almost unknown in Australia, the disease is an important limitation to the growth of eucalypts in such hot, humid climates as Kerala. The introduction of a species to an arearemote from its natural distribution may also expose it to pathogens to which it has very poor resistance. In this lies the origin of the spectacular outbreaks of disease in *Pinus strobus* caused by *Cronartium ribicola* after an introduction of that pine to Europe. The unintentional introduction by man of a pathogen into a new area may have equally spectacular results as evidenced by the recent transfer to Europe from North America of an especially virulent strain of *Seratocystis ulmi*, a pathogen which in a less virulent form had earlier been transported in the reverse direction with dire impact on American elms. Agricultural plant pathogists experience similar problems, of course, but with annual plants the possibilities of overcoming the problem by plant breeding are very much greater.

If a tree is to have a long life at a given site, it must be able to tolerate not only the usual environment but also those events of relative rarity that occur: perhaps a severe frost, an exceptional drought or flooding. The environmental factors which together define the boundary of distribution of a species are thus hard to specify, and within a large complex group such as *Eucalyptus* are particularly baffling. This has a number of implications for pathology. Thus many species in climates given to periodic aridity are possessed of a larger root system than that required for water uptake in the normal season. The full system is, however, required at times of drought. As first demonstrated for *Eugenia* attacked by *Valsa eugeniae* in Zanzibar, such trees undergo a slow or even no decline during clement seasons when attacked by a root pathogen but suffer sudden death, even within a few days, when heat and drought strike. Then, the reduced root system is no longer able to meet the demand for water induced by enhanced transpiration.

The environment, however, does not affect only the host and all pathologists will be aware of the disease 'triangle', which indicates that all the interactions between host, pathogen and environment affect the incidence and severity of disease. Unfortunately, the details of this triangle are often difficult to determine for diseases of trees, so great are the number of variables and possible interactions. This is well-exemplified by the disease of many arborescent plants in Australia caused by *Phytopthora cinnamomi*. Only now is the situation being elucidated. Some factors, such as high soluble aluminium ion concentrations, seem to act mainly on the host — and then differentially between hosts, so affecting susceptibility. In fact, susceptibility to infection as such may not be of great importance. Rather, it is the ability to restrict or compensate for infection that mainly determines whether overt disease expression occurs. Other factors, such as temperature and soil organic matter contact (acting via microbial antagonism), appear to affect mainly the pathogen. Sometimes, however, it is a complex interaction between host, pathogen and environment which determines disease. Soil water seems to be such an environmental variable.

A major group of pathology issues directly relate to the value of the economic product. Considering arborescent perennials as a whole, products of economic value may become available annually, periodically, essentially at the end of a rotation or as any combination of these. Traditionally, annually produced fruits, the pathology of which was a major interest of Cunningham, have been seen as the province of the horticulturalist and it is probably appropriate if they remain largely outside the purview of the forest or tree pathologist. The nature and control of fruit diseases in most cases lies closer to agricultural rather than forest diseases in most cases lies closer to agricultural rather than forest pathology because of the high annual economic value of the product. Other annual or periodical products are leaves, resin, latex and cork. The collection of all involves damage to the plant which can be exploited by pathogens. The pathological effects of lopping, coppicing and pollarding, in particular, are poorly known for the many species so treated in tropical and sub-tropical countries.

The final product is wood itself, whether used as timber, veneer, chips, particles or pulp. Here value can be severely affected by microbial activity in the wood in the standing tree, or in the post-harvest log or in use. Talking in very rough figures, about ten percent of the annual value of agricultural crops is thought to be lost due to microbial attack on the living plant. The corresponding figure for forestry is double — over twenty per cent. The reasons for this are manifold and some reflect historical and economic rather than biological factors, but the latter are very significant. Even now, regrettably little can be done to reduce wood degradation in the standing tree: prevention by care in avoiding physical damage is far easier than control once a fungus is established. When wood is the final product, usually after many decades of growth, the cumulative impact of relatively small annual losses in productivity averaged across a population with diverse genomes, becomes significant although, truth-to-tell, the losses have rarely been accurately measured. It would seem, however, that repeated partial defoliation or loss of feeder roots must affect growth. Forests experience a natural rotation whereby the death of one individual provides space for the growth of replacements. The period of this natural rotation may be very great but may be shortened by disease, giving rise to the concept of a 'pathologically-controlled rotation'. The felling rotation is normally comparatively short when compared with the potential life of the tree and is determined by the interaction between economic factors and a declining mean annual increment. Disease can affect this period, too.

