Eur. J. For. Path. 21 (1991) 71–81

ISSN 0300-1237

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Perspectives of disease threat in large-scale Pinus radiata monoculture – the New Zealand experience

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Abstract

Large scale *P. radiata* monoculture has been in existence in New Zealand for over 60 years. During this time a number of damaging diseases (e. g. *Sirex-Amylostereum, Dothistroma, Armillaria, Cyclaneusma* needle cast. *Sphaeropsis* die-back etc.) have occurred but the impact was by no means devastating as these diseases are either controllable or in some the losses were deemed acceptable. As a large proportion of the biomass produced before age 10–12 is waste-thinned, the acceptable level of disease loss is thereby very high. The practice of clear felling and short rotation reduces some of the disease risks common in other forest systems. Outlook for future disease threat is discussed with respect to current trends toward clonal forestry and the possible arrival of additional exotic pathogens.

1 Evolution of Pinus radiata monoculture

A unique feature of New Zealand forestry today is the separation in functions of two forests – the native forest, and exotic plantations. Native forest, which covers about 24 % of the land area (6.2 million ha), plays an insignificant role in wood production, its main functions being soil and water protection and recreation. The forest is administered by the Department of Conservation. Ninety four percent of our wood production comes from 1.1 million ha of exotic plantations (3 % of the land area) which is dominated by a single species, *Pinus radiata* D. Don (ca. 950000 ha). Most of these exotic forestry plantations belong to several big private companies and a state-owned enterprise, the Forestry Corporation. A separate government department, the Ministry of Forestry coordinates and regulates the functions of this plantation forestry.

The evolution of this began in 1913 when it was recognized that the native forest which supplied most of the wood required was rapidly shrinking in size and could be depleted by 1965. To ensure future wood supply, as well as conserving the native forest, the only option was to start large scale planting of fast growing exotic tree species (Bunn 1979; Sutton 1984). There were two reasons for choosing to establish exotics: first the slow growth of native timber species (Table 1), and second, the fragility of the native ecosystem.

Table 1. **Comparative productivity of Pinus radiata with common indigenous timber species**

Species	Estimated mean annual increment m^3/ha/yr
Pinus radiata	20–25
Agathis australis	6
Nothofagus spp.	6
Beilschmiedia tawa	1–2
Podocarpus cupressinum	1–2

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Amongst the many exotics introduced was *P. radiata* which was planted here as early as 1856 (Sutton 1984). Hence recognition of its potential as a valuable plantation species came from fairly extensive early experience. From the late 1920s until 1935 over 300 000 ha of exotics (mainly *P. radiata*) had been established mostly on scrub land in the central plateau of the North Island where the volcanic ash soils were unsuitable for agriculture. The next 25 years, from 1936 to 1960, a second planting boom increased total area of *P. radiata* plantations to nearly one million hectares today. *P. radiata* established in the first planting boom, often referred to as the "old crop", was untended, i. e., received virtually no thinning or pruning. These stands were densely planted at 2500 stems or more per hectare. Today all stands are thinned and pruned and later clearfelled at predetermined ages according to the tending schedule deemed suited to the site and the end products required (Bunn 1979; Sutton 1984).

Broadly, the management trends today are as follows:

1. use of progressively improved seed (selected for form, growth rate, wood quality, disease resistance etc. from New Zealands own stands),
2. achievement of clonal forestry by methods of micropropagation (tissue culture, conventional cuttings),
3. standardizing nursery practices for good planting stock,
4. planting at 750 to 1000 stems per ha, followed by 2–3 thinnings and 2-step pruning for a final crop of around 250 stems per ha,
5. spraying to control Dothistroma needle blight, fertilizer application and weed control when necessary,
6. clearfelling at age 30 and continuous cropping (some areas are into their 3rd rotation).

An adjunct to this main-stream forestry is the development of agroforestry, i. e., planting trees on pasture, combining sheep and dairy farming with wood production, and the adoption of grazing for weed control with the additional benefit of increasing soil fertility and hence tree growth.

2 The fear of disease risks

From the beginning of the first "planting boom" in the late 1920s forest managers in New Zealand have been haunted by a feeling of uncertainty or apprehension arising from the alleged disease and pest risks of forest monocultures (Chou 1981; Fenton 1978; Cowling 1978). For example, J. J. de Gryse (1955) cautioned us in an advisory visit: "... 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."

