

MONOCULTURE, SPECIES DIVERSIFICATION, AND DISEASE HAZARDS IN FORESTRY

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ABSTRACT

The large-scale establishment of even-aged pure stands, commonly known as monocultures, often arouses serious objections on grounds of increased disease and pest risks. A critical survey of literature showed little to support such a view; serious diseases have occurred in mixed and exploitable natural forest, while there is no clear evidence that outbreaks of diseases in pure stands can be ascribed to lack of species diversity. The theoretical basis of such a view is also questionable. There are, first of all, no definitions that adequately characterise typical epidemics, mixtures, or pure stands, so it is hardly possible to make broad generalisations about the effect of stand composition on disease. The very concept of disease occurrence viewed, say, as a "triangle" of host, pathogen, and environment, precludes overemphasis on any one factor in isolation from others.

An undisturbed natural forest or ecosystem may be stable within a certain period. The principles governing such stability are still little known, but it is certainly reasonable to assume that an understanding of such principles is highly important to our forestry practice. It can be questioned, though, whether they are all applicable to man-made systems. At present there is little reason to assume that any departure from "nature" must increase disease hazards. Besides, the use of monocultures need not entail sacrificing the tree-to-tree genetic variation that provides some protection from major disease losses.

There is a tendency to exaggerate the advantages of diversifying into a range of major production species to the point of ignoring its disadvantages, difficulties, and its sheer practical unrealities in some countries. For example, increased importation of planting material for diversification may increase disease risks by introducing pathogens. We have witnessed this over the last two decades. Moreover, with a greater number of tree species and therefore of potential disease problems, one must either increase the cost of

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plant protection or give less attention to any one species. The role of diversification in pathology can be very negative if the ultimate goal of pathology is to allow us to grow a good healthy crop where and when we want it.

INTRODUCTION

Ninety percent of New Zealand's wood production comes from only about 750 000 ha of plantation forests, largely of a single exotic species, *Pinus radiata* D. Don. This compact forestry industry earned around \$400 million in foreign exchange in 1979. The large-scale establishment of plantations consisting of a single species, generally known as monoculture, is not favoured by some foresters; in fact there are commonly serious doubts and objections to it for a variety of reasons, among which disease risk is a major one.

We are all too familiar with de Gryse's (1955) warning: "... to ignore the notorious susceptibility of *P. radiata* to insects and fungi, the extreme vulnerability of the extensive monoculture in which it occurs ... is tantamount to challenging all the laws of Nature". Our foresters' concern about this problem has been well expressed and documented in the various symposia organised by the Forest Service (Burdon and Thulin, 1966 pp. 47, 49-51, 76; Weston, 1971 pp. 15, 17, 19, 20, 22, 32, 51, 247, 253, 254; James and Bunn, 1978 pp. xiii, 105, 115-16).

Throughout the world such concern can be traced back to the early days of plantation forestry, and indeed the very beginning of modern forest pathology. Robert Hartig, father of modern forest pathology, wrote at the opening of his book (Hartig, 1894):

During the present century, and especially during the last few decades the forests of Germany have been threatened with dangers of a magnitude formerly unknown. These have been occasioned by the gradual relinquishment of natural regeneration, and by the substitution of pure even-aged woods for woods consisting of trees of different species and of various ages, but most of all ... displacement of broad-leaved trees by pure coniferous woods.

A report entitled "The relation of stand composition to crop security" prepared by the Committee on Silviculture of the New England Section of American Foresters and endorsed by the noted American forest pathologist, J. S. Boyce, perhaps marked the first "majority vote" on the dangers of monoculture in recent time (Anon., 1939). This view was reiterated in greater detail by Boyce (1954) in a paper written for FAO. Both documents were pessimistic about the long-term success of intensively managed

pure stands, as well as exotics, on the basis of "ample proof", being "axiomatic", or a matter of "principles". As Boyce (1954) said:

Pure stands are more susceptible to diseases, particularly those caused by introduced parasites than mixed stands A pure stand is ideal for a pathogen to build up to epidemic proportions The most hazardous are pure, even-aged stands The chances are strongly against the long time success of pure stands unless a species naturally forms such stands.

