THE RISK OF PITCH CANKER ESTABLISHMENT IN NEW ZEALAND.

by

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Forestry is one of New Zealand's most important export industries and accounts for approximate 7% of New Zealand's land use[1]. Exotic softwoods represent 97% of the plantation forestry industry, of which *Pinus radiata* is the primary timber species, accounting for 92% of all softwoods planted[1]. The next most predominant softwood species is *Pseudotsuga menziesii*, which comprises approximately 6% of planted areas[1]. The introduction of pitch canker could have devastating effects on the pine industry through mortality, growth suppression and stem deformation of the softwood species planted, specifically *P. radiata*. As a result, *F. circinatum*, is considered the most undesirable and unwanted exotic forest pest. In this review, the risk and potential of establishment of pitch canker in New Zealand is discussed along with the possible impact and preventative methods that have or could be employed.

Host species and genetic resistance:

Genetic resistance to pitch canker has been demonstrated in all pine species tested. However, the degree of resistance is variable. The predominant softwood species used in the forestry industry in New Zealand, *P. radiata* and *Pseudotsuga menziesii,* are both susceptible to *F. circinatum*[2-4]. Specifically, *P. radiata* is considered one of the most susceptible species and it has been predicted that only 0.3-2.1% of the stock used in New Zealand is resistant to the disease[2]. However, genetic heritability has been reported and thus, useful genetic gains would be expected from selection. Furthermore, there appears to be an age related factor, as resistance does not appear to be operative in *P. radiata* seedlings, suggesting that the low level of resistance from greenhouse trials may not be applicable for more mature plants[5]. *Pseudotsuga menziesii* is considered moderately resistant to the disease, although little is know about the epidemiology or the effects of pitch canker on this tree species. The other pine species grown in New Zealand, *P. attenuata, P. contorta, P. elliottii, P. muricata, P. patula, P. ponderosa, P. radiata x attenuata, P. strobes and P. taeda*[1,6], are also all susceptible to pitch canker[2].

In addition to genetic resistance, induced resistance responses have also been demonstrated in the native populations of *P. radiata*, in California. Repeated inoculation of *P. radiata* trees has been found to result in a reduction in disease incidence and severity[7]; the long-term effects and

mechanisms behind this resistance are currently being investigated. It is unknown whether induced resistance mechanisms, such as observed in California, would also occur in New Zealand. Initially, it was predicted that California would lose at least 85% of its *P. radiata* population (based on 15% resistance) as the disease spread through urban and native settings causing widespread mortality[8]. More recently, the level of mortality and severity of infections has reduced, and pitch canker appears to be becoming a balanced part of the *P. radiata* ecosystem, potentially replacing fire as an agent to open and regenerate stands. In urban settings, the disease levels in the remaining *P. radiata* appear to be lower; diseased *P. radiata* trees are removed if they pose a safety risk and are generally replaced with other tree species. The current incidence of pitch canker in Christmas tree plantations is unknown, as is the long-term effect and economic impact of pitch canker on this industry.

P. radiata accounts for 92% of all softwoods planted in New Zealand and with the low genetic resistance of the New Zealand-bred *P. radiata* it would be expected that 98% of *P. radiata* seedlings could be infected by *F. circinatum*, should the disease be introduced. The number of mature *P. radiata* trees that would become infected is unknown but would be expected to be much lower than what has been predicted for seedlings. The percentage that would die from the disease or secondary factors is also unknown. Breeding for resistance in *P. radiata* could decrease the number of at-risk hosts and could be used to minimise the effects of this disease but it is unknown whether such resistance would also be linked with other undesirable traits. Genes involved in resistance/susceptibility are currently being elucidated at the University of Florida and could provide information for the genetic improvement of resistance levels[9]. Likewise, further understanding of the epidemiology of the disease, such as the influence of resin or the localised response to infection, could be used to select for desirable traits. Additionally, *P. radiata* could potentially be hybridised with a more resistant pine species such as *P. oocarpa*[10], although care would be needed to ensure that any hybridisation would not result in increased host range of other pathogens of the host tree species' through hybridisation that could be more problematic than the risk of pitch canker itself. It is unknown whether the induced resistance responses would be functional in New Zealand, if so, the severity, and potentially the incidence, of the disease would be expected to be lower.

Risk of entry:

F. circinatum can be disseminated vertically, through infected seed, or horizontally by spores, which can be vectored by a variety of agents such as wind, rain, animals, insects or soil. It is possible that pitch canker could be introduced to New Zealand through infested seed or soil, or

on insects, and in addition, in infected live or dead plant material, such as occurred with the incursion on infected *Pseudotsuga menziesii* scions imported from the USA in 2003[11].