3 Disease hazards in retrospect

Looking into the past 60 years of large scale monoculture, what in fact has been the disease impact?

3.1 The quiet era

Up until the late 1940s, there were only sporadic incidences of disease having little serious impact. Research at this stage was very much a one man's effort, notably that of Birch. All-told, 6 fungi pathogenic to *P. radiata* was recorded (Birch 1937). Of these six, only *Armillaria* and *Sphaeropsis sapinea* (Fr.) Dyko & Sutton comb. nov. (= *Diplodia pinea*) have caused some trouble to justify more detailed studies in recent times. The others comprising *Botrytis*, *Pestalotia*, *Phomopsis pseudotsugae* Wilson (= *Phacidiopycnis pseudotsugae* (Wilson) Hahn; *Phomopsis strobi* Sydow), and *Lophodermium* have been of negligible significance to this day.

3.2 The first shock wave

This quiet period was abruptly broken by a *Sirex* epidemic in the late 1940s that apparently vindicated the widely voiced warnings of the risks involved with a monoculture. Between 1946 and 1950, *Sirex noctilio* F., a wood wasp native to Europe which had been introduced to New Zealand around 1900, suddenly began to devastate *P. radiata* stands in the age group 15 to 20 years. The wasp lays eggs in living trees and at the same time deposits mucus and a fungus, *Amylostereum* sp., into the wood. The combined effects of mucus and the fungus are first wilting and then death of the tree which assists the hatched larvae to tunnel through the fungus-infected wood and the adults to make exit holes when emerging. The mean mortality from this epidemic was estimated to be an alarming 30 %. However, the biological loss from the disease is not equivalent to the economic loss under our forest management systems. For example a reduction of an initial 1500 stems/ha to 300 stems at age 35 through mortality means a volume loss of around 450 m^3/ha over 35 years (an MAI equivalent of 13 m^3/ha/yr). However, this represented only about a third of the total increment. Over 800 m^3/ha (an MAI equivalent of 23 m^3/ha/yr) is still standing at age 35 (SUTTON 1984). Because *Sirex* mostly attacked suppressed and codominant trees, some considered the epidemic equivalent to no more than a light thinning of an overstocked stand, and viewed that the effect may even have been considered beneficial (GILMOUR 1966).

It has since become evident that the combined effects of three separate factors contributed to the outbreak of the *Sirex* epidemic (ZONDAG, Pers. comm.):

1. a build-up of *Sirex* population due to a forest fire in 1946 in an area which is close to the center of *P. radiata* plantations.
2. the severe drought of 1947–48 and
3. overcrowding of the untended "old crop".

Today *Sirex* is no longer a problem of much concern because of the change in management regimes and the extra control given by the use of biological control agents (NUTTALL 1980).

As a direct result of the *Sirex* epidemic a network of Forest Biology Observers (FBO), now known as Forest Health Officers (FHO), was subsequently established to undertake the task of surveying the forests for disease and pests and assisting with control and research work. Another spin-off of *Sirex* epidemic was the vast amount of good research on the host/insect/fungus relationship. This came mainly from Australia where the drought threat is more imminent (TALBOT 1977).

3.3 The Dothistroma era – active research and keen alertness

The sudden outbreak of *Dothistroma* needle blight in 1964 (GILMOUR 1967) marked the beginning of a "boom" period in forest pathology activities and research. One of the reasons for this focus on pathology was that the dothistroma epidemic coincided with the second wave of largescale *P. radiata* planting, hence there was an increased concern about the disease threat from an expanding industry. Also during this period several other forest diseases hitherto unknown to this country has suddenly occurred.

In 1959 *Phaeocryptopus gaeumannii* (Rhode) Petrak was suddenly found on Douglas fir, our second most important exotic timber species (around 50 000 ha) which was previously considered to be free of major pathogens (HOOD and KERWSHAW 1973, unpub. 1977).

This was soon followed by a severe outbreak of an unknown disease (terminal crook) in a *P. radiata* nursery in 1963 the causal agent of which was subsequently identified as *Colletotrichum acutatum* Simmds. F. Sp. *pinea*, a fungus hitherto unknown to be a pathogen of this host (GILMOUR 1965; DINGLEY and GILMOUR 1972).