In the foregoing, there is much that implicitly relates to the control, or more importantly the avoidance, of disease. Control, in any absolute sense, is very difficult in forestry for both biological and economic reasons. In most forms of forestry, the annual increment in economic value is relatively small but the final value of the crop, often after many decades, is high. Such a situation does not encourage annual extensive spraying, for instance. Spraying is a normal practice only in three situations. The first is to eliminate a newly-introduced foliar pathogen from a restricted area. The second is in those unusual instances, such as *Dothistroma* on pines, where spraying at relatively long intervals is adequate. Finally, annual spraying may be justified if the economic value of the product is particularly high as in the case of rubber. Otherwise control or avoidance of disease by manipulation of the stand is the indicated strategy and here forest pathology differs very significantly from horticultural or agricultural pathology. None-the-less, forest pathology provides an outstanding example of biological control in the prevention of stump colonization by *Fomitopsis annosa* through inoculation with *Peniphora gigantea*.

In my Daniel McAlpine Memorial Lecture five years ago I reflected on other very general aspects of the inter-relationships between forest management, disease and socio-economic factors and I shall not cover that ground again now. Rather I shall conclude by expressing my thanks to the Society for having elected me as its President two years ago and thus affording me this opportunity to provide a bird's-eye view of some aspects of forest pathology.

Australian Plant Nematodes: New Records of *Criconemella* and *Discocriconemella* (Nematoda : Criconematidae).

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Various criconematids or ring nematodes were collected in a study of plant-parasitic nematodes associated with naturally occurring vegetation in Australia. These collections represent new locality records. The nematodes were identified from specimens mounted in glycerol.

In 1981, *Macroposthonia* De Man 1880, *Criconemoides* Taylor 1936 and *Xenocriconemella* De Grisse & Loof 1965 were placed in one genus *Criconemella* De Grisse & Loof 1965, by Luc and Raski (4). The type species of two genera, *Criconemoides morgensis* (Hoffmanner & Menzel, 1914), Taylor 1936 and *Macroposthonia annulata* De Man 1880 are both considered as *genus et species dubia*. With the names *Macroposthonia* and *Criconemoides* unavailable, the name *Criconemella* has been retained, following the rule of priority. In the localities which follow N.P. represents a National Park.

Criconemella curvata (Raski 1952) Luc & Raski 1981 syn. *Macroposthonia curvata* (Raski 1952) De Grisse & Loof 1965.

Originally described from California, this species appears to be uncommon in Australia and previously had been recorded only from Queensland (3). Pilliga East State Forest, N.S.W. Dominant vegetation includes *Callitris columellaris* F. Muell., and *Allocasuarina luehmannii* (R. Baker) L. Johnson.

Criconemella macrodora (Taylor 1936) Luc & Raski 1981. syn. *Xenocriconemella macrodora* (Taylor 1936) De Grisse & Loof 1965.

This appears to be a cosmopolitan species and has been recorded from U.S.A., India, Africa, Europe, Papua New Guinea (1) and Australia (3). Within Australia records to date are few and confined to Queensland. This is one of the most frequently occurring species of criconematid in soils collected from eastern Australia. It appears to be more often associated with woodlands and forests rather than rainforests.

Eucalyptus woodland and forest: Forty Mile Scrub N.P., Qld.; 57 km west of Mt. Garnet, Qld; Kurrimine N.P., Qld; Ravensbourne N.P., Qld; The Knoll N.P., Qld; Nundle State Forest, N.S.W.; Cumberland State Forest, N.S.W.; North Rocks State Forest, N.S.W.; Jenolan State Forest, N.S.W.; Morton N.P., N.S.W.; Murramarang N.P., N.S.W.: Pigeon House Mountain, N.S.W.; Ben Boyd N.P., N.S.W.

Melaleuca woodland: Kurrimine N.P., Qld.

Casuarina littoralis Salisb.woodland: Eungella N.P., Qld.

Tropical rainforest: Eungella N.P., Qld.; Ravensbourne N.P., Qld.

Criconemella onoensis (Luc 1959) Luc & Raski 1981 syn. *Criconemoides onoensis* Luc 1959. syn. *Macroposthonia onoensis* (Luc 1959) De Grisse & Loof 1965.