Such views were described by the noted British forest pathologist Peace (1957) as "condemnation of monoculture", "whole-hearted acceptance of a partial truth as a principle", and "belief not based on proper investigation of the individual plantation, the site and the disease influence present or potential, but on vague generalisation about disturbances of the biological balance, silviculture unnatural to the site or ecological inadequacies bound to lead to diseases".

In recent years forest pathologists have written more cautiously about the risks of pure plantations. Patton (1962), in discussing plantation disease problems with American foresters, expected more disease problems in the future from a consideration of principles associated with the inherent structure of plantations. In his opinion some of these principles deserved re-emphasis, but others could stand some de-emphasis. Heather and Griffin (1978), examining the epidemic potential of eucalypt plantations in Australia, concluded, "In planning eucalypt plantations in Australia it is essential to avoid generalisations regarding the risks of disease". Schmidt (1978), discussing the relationship of ecosystem diversity and forest diseases, said, "An important point is that diversity in itself is no safeguard against pathogens, especially introduced pathogens". The views of others may be somewhat ambiguous and perhaps even baffling to the non-pathologists.

In a recent review on forest monoculture as the origin of pests and diseases, Gibson and Jones (1977) deplored the exaggerated risk of monoculture with the following comments: ". . . such arguments have frequently been overstated to the point of asserting that all attempts to develop forest crops using intensive management systems on a large scale are doomed to failure. Indeed, it is surprising how these views have persisted despite the evident success of 'tree farming' in most places where it has been tried." However, it seems puzzling that the following remarks appear at the conclusion of their paper: "If we are now to answer the question 'has the adoption of monoculture systems led directly

to an increase in the number and severity of pests and diseases of forest crops?' the answer would have to be 'Yes' It appears that the most pessimistic forecasts of traditional foresters on the dangers arising from forest monocultures have been fully vindicated However, this conclusion needs qualification. We have already observed that few if any, of the most disastrous outbreaks of forest pests and diseases, the classic cases, can be attributed to the introduction of monoculture systems." This they ascribed to the compact nature of plantation crops providing clear advantages for the application of effective control measures.

In a recent treatise on plant disease, Cowling (1978) made the following remarks:

Another illustration is the large-scale replacement of genetically diverse native forests with plantations of exotic pines in New Zealand, Australia, East and West Africa, and Chile. This practice has drastically increased the genetic vulnerability of the forest resources of these nations and regions of the world. The current epidemics of *Dothistroma* blight of *Pinus radiata* in all these regions [*sic*] provides persuasive evidence of the hazards inherent in this forest practice.

Dothistroma is actually of little significance in the main *P. radiata*-growing area of Chile, and in Australia the disease was found only in the last 5 years or so, and no serious or widespread outbreaks have been reported.

No wonder our foresters are confused; this is epitomised by Fenton (1978, p. 105):

With regretful respect to the pathologists, having read a lot of this literature over the last 3 months, I must say that it doesn't appear to be quite logical . . . in the past we have diversified into species other than our main ones . . . we have already lost a fortune on diversifying . . . We planted, justified on the grounds of reducing the risk from absent and unknown pathogens.

Fenton then legitimately raised the question whether or not these forecasts represent a deduction from a law, a theory, or a hypothesis on the basis of appropriate data, or the acceptance of dogma from books as in law or religion.

It seems that the question of monoculture in relation to disease hazards warrants further review, particularly with regard to New Zealand.

PURE vs. MIXED STANDS

Stand composition may well have a significant effect on disease occurrence, hence on crop security, but the question is: Can such an effect be simplified into a generalisation of pure vs. mixed or