Sphaeropsis sapinea (Fr.) Dyko & Sutton comb. nov. (= *Diplodia pinea*), though known for a long time in this country (BIRCH 1937) raised a lot of concern since the mid 1960s (CHOU 1976, 1987, 1988). Two poplar rusts *Melampsora medusae* and *M. larici-populina* arrived in Australia in 1972 after at least a hundred years of growing poplars there without serious disease threat (CHOU 1973; WALKER et al. 1974; WILKINSON and SPIERS 1976). The arrival of poplar rusts spurred efforts to select and breed for disease resistance by introducing breeding stocks from abroad. This had the unfortunate consequence of introducing another dangerous pathogen, *Marssonina brunnea* (Ell. + Ev.) Magn., the impact of which on poplars here is yet to be evaluated (SPIERS 1983).

These events coupled with the outbreak of several devasting forest diseases abroad (Dutch elm disease in England, *Phytophthora cinnamomi* in Australia, pine wood nematode in Japan, cypress canker in Italy) all added up to heighten the fear of disease threat and pointed to the need for more pathology work.

Increased research during the "Dothistroma era" has not only added much knowledge to this disease but also to several other diseases hitherto little understood in this country, notably *Armillaria* root rot and *Cyclaneusma* needlecast, *S. sapinea* shoot blight and crown wilt, and cypress canker. A brief account of the impact of some of these diseases on *P. radiata* plantations in New Zealand will now be given.

Dothistroma needle blight – *Dothistroma* was first noticed in New Zealand in 1962, but was not positively identified until 1964. The disease was probably introduced in the late 1950s, though how this occurred is not known (GILMOUR 1967; GILMOUR et al. 1973). Some general aspects of and early experience with this disease have been reviewed by GIBSON (1972, 1973). Early work on its control by copper fungicides carried out in East Africa was soon tested here, and results of these studies promptly put into practice. Large-scale aerial spraying started in 1966, and a Dothistroma Control Committee was organized to supervise and coordinate the spray operation. Details of technical procedures and specifications followed in New Zealand are given in a handbook by KERSHAW et al. (1988). Briefly, ground and aerial survey of the disease level are conducted 3 months before spraying commences in November (late spring) to determine the area to be sprayed. The threshold disease level recommended for spraying is when infection reaches 25 % of unsuppressed green crown based on findings that growth losses are detectable only when infection exceeds this level (GILMOUR et al. 1973; WHYTE 1976). Initially cuprous oxide was used but this has been replaced by the cheaper but equally effective copper oxychloride. With improvements in spray technology, the cost of spraying has been progressively reduced and is now 50 % of what it was in 1966. Improvements include a reduction of spray volume from 56 l/ha to 5 l/ha and a reduction of droplet size from 200 μ to 65 μ achieved by using improved atomizers like micronair (RAY 1988; VAN DER PAS et al. 1984b). Except in areas of very high rainfall, *P. radiata* develops maturity resistance to *Dothistroma* by the age of around 15 at which stage chemical protection is no longer required. Another factor that makes chemical control feasible is its durable effect – a single spray can provide protection for 2–3 years (GILMOUR et al. 1973). This coupled with year to year fluctuations in the level of infection caused by varying weather patterns means that only a small proportion of stands within the susceptible age group requires chemical protection in any particular year. Between 1966 and 1982 the proportion of susceptible stands requiring spraying varied from a maximum of 34 % to as low as 2 % (VAN DER PAS et al. 1984a). Nationwide statistics on the frequency of spraying during the 30 year rotation of a stand is not yet available. From the 20-year records of one large timber company, the New Zealand Forest Products Ltd in the Central North Island a mean of 5.45 and range of 2–9 sprays per rotation is indicated (DICK 1989, unpub.). The economic impact of *Dothistroma* can best be seen from the total cost of spraying over the period 1966 to 1988. In this period a total of 1.185 mill. ha of plantation had been sprayed, and 37478 tons of copper fungicides used. The current cost of spray, including flight, material, survey and supervision cost, according to one estimate (DICK 1989, unpubl.), is NZ$ 17.74/ha (around US$ 10.28 at current exchange rate of 1 NZ$ = 0.58 US$). It should be noted that the cost was about double some ten years ago. Added onto this is the cost of research which could easily be 20 scientist years and 20 technician years which plus other expenses could easily amount to 1–2 million NZ dollars. Although the effectiveness of spraying in reducing infection and volume loss has been amply demonstrated, the cost-effectiveness of spraying is still debatable (VAN DER PAS et al. 1984a; WOOLONS and HAYWARD 1984; WHYTE 1976; DICK 1989, unpub.). Analysis in one study showed that the cost of extra wood produced in a stand sprayed 3 times during a 30 year rotation ranges from NZ$ 18–25/m^3 (depending on variation in spray cost) compared with NZ$ 18–25/m^3 for fibre wood and $ 40/m^3 for sawlogs/peelers produced without spraying (VAN DER PAS et al. 1984a). Another study showed that volume loss in the final crop was little affected by a reduction in the overall disease level by spray control, therefore if the final crop is the major objective, the necessity of spray is open to question (WOOLONS and HAYWARD 1984). It was also estimated that the cost of four sprayings for a crop on a 26 year rotation is equivalent to about 1 % of the average wholesale price of the final product (SUTTON 1971). Assuming the total cost of production to be NZ$ 50000, 1 % of this for spray cost is NZ$ 500. Assuming profit to be $ 5000 on a 10 % profit margin, the cut in profit due to the spraying would amount to 10 %. Thus 1 % increase in production cost could mean a 10 % cut in profit (HORGAN, priv. comm.). Still forest managers are quite unwilling to discontinue spraying for the possible unknown risks that might result.