The risk of pitch canker being introduced to New Zealand through contaminated seed was identified early and as a result increased restrictions were placed on seed imported from countries known to have pitch canker, and additionally, seed must also be screened by Forest Research for the presence of *F. circinatum*[6]. Despite these procedures, it is still possible that contaminated seed could escape detection and potentially become established in nursery stock, such as what is believe to have occurred in both South Africa and Chile, where the disease is present in the nurseries but not in the plantations. It is also possible that the pathogen could be introduced on live plant material. However, with the quarantine regulations enforced for the introduction of live plant material, the probably of this occurring would be low. Likewise, timber and wood products also pose a risk for the introduction of *F. circinatum*, as studies have found that the pathogen can survive in wood chips and branches for at least one year[12]. Heating wood products at 50^{o}C for at least ten days can kill the pathogen but can be a costly process[12]. No sawn timber of *P. radiata* is imported from the USA but there is a possibility that the fungus could inadvertently be introduced on crates or pallets made from contaminated material.

Besides infected plant materials, the likelihood of agents that can vector the pathogen into New Zealand is high. For instance, insects, which are known to vector the fungus (such as species of *Ernobius, Ips, Pissodes* and *Pithyophthorus*), are frequently intercepted at New Zealand ports and soil, which the fungus can survive in for potentially up to 3 years, is also a high risk factor. Soil, plant debris and insects can be introduced on a variety of items such as tents and camping equipment, shoes, imported second-hand vehicles and logging equipment. Quarantine inspections at airports and other ports of entry can minimise the risk of entry through these means. However, the possibility that the pathogen will escape detection or infected material will be illegally imported always increases the risk of introduction.

Wounding agents, vectors and climate:

If *F. circinatum* should be introduced into New Zealand the potential for the pitch canker disease to become established has been predicted to be high, based on the similarity of New Zealand's climate conditions to those in California and the rapid spread of the disease through urban and native stands of *P. radiata.* However, New Zealand's climate is also similar to Chile's, yet the pathogen, which is known to be present in both South African and Chilean nurseries for several years, has not spread to the plantations (*P. radiata* and other pine species) despite the abundance

of spores present. The reason for lack of establishment is unclear. Studies and observations of the disease have suggested that a complex variety of factors are responsible for successful infections to occur. These include, but are not necessarily limited to, wounding agents, vectors and climate.

In the southeast USA and California, insects have been shown to be wounding agents and, in some cases, vectors of the pathogen. Wounds caused by natural injuries or weather have also been found to become infected in the southeast USA and wounds of this type constitute a large number of the total infections observed[13,14]. In contrast, the frequency of infection of weather- or injury-related wounds in California is significantly lower and is not considered an important part of the disease cycle. Infections have also been found to occur from cattle hoof damage in the southeastern USA, however, no infections have been observed from bird claw marks[15]. In South Africa and Chile, natural-or weather-related injuries are evident in some stands and some pine-associated insects, known to be involved in pitch canker in the USA, are present but despite this, the disease has not become established. Thus, although wounding is an important factor (i.e. no wound = no infection), it is apparent that not all types of wounds result in pitch canker and there are also likely to be differences based on location suggesting a strong environmental influence.

The type of wound created and wound/climate conditions may be more important for successful infection to occur. For instance, there is evidence to suggest that wounds that result in high resin exudation may effectively seal the wound, preventing infection and that wounds that are exposed to moisture, such as from plant moisture (deeper wounds) or atmospheric conditions, are more likely to result in successful infection[12,15]. These factors could help explain the differences in infections from different feeding insects and also the variation in infection of injury-related wounds in the southeast USA versus California. For example, the high humidity and temperature in the southeast USA may be more conducive of infection. Although coastal California is frequently subjected to a belt of fog, it is possible that the lower temperature in these regions suppresses pathogen growth, as temperatures of 10^{o}C in the laboratory have been found to prevent growth of the pathogen[16], and when the temperature has risen, the fog belt may have effectively been "burned off". For insects, infections may only occur from the creation of wounds deep enough to provide adequate moisture, wounds created by insects that also vector the pathogen (i.e. spores present at wounds before wound can dry out), or wounds that do not result in substantial resin production. The effects of spore load on infection is unclear, in some studies a direct correlation between the number of spores and disease severity has been demonstrated[17,18], whereas, in other studies no difference in disease levels has been reported with

varying spore loads[2,19]. It is also likely that native host-insect associations are extremely important. For instance, the incidence of infections in exotic pines, susceptible to pitch canker, planted in California have been substantially lower[19]. This variation has been attributed to a lack of visitation by the native, species-specific insect populations present. Thus, for South Africa and Chile the low frequency of suitable insect wounding agents/vectors and variation in climate conditions, potentially not conducive to infection, may have significantly lowered the likelihood of infections in these regions.