mixed being safer than a pure? Clearly not. The so-called "abundant proof" (Anon., 1939) supporting such a generalisation did not stand up to critical examination by Peace (1957) who pointed out that, in many of the cases he examined, disease increase in pure stands was likely to be caused by the choice of wrong species, wrong provenances, wrong site, and a host of other factors, rather than the effect of stand composition. Clearly, to be valid a piece of evidence must not only demonstrate that a pure stand has more disease trouble than a mixture containing the same species but, more importantly, that disease difference is occasioned solely by stand composition and not by other confounding factors. This requires that the two stands, one pure and one mixed, to be compared should be, for all practical purposes, identical in every way (site, management, climate, genetic base of the host species, etc.) except stand composition. A comparison between a natural mixture and a pure stand established from one of its component species can be fraught with difficulties. To begin with, management practices could hardly be the same in the two situations. From the point of view of "system" (natural or man-made) the two stands would be basically different, as will be discussed later. In many situations the effect of stand composition is unknown because of inadequate comparisons. Thus in New Zealand the "pure stand effect" of *P. radiata* on disease occurrence, if any, is simply unknown; it is just as unfounded to say that the origin of a particular disease (*Dothistroma*, for example) is intimately related to monoculture as to say that it is not related. Until such time as it is shown conclusively that the disease can be discouraged by mixed planting, the matter remains unresolved.

Is a pure stand ideal for a pathogen to build up to epidemic proportions, as Boyce (1954) suggested? The answer is "It all depends". It depends on the pathogen, the host species and, perhaps more important, the varieties or provenances, the site, the management practices, the climatic conditions, etc. This is quite evident from our basic concept about disease occurrence — *i.e.*, the "disease triangle" or the inter-relationship between host, pathogen, and environment that determines the outcome of a disease situation. It is a conceptual mistake to consider the "pure stand effect" independently of the other factors affecting disease. The idea that a pure stand favours the spread of a pathogen from tree to tree has an element of truth, but has limited application, and to deduce from this that a pure stand is more hazardous and that a mixture is safer is a gross oversimplification of the complexities of a disease situation. There is no such thing as a

general or typical epidemic — the varieties of epidemic in plant pathology are infinite (van der Plank, 1960). We can say the same thing about mixtures and pure stands. How mixed is a mixture, and how pure is a pure stand? How are we going to define these things and how can we assign stand composition an overriding effect on disease occurrence?

A pure stand is often mistakenly equated with dense stocking and, as disease is generally favoured by greater proximity between plants, increased disease problems in pure stands are thus inferred. Yet the suggestion that there is a greater diversity of disease and pest problems under nursery conditions than in out-plantings, and that nursery or trial plots may be taken as "primitive precursors" of extensive monoculture (Gibson and Jones, 1977), seems hardly a tenable argument. Besides, the relationship between planting density and disease intensity is not simple. Some diseases are more influenced by dense planting than others; these are called "crowd diseases", which may be controlled by mixed planting (van der Plank, 1960). The theoretical aspect of the relationship between the abundance and distribution of host plants, and epidemics has been dealt with by van der Plank (1960) who pointed out that the least exploited method of reducing plant disease is by planning the pattern of farming in directions other than crop rotations. Thus, he wrote "the paradox is this, bringing plants together into fields increases the chance of epidemic; bringing them still further together, by increasing the area of the fields and correspondingly reducing their number may reduce the chance of a general epidemic".

The validity of a generalisation lies in its wide application, so let us now examine whether mixtures are generally safer. Obviously mixtures are not necessarily safe when a pathogen can attack several host species, and Peace (1957) reminded us that chestnut blight destroyed American chestnut in both mixed and near pure natural stands. Let us not forget that chestnut blight is perhaps the only known example of a pathogen which has virtually obliterated a plant species in its natural habitat. Another extremely destructive disease is that caused by *Phytophthora cinnamomi*, the host range of which covers 444 species belonging to 131 genera of 48 families including both gymnosperms and angiosperms (Newhook and Podger, 1972). Thus, in Australia not only the eucalypts are attacked in their natural stands, but a great variety of understorey species are also devastated.

The host range of *Armillaria* is equally impressive — 677 species belonging to 276 genera including both conifers and

broad-leaved trees (Raabe, 1962). Even obligate parasites such as rusts are not restricted to a single host species, and for those which require alternate hosts to complete their life cycle, the host species usually belong to quite remotely related genera; for example, *Cronartium fusiforme* requires oak and southern pines. There are three rusts in the native stands of *P. radiata* in California (Offord, 1964); two of them require alternate hosts which we do not have in New Zealand; on this basis, *P. radiata* would be healthier here than in its native home.