Wide variability in tree susceptibility to *Dothistroma* is apparent in the field. Work on selection and breeding for disease resistance showed high heritability estimate in field trials and the absence of genotype/environment interaction (WILCOX 1982; CARSON 1988). Seed with *Dothistroma* resistance is now available. A 12 % reduction in crown infection and 11 % gain in growth is expected (CARSON 1989).

Armillaria root-rot – *Armillaria* was not a problem when *P. radiata* was largely established on pumice country during the first planting boom. However, during the second planting boom a threat from *Armillaria* did emerge when plantations began to be established on sites freshly cleared of cutover indigenous forest (Podocarp/*Beilschmiedia tawa*). Two species of *Armillaria, A. novae-zelandia* (Stevenson) Boesewinkel and *A. limonea* (Stevenson) Boesewinkel are involved in the killing of young trees after planting (MACKENZIE and SHAW 1977). It was demonstrated that trees that survived infection could suffer from a "sublethal effect" resulting in significant volume less amounting to 144 m^3/ha or 29 % of potential yield on a 26 year old sawlog regime (SHAW and TOES 1977; SHAW and CULDERON 1977). However, subsequent study of the same stand over a longer period gave an estimate of only 6–13 % loss of the projected 571 m^3/ha yield (MACKENZIE 1987). A survey of 50000 ha of converted plantations showed overall mortality to be around 5 % (VAN DER PAS 1981). Clearing of cutover indigenous forest for exotic plantations is unlikely to continue in the near future because of objections from conservationists, hence there is less reason for concern about the threat from *Armillaria* on such sites. What is of more concern is the threat of *Armillaria* to second or third rotation plantations as these are rapidly increasing. An early survey showed mortality to be under 3 % (SHAW 1976), but later study showed mortality could be as high as 40 % on sites initially stocked with *P. nigra, P. contorta* and *P. ponderosa* which were poison-thinned (VAN DER PAS 1981). Intercompatibility study at three different sites showed 15–93 clones/ha of the two *Armillaria* species. This diversity is very high compared with North America or Europe and suggests that *Armillaria* infection centres in young stands of *P. radiata* are initiated by basidiospores (HOOD and SANDBERG 1987). The potential threat of *Armillaria* to second or third rotation therefore warrants attention.

Cyclaneusma needle cast – *Cyclaneusma minus* (Butin) DiCosmo, Peredo & Minter (= *Naemacyclus minor* Butin) was first recorded in New Zealand in 1959 as *N. niveus*. For many years this fungus was known to be associated with periodic abnormal casting of one year or older *P. radiata* needles, but its pathogenic role, epidemiology and impact have not been elucidated until recently (GADGIL 1984; VAN DER PAS et al. 1984b). In New Zealand, peak infection occurs in lage autumn/early winter, while peak needle cast occurs in spring and autumn. Although more needles are lost in the spring cast, spore-bearing apothecia are more abundant in the autumn. Current-season needles are first colonized by *Cyclaneusma*, and one to two months later by *Lophodermium* spp. Inoculation tests confirmed the pathogenicity of *C. minus* but not *Lophodermium*, suggesting the latter to be secondary (GADGIL 1984).

From 1983 to 1985 inclusive, a nationwide aerial survey for needle cast undertaken covering a 586700 ha of *P. radiata* plantations (sampling intensity = 12 ha per 100 ha) which ranged in latitude from 35°39′–46°05′ (longitude 170°06′–174°21′) and had an annual rainfall varying from 700–2600 mm. This survey showed the disease to be present throughout New Zealand, but the infection level varied considerably according to locality, tree age classes and year. Most of the infected stands fell into the age class 6 to 20, while young stands under 5 years of age had a very low level of infection. Infection level increased with tree age until about 20 and then began to drop. Stands over 25 years of age were rarely affected. The infection level varied from year to year in any particular locality, but remained relatively constant nationwide. Within a single stand, considerable variation in infection level from tree to tree was observed and these differences tended to be maintained from year to year. Observations of tagged trees, for example, over a six year period beginning at age nine, showed that trees with initial low levels of infection (mean 6% crown symptom) tended to stay low. Whereas trees with high levels of infection (mean 67% crown symptoms) remained high (VAN DER PAS et al. 1984c). Studies by stem analysis have shown volume increment to be linearly related to the percentage of crown infection (yellowing or defoliation of unsuppressed green crown) in the form of: $Y = 537 - 4.29\,X$ (Y = vol. increment in dm^3, X = % disease severity). For every 10% increase in disease severity there is approximately 8% volume loss (VAN DER PAS et al. 1984b). Although volume and revenue loss in a heavily infected stand can be considerable [e.g., the estimate for one stand investigated was up to 43% loss in revenue (VAN DER PAS et al. 1984b)], the estimated overall volume loss due to this disease nationwide is no more than 5% (BULMAN 1990).

Cyclaneusma needle cast cannot be economically controlled by spraying or tree injection (HOOD and VANNER 1984). Selection and resistance may be the only option (CARSON 1983).

4 Concluding remarks

There seem to be a number of factors in our *P. radiata* management system that reduce the impact of disease. With the prevailing silvicultural practice which "wastes" 75–85% of the biomass produced before age 10–12 by thinning, the level of acceptable disease loss is extremely high, hence the impact of disease is lessened.

Other advantages of the present system which assists in dampening disease impact may be a short rotation and clearfelling. The short rotation period of 25 to 30 years reduces risks to pathogens which attack old trees such as heart rot fungi which commonly occur in traditional forest practice. Clearfelling obviates many troubles associated with selective logging. The nutrients loss caused by harvesting *P. radiata* is many times lower than that which occurs with agricultural crops, thus soil deterioration does not seem to be a major problem (WILL and BALLARD 1976). The rapid degradation of the root and stem residue under New Zealand conditions may not be conducive to the build-up of some root pathogens (SHAW 1976). Surely the geographical isolation and remoteness of the country to the main centres of the world have helped to slow down the arrival of damaging pests and pathogens.

However conditions may change. With increasing use of improved planting stock, initial to final crop stocking ratios will decline as these stocks are much more expensive to produce and hence less affordable to "waste". The "old crop" was planted at 2500 or more stems/ha, the second crop using select seed (collected from plus trees) or open-pollinated orchard seed, about 1500 to 1200 stems/ha, and now there is the option of using controlled pollinated orchard seed, or clones produced by cuttings or tissue culture, thereby enabling the initial stocking to be reduced to 750 or lower. A kilogram of controlled pollinated seed costs NZ$ 1200 vs NZ$ 200 for orchard seed, and the cost of producing 100 cuttings is between NZ$ 100–140 vs NZ$ 50–60 for seedlings (SHELBOURNE 1988; GLEED et al. 1988). Thus the acceptable level of disease loss is likely to drop.