Our knowledge about the host range of a pathogen is often very incomplete, and pathologists would be hard put to prescribe a safe mixture. Powdery mildew (*Oidium hevea*) of para rubber (*Hevea brasiliensis*), a serious disease in rubber plantations in Ceylon and unknown in the natural habitat of rubber trees in the Amazon jungle, originated from a herbaceous plant *Euphorbia pilarifera* indigenous to South-east Asia (Young, 1949). The host range and origin of terminal crook (*Colletotrichum acutatum*) is not quite clear, although blue lupin was suggested to be a possible source (Dingley and Gilmour, 1972). One could produce many such examples, but these suffice to illustrate how impossible it is to assure crop security just by using mixtures.

Consider another situation. If species A is highly susceptible to pathogen X and species B is moderately resistant to it, then it is possible that B may suffer more severe attack by X when the two species are brought together in a mixture, because B is now under the threat of a higher inoculum potential. Peace (1957) discussed the greater severity of *Fomes annosus* attack of hardwood species when they were mixed with conifers. If we are to mix *P. radiata* with hardwood species, we must first consider a possible increase of *Armillaria* and *P. cinnamomi*, among other unknown and unforeseeable dangers. It is an oversimplification that any "mixture effect" on disease occurrence would be merely a non-host barricade effect on the pathogen. Peace (1957) cited how *Mycosphaerella laricina* attacked larch more severely when it was mixed with spruce, because the falling needles of infected larch were intercepted by the spruce and thus infection droplets from these trapped needles could reach the larch crown much more easily than from the forest floor.

From a management point of view, pure even-aged stands facilitate disease control. On the other hand, selective logging as it must be practised in mixed uneven-aged stands, may create its own disease problems. The *P. cinnamomi* problem in Australia is at least partly related to such a practice. Tree decline

and mortality due to *Armillaria* in selectively logged eucalypt forest in central Victoria (Edgar *et al.*, 1976) is another example. A serious disease problem in Germany today, recently seen by the writer, is wound rot caused by the use of big machines during selective logging of mixed forests.

Let us examine further the complexity of stand composition effects on disease epidemics through two examples.

The fusiform rust (*Cronartium fusiforme*) of southern pines in the United States, a rarity in natural stands before 1900, is now epidemic in slash and loblolly pine plantations in the southern United States, and was quoted as an example of dangers of pure stand plantations in the Silviculture Committee Report of 1939 (Anon., 1939). The commercial southern pine forests, of which slash and loblolly are the main constituents, in 1970 totalled about 78 million ha or 14% of the forest land of the southern United States, and produced one-third of the softwood timber and two-thirds of the pulpwood harvest (Dinus, 1974). Loss due to fusiform rust was estimated to be 99.4 million cu. ft or \$28 million in 1972 (Dinus, 1974). The rust fungus alternates between pine and oak hosts. About 26 species of pine and 20 species of oak have been listed as hosts; among them, for our interest, is *P. radiata* (Czabator, 1971). Both the hosts and the pathogen are indigenous to the United States. The rise of the disease problem is intimately associated with the serious disturbance and destruction of the original southern pine forests (a fire sub-climax) by Europeans. Excessive logging with near-total removal of longleaf pine, land-clearing for agriculture, fire control, mechanical site preparation, and a host of other management practices have drastically altered the distribution and relative abundance of species in the southern pine forests so that the highly susceptible slash and loblolly pine have gradually occupied a more extensive area than previously through planting and natural regeneration; in the meantime, these factors also favoured an increase in abundance of the alternative hosts intermixing with the susceptible pine. Thus the stage was set for an epidemic. It should be noted that slash pine grows naturally as a pure stand, and monoculture in itself cannot be blamed for the epidemic (Schmidt, 1978). Schmidt (1978) gave seven reasons for the increase of this disease in plantations, which can be summarised as:

- (a) The use of diseased seedlings.
- (b) The use of genetically susceptible material (species or variety).

- (c) Increased association with alternate hosts through wrong siting or other management practices.
- (d) Predisposition or non-genetic increase of susceptibility (fertiliser, change in age distribution, etc.).