The trend towards clonal forestry is also a march towards genetic uniformity, which with agricultural crops could mean an increasing disease hazard. However, direct applicability of experience in agricultural pathology to that of the many forestry crops has been questioned (ZOBEL 1982; HEYBROEK et al. 1982). Pines are considered naturally highly heterozygous, and in a pine breeding programme there is a minimum number of seed parents that must be maintained in a seed orchard in order to prevent a deleterious inbreeding effect (CARSON 1988). Hence it seems that a certain level of genetic diversity would be maintained even in a single clone, and that breeding would never produce genetically homozygous offspring as has occurred with some agricultural crops. Preliminary testing of clonal vs. seedling resistance against existing diseases (i. e., *Dothistroma*, *Armillaria*, *Sphaeropsis sapinea* and *Cyclaneusma*) gave no indication of increased susceptibility in the clones to these diseases (CARSON 1988). In addition some theoretical analyses have led to the belief that planting of a few genotypes (7–30) represents very little increased risk as compared with a more diverse population (LIBBY 1982). These appraisals, however, should not be taken as proof or assurances, and the problem needs long term evaluation and carefully planned studies. How these improved planting materials will perform with newly introduced pathogens is not known.

New Zealand's geographical isolation and remoteness has not been totally effective in preventing pests and pathogens arriving despite strict quarantine measures. The difficult question has always been: which ones should be listed as dangerous pathogens? Naturally our attention has been focused on pathogens in the native *P. radiata* stands and plantations in California. These numbered 72 as listed by OFFORD (1964) with a few more additions in a recent study (OLD 1979). This contrasts with only 20 pathogenic fungi on *P. radiata* in New Zealand as listed by GILMOUR (1966). The number now is no greater than 30 representing only a smal share of these potential pathogens. Western gall rust caused by *Endocronartium harknessii* Hiratsuka is one high on the alert list (OLD et al. 1986). *Heterobasidion annosum* (Fries.) Brefeld should now also be listed as a dangerous pathogen. This fungus had baffled us for some time because although it was long thought to be indigenous to this part of the world, it has caused little damage to the exotic pines. It is now evident that the Australasian *Heteobasidion* should be considered either as a separate form (HOOD 1985), or a separate species, *H. araucariae* sp. nov. (BUCHANAN 1988). It may be short sighted to focus our attention only on pathogens attacking *P. radiata*. *Dothistroma*, for example, was considered a new introduction to California (COBB and MILLER 1968). It was not in OFFORD's (1964) list, nor could it be found in indigenous *P. radiata* stands in the early days of its spread in California (MUIR 1974; OLD 1979), and the geographical origin of this pathogen is still unknown (PODGER 1978). It is quite common that an insignificant pathogen on one host species in one region can be a disaster for another host in a different region – e. g. chestnut blight, white pine blister rust, pine wood nematode. The place of pathogen introduction may be expected to quicken with increased flow of international traffic. Hence it would be short-sighted to relax our alertness and reduce our research effort when apparently it is a time of "peace".

Acknowledgements

The author gratefully acknowledges helpful discussions with Dr. R. James, Mr. G. Horgan and Mr. Bulman on certain parts of the paper, L. Whitehouse in helping with editorial matters, and Dr. L. Shain in critically reading the manuscript.

Summary

Large scale planting of *P. radiata* began in New Zealand in the late 1920s because of a depleting wood resource from the indigenous forest. Many native tree species are too slow-growing for plantation use. Currently almost all wood for domestic use as well as export comes from 1.1 million ha of *P. radiata* (3 % of land area). In the late 1940s the *Sirex-Amylostereum* epidemic with drought and high stocking resulted in high tree mortality. Economic loss was, however, little, because the stands were overdue for thinning. *Dothistroma* needle blight epidemic began in mid 1960s. The disease has cost millions of dollars in aerial spraying with copper which successfully controls the disease. Although the economic benefit of spraying is debatable, the increase in cost of wood production is only 1 %. *Armillaria* became a problem on converted cut-over indigenous forest, causing mortality of young trees and volume loss due to sublethal infection. *Cyclaneusma* needlecast can also cause serious volume loss in some stands, but the overall impact is still not great. The current forest practice of waste thinning 75–85 % of the initial stocking before age 10–12 means a very high level of disease loss is acceptable provided the final crop trees are not seriously damaged. This can change due to increasing use of improved planting stock and unknown risks associated with increased genetic uniformity. There is also the threat from additional pathogens from different pine regions of the world.

Résumé

Les perspectives de la menace sanitaire dans la monoculture de Pinus radiata à grande échelle. L'expérience néo-zélandaise

La plantation de *P. radiata* à grande échelle a commencé en Nouvelle Zélande à la fin des années 1920, à cause du manque de ressource ligneuse à partir des forêts indigènes, dont beaucoup d'espèces ont une croissance trop lente pour être utilisées en plantation. Actuellement l'essentiel de la production ligneuse pour l'usage national et l'exportation est fourni par 1,1 million d'hectares de *P. radiata* (3 % du territoire) alors que la forêt naturelle (24 % du territoire) a une fonction de protection de l'environnement. A la fin des années 1940, l'épidémie de *Sirex-Amylostereum* due à la sécheresse et à la surdensité a provoqué une forte mortalité. La perte économique fut cependant faible car les peuplements demandaient à être éclaircis. L'épidémie de *Dothistroma* commença au milieu des années 1960. Elle coûta des millions de dollars en traitements cupriques aériens qui maîtrisent efficacement la maladie. Bien que le bénéfice économique de ces traitements reste discutable, l'augmentation du coût de production du bois n'est que de 1 %. L'Armillaire devint un problème dans les forêts naturelles reconverties après coupe, en provoquant la mort des jeunes arbres et des pertes de volume suite aux infections sub-létales. *Cyclaneusma* peut aussi entraîner de sérieuses baisses de volume dans certains peuplements, mais l'impact général demeure faible. La pratique actuelle qui élimine en éclaircie 75–85 % des arbres avant l'âge de 10–12 ans, fait qu'un haut niveau de pertes par maladies est acceptable à condition que le peuplement final ne soit pas sérieusement endommagé. Ceci peut changer par le recours croissant à du matériel sélectionné plus coûteux, entraînant des risques accrus et inconnus associés à une plus grande uniformité génétique. Il existe aussi la menace des pathogènes des pins existant dans différentes régions du monde.

Zusammenfassung

Über die Bedrohung von großflächigen Pinus radiata-Monokulturen durch Krankheiten. Erfahrungen aus Neuseeland

Der großflächige Anbau von *Pinus radiata* begann in Neuseeland in den späten 20er Jahren dieses Jahrhunderts als Folge der Übernutzung der Holzreserven der natürlichen Wälder. Viele einheimische Baumarten wachsen zu langsam, als daß sie in Plantagen genutzt werden könnten. Derzeit stammt fast alles Holz für den eigenen Bedarf und für den Export von 1,1 Mio ha Anbaufläche mit *P. radiata*. In den späten 40er Jahren führte eine *Sirex-Amylostereum*-Epidemie zusammen mit Trockenheit und zu dichten Beständen zu hohen Ausfällen. Allerdings war der wirtschaftliche Verlust gering, da die Bestände längst zur Durchforstung anstanden. Eine *Dothistroma*-Epidemie begann in der Mitte der 60er Jahre. Die Krankheit verursachte Kosten in Millionenhöhe ($)

für Bekämpfungsmaßnahmen aus der Luft mit Kupferpräparaten, mit denen die Krankheit erfolgreich bekämpft werden kann. Obwohl die wirtschaftlichen Vorteile solcher Maßnahmen umstritten sind, beträgt die Zunahme der Kosten für die Holzproduktion nur 1 %. *Armillaria* wurde zum Problem in umgewandelten natürlichen Wäldern und führte zum Absterben junger Bäume und zu Zuwachsverlusten durch subletale Infektionen. Die *Cyclaneusma*-Nadelschütte kann in einigen Beständen ebenfalls schwere Zuwachsverluste bewirken, insgesamt ist die Bedeutung dieser Krankheit aber noch nicht groß. Die gegenwärtige Praxis, vor dem Alter 10–12 bei der Durchforstung 75–85 % der Ausgangsbestockung zu entnehmen, geht davon aus, daß auch sehr große Ausfälle durch Krankheiten tragbar sind, vorausgesetzt, die für die Endnutzung verbleibenden Bäume sind nicht zu sehr geschädigt. Dies kann sich jedoch durch den zunehmenden Anteil züchterisch bearbeiteten Materials und die unbekannten Risiken im Zusammenhang mit genetischer Uniformität ändern. Außerdem besteht eine Bedrohung durch zusätzliche Pathogene aus den verschiedenen Kieferngebieten der Welt.

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Receipt of ms.: 2.3.1990