For our second example, let us look at South American leaf blight (SALB) of para rubber (*Hevea brasiliensis*), frequently quoted as a classical example of disease hazard associated with the use of pure stands. SALB, incited by the fungus *Microcyclus ulei*, is generally considered to be the cause of failure to establish rubber plantations in tropical America before and during World War 2 (Langford, 1945; Hilton, 1955; Holliday, 1970). Rubber trees belonging to nine different species of *Hevea* are sparsely distributed (at the most a few trees per hectare) in the jungles of several South and Central American countries where the pathogen is also indigenous but is apparently doing little harm to the host species. When *H. brasiliensis* was grown under plantation conditions in tropical America, disastrous attack by the fungus ensued and many thousands of hectares of rubber plantations had to be abandoned. In Malaysia, Indonesia, Ceylon, and other parts of South-east Asia where the pathogen is not present, the rubber industry has thrived, but the pathogen poses a serious threat. If the pathogen arrives there, as seems inevitable, will it mean the end of the rubber industry? The crucial question is: Does the survival of rubber trees or the absence of serious SALB attack in native stands depend on the sparseness of host distribution? Undoubtedly, crowding of plants under plantation conditions may have played an important part in the outbreak of the disease, but was this the only factor in the failure of the plantations? There is first the fact that the material used to establish the tropical American plantations was obtained mostly from Malaysian plantations which were established with progenies originating from only 22 seedlings of *H. brasiliensis* planted in the Botanical Garden of Singapore in 1877 (Chee, 1977). There is also ample evidence now for the existence of resistant genes in the wild population of *Hevea* spp. (Chee, 1977). Therefore, there seems to be a connection between a narrow genetic base of the planted host population and the serious outbreak of this disease, and there is hope that the disease may to some extent be controlled through the use of more resistant planting material. There is no reason to believe that in the long run, with progress in chemical control and other methods of disease control, we cannot grow rubber in pure stands in the presence of the pathogen.

So far as South-east Asia is concerned, there is still hope that exclusion of the pathogen or its eradication after arrival may be possible.

Finally, let us consider the long-term effects of pure stands. What is the effect of continuous cropping on disease?

In view of the shorter history of plantation forestry it is better to consider this a wide open question. In agriculture with regard to annual crops, the consensus has been that crop health would generally deteriorate under continuous cropping because of the accumulation of soil-borne pathogens, and crop rotation is the normal practice to combat such a problem. Even such a long-held view has been challenged recently by Shipton (1977): "Only recently has the traditional respect for the system (crop rotation) been challenged to the extent that suggestions have been made that rotation might now have become obsolete and that monoculture might be more appropriate to modern conditions." In forestry and other perennial crops there are undoubtedly genuine cases of "replant disease" or "second-rotation decline" (Savoy and Durkee, 1956; Chu-Chou, 1978). The question is, of course, how general is the phenomenon, and is crop rotation or mixed planting the only solution to the problem? It is better to say we do not know, and advise that more effort should be devoted to the study of such a problem, and as soon as possible before our grandchildren accuse us of short-sightedness.

NATURAL AND UNNATURAL

The origin of the condemnation of monoculture was traced to certain general forestry or naturalistic theories which include the idea that the natural forest is the ideal environment, and that any departure from nature must encourage disease development (Peace, 1957). No doubt there is some truth in such ideas but, as Peace (1957) pointed out, the very gloomy picture that many have painted of the future of forestry in Western Europe and particularly in Great Britain has been based on an uncritical acceptance of those items. The planting of pines in the Southern Hemisphere may appear to put them way out of their natural range. However, the lack of pines in the Southern Hemisphere is merely an accident of the plate tectonic history of the earth, not because the environment is unsuitable for them. Prehistoric migration of pines southward occurred despite geographic barriers (Mirov and Hasbrouck, 1976). So the planting of exotic pines here is perhaps not as unnatural as one might have thought.

A distinction must be made between natural phenomena and natural laws. Natural phenomena are governed by natural laws which are certainly inviolable. Unnatural, if understood as artificial or man-made, surely represents a departure from nature, but not necessarily violation of natural laws. Such things as aeroplanes and test tube plants can work very well. As Peace (1957) remarked, "unnatural is not necessarily synonymous with unhealthy". What could be healthy and fit in a natural stand from a tree's point of view, may not necessarily be desirable or healthy from Man's point of view. Large branches and precocious development of abundant cones betoken a sign of health and fitness for survival in a natural *P. radiata* stand. Man would normally consider such characteristics undesirable. Clearly not all survival values of a tree species in a natural stand are of use to us or essential in plantations. Elimination of competitors (pathogens, pests, and other higher plants) is a main feature of an artificial ecosystem. Then we apply fertilisers, prune and thin, and we may irrigate. Perhaps most important of all is the modification of the genetic make-up of the host population in cultivation through breeding and selection; this is domestication or artificial evolution (Robinson, 1976).

These various practices certainly affect disease occurrences but, as pointed out previously, we know too little about their overall effect on diseases. Some diseases may be encouraged, and others discouraged; this is as broad a generalization as we can make. An understanding of how natural ecosystems maintain stability will surely help us greatly to improve our pest and disease management programmes, but it is questionable that the principles governing the stability of a natural ecosystem are all directly applicable to a man-made system. There is certainly no reason to assume that the natural system is the best one, that it cannot be modified or improved, and that it is applicable in all circumstances. Besides, we know, regrettably, very little about how the natural system maintains its stability. We assume that it is maintained by a variety of mechanisms (Browning, 1974; Schmidt, 1978), of which the best known is genetic resistance (Leppik, 1970; van der Plank, 1963, 1968).

Most importantly, the use of monocultures need not entail sacrificing the tree-to-tree genetic variation that undoubtedly provides substantial protection from disease epidemics in natural stands. It should be pointed out, though, that components of a natural forest can also be defenceless against an exotic pathogen with which the host species have not co-evolved and thus have

developed little or no resistance. Chestnut blight, Dutch elm disease, and white pine blister rust are well-known examples.

There is a theory derived from the study of natural succession that the stability of a community and/or its constituent species is positively related to its diversity. The application of such a theory to man-made systems has been questioned (Apple, 1977; Way, 1977). Van Emden and Williams (1974) commented: "Although pest outbreak is often regarded as a general consequence of reducing diversity through monoculture, it is surprisingly hard to find documented examples". It seems absurd, from our point of view, that indiscriminate addition of species can lead to stability. Our plantations are better without some of the alternate hosts of rusts present in the native *P. radiata* stands. The damage caused by brush-tailed possums, deer, and rabbits in our forests is also well known.

DIVERSIFICATION AND DISEASE RISKS

The advantages of diversity of natural resources are simple enough for anyone to perceive. If we have several major species instead of one, then, in the event of arrival of a highly destructive pathogen, there is a possibility that only one or two species are affected and that the loss is partial. The socio-economic impact of chestnut blight was not as great as one might have thought (Hepting, 1974), because the United States is richly endowed with alternative timber species. However, for countries whose timber supply depends on a limited area of plantations, the question is not whether one desires but whether one is able to diversify into a range of major species. There are two requirements for such a scheme to work. First, the species chosen should have roughly equal value (productivity/utility), so that there would be no major productivity loss through planting less of any of the species. Secondly, the chosen species should share as few common pathogens as possible. It should be noted that the idea of such a scheme is to reduce loss of one's investment owing to failure of any particular species by reducing its importance in the overall timber supply or its size of planting. The scheme would not reduce the disease risks within any one particular species.

To prevent or reduce disease loss within a species one must achieve capability of combatting diseases. It should be realized that diversification is not without disadvantages and difficulties. With more species there are bound to be more disease problems. hence one must either increase the defence budget, or give less attention to each individual species. For New Zealand and for

many other countries, increased diversification could mean importing more material for planting, which in turn could increase disease risk by introducing new pathogens. The forest pathogen most feared in New Zealand, western gall rust (*Endocronartium harknesii*), is widely distributed on many pine species in the United States, Canada, and Northern Mexico (Lowe and Ziller, 1971). The disease may be introduced on these pines, or even on contaminated non-host species growing in the disease region. A needle rust of Scots pine (*Coleosporium sonchi-arvensis*) was introduced into Wisconsin from Europe when seeds of Norway spruce contaminated with infected leaves of sow thistle (*Sonchus asper*), an alternative host of the rust, were imported (Boyce, 1954).

It is only fair to say that we have made a considerable effort to diversify our forests and, as Fenton (1978) put it, it has cost us a fortune, but we have not yet found even a second-best species. It is unreasonable to expect us to plant a species which has more disease problems and at the same time is less productive and profitable than *P. radiata*. The planting of *P. ponderosa* and *P. nigra* has been suspended in most places, their high susceptibility to *Dothistroma* being one of the reasons. Douglas fir used to be our second-best species, but it has lost favour for various reasons including pests and disease problems. Our enthusiasm for poplar must have been dampened a bit now with the arrival of the rust (*Melampsora larici-populina*) followed by another dangerous pathogen, *Marssonina* — and larch is an alternate host of poplar rust. We are wary of rushing into growing cypress on a large scale before there is some answer to the canker problem (caused by *Monochaetia unicornis*). There is a growing interest in eucalypts, but from the pathology point of view we are nearer to the source of indigenous pathogens than either Brazil or South Africa, and therefore stand at a disadvantage. These difficulties in diversification confronting us with regard to pest and disease problems must be realised. We should note that *Dothistroma*, though undesirable, is by no means disastrous to *P. radiata*. The disease has increased our costs; since 1966 we have sprayed an average of 28 000 ha per year and the present cost is \$8.22 per hectare. But although representing a formidable total this is still small in relation to aggregate growing costs. In fact, the annual plantings of *P. radiata* have increased steadily since the arrival of *Dothistroma*.

It is debatable how to define diversification. Our major production species is *P. radiata*, but over the years we have acquired

experience with a variety of different species, and have been trying new ones all the time. Our exotic forest plantations were largely established on currently unforested areas that were not suited for agriculture. Our natural forests were replaced first by sheep and cows, not by exotic tree species. In fact, the establishment of fast-growing exotic species has enriched our natural resources; it plays a role in conserving our remaining natural forest, hence the genetic vulnerability of crops in the country could be decreased rather than increased. It is unfortunate that this situation is often not well understood and is misrepresented by some overseas observers.

Although diversification into a range of major species has its advantages, it also has its difficulties and disadvantages. In the long run if one cannot grow one species well, what is the hope of growing more than one? The ultimate goal of plant pathology, as Bawden (1970) put it, is to allow farmers (foresters too) to grow safely the crops they wish, where they wish, and as often as they wish. From this point of view the role of diversification in disease risks can be a somewhat negative one.

CONCLUSION

There is no doubt that successful forestry practice or a sound pest and disease management programme should be based on an understanding of principles governing the stability of natural ecosystems. Regrettably our present knowledge is still inadequate for making broad generalisations about the effect of various forest practices on the maintenance of system stability. It should also be remembered that a natural forest was not formed or "designed" for our need (productivity), and it is questionable how widely applicable are the principles governing the functioning of a natural forest. We can ask, "If the natural forest is managed according to natural principles, can our increasing demand for timber still be satisfied?" Over-exploitation of natural forest, leading to breakdown of its stability, is deplorable but it is a reality, and the problem cannot be solved simply by telling people not to disturb the natural forest. Undeniably, artificial forests or monocultures carry an element of risk. But it is unreasonable to compare the disease level of a plantation with the stability of a theoretical natural forest, and conclude that monoculture is more hazardous — even worse, that it is doomed to failure. Clearly, if a value judgement is to be based on a comparison between one form of forest practice and another,

then productivity as well as disease risk must be taken into account. Then, a natural forest for production is perhaps no less risky than a man-made forest. The total global area of plantation forest stood at 80 million ha in 1968, and it was estimated that this would double by 1985 (A. I. Fraser, 1975, unpubl. FAO Working Paper). Most of this is presumably monoculture. Although it does not guarantee the success of monoculture, it is hardly reasonable to take this as indication of failure. No doubt we should continue to try to understand and elucidate the principles governing the stability of natural ecosystems, but we should realise that the principles governing the stability of man-made forest are also worth enquiring into.

The author heartily agrees with Peace's (1957) conclusion to his paper, and it is worth quoting here:

... broad assumptions at the present stage can lead to false simplifications. We know regrettably little about health and disease in trees. We should therefore beware of cramping further advances by an attempt to base them on partially false premises.

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