Essentially these finding provide good news for New Zealand. Although 150 species of insects have been recorded on *P. radiata* in New Zealand, those insects closely associated with pitch canker in the USA, such as *Ips* spp., *Ernobius punctulatus, Pissodes nemorensis*, are not present. It is possible that many of these insects in New Zealand may be able to vector the disease but as the majority would not feed or create suitable wounds, the likelihood of disease establishment would be low unless favourable wound conditions were encountered. In addition to insects, other potential wounding agents/vectors in New Zealand that must be considered are possums, birds and livestock. Possums are known to cause substantial damage to young plantations of *P. radiata*, including damage to the terminal shoots and lateral branches through browsing[20]. In general, possums have little effect on trees over 14 years old[20]. Likewise, livestock run through plantations could create wounds on the roots that potential could become infected. Birds would unlikely to have an effect on the establishment on pitch canker infections unless they bent branches or caused considerable damage with their beaks or claws to the branches. It is unknown if possum fir and/or bird feathers could vector the pathogen. For all three groups of animals, the likelihood of successful infection would probably not only rely on the wound created but also amenable climate conditions, to facilitate infection by the pathogen. Whether New Zealand's climate would aid in the establishment and spread of pitch canker, should *F. circinatum* be introduced, is unknown. Further understanding on the effects of moisture and temperature is required to determine this. Nevertheless, based on observations from southeast USA and California, it would appear that the moisture levels would be sufficient but the low temperatures may hinder pathogen growth, thus, minimising the risk of weather- or injury-related wounds becoming infected. However, it would be expected that infections would be more likely to occur in Northland regions, exposed to tropical weather conditions, and coastal regions, than in colder inland areas.

Silvicultural practices:

Silvicultural practices such as fertilisation, stand density and irrigation have been found to influence the incidence and severity of pitch canker. Fertilisation or increased levels of nitrogen (N) in pine stands can result in intensification of the disease[21,22]. Although a direct correlation between N and pitch canker has been demonstrated[21], other studies have found that fertilisers containing potassium (plus nutrients), but lacking in nitrogen, also increase disease severity[22]. In contrast to fertilisation, thinning has been found to reduce the effects of pitch canker[22]. It is postulated that the reduction in disease from thinning alleviates moisture stress, another factor known to increase the incidence and severity of pitch canker and, as would be expected, irrigation can reduce the intensity of pitch canker infections[15]. Interestingly, fertilisation combined with thinning still resulted in high disease levels, equivalent to those found with fertilisation alone, indicating the importance of nutrient levels for this disease[22].

Stand density and irrigation are unlikely to be influential in the establishment of pitch canker in New Zealand as *P. radiata* plantations are generally not over stocked, approximately one quarter undergo production thinning, and stands are rarely exposed to drought conditions. However, the majority are subjected to fertilisation treatments or are planted in areas with high nutrients levels, which is of major concern. Studies on the effects of N from chicken houses on the levels of pitch canker in *P. elliottii* plantations in Florida found that areas with extremely high foliar N levels (1.8%-2.2%) had greater than 87% mortality[21]. Background levels of foliar N for Florida are approximately 0.9%, fertilisation levels are generally 1.2% and intensive fertilisation reaches levels of 1.5% foliar N[23]. In New Zealand, foliar N levels less than 1.2% are considered deficient, levels between 1.2%-1.5%, slight deficient, and levels greater than 1.5% are considered sufficient. High levels of potassium have also been found to increase disease severity and other nutrients are known to have additive effects. Thus, fertilisation or use of sites with high nutrient levels is likely to be problematic if *F. circinatum* should ever be introduced.

Impact and control:

Losses from pitch canker can result from reduced lumber quality, reduced growth and tree mortality. This could have significant effects on the $5 billion New Zealand timber industry. The economic impact has been predicted in other reports. However, control of the disease could help minimise disease impacts. Control methods to prevent establishment of the disease have been investigated. Treatment of pruning wounds with thiabendazole in paint can prevent infections from occurring[24] but would be unlikely to be a feasible method for large-scale prevention. Removal of infected material can also help alleviate disease levels by reducing inoculum levels.

Likewise, in California, the application of the insecticide carbofuran was effective at reducing the incidence of insect damage, thus decreasing the number of pitch canker infections[25]. However, insecticides would only be effective in New Zealand if infections were insect-related. A few studies have found chemical (thiabendazole) and biological (*Arthrobacter* spp.) controls that are effective at reducing pathogen damage in the greenhouse[25-27]. Whether these controls would provide significant differences or could be feasibly applied to plantations is unknown. Several chemicals have been found to reduce external contamination of infected seed[28,29], however, no methods have been effective at eliminating *F. circinatum* from internal-borne infections. In view of this, the current restrictions on the importation of pine seed should be retained.

Genetic diversity and pathogen virulence:

The genetic diversity of *F. circinatum* has been investigated using molecular markers and vegetative compatibility groups (VCGs). Results of genetic diversity from the molecular marker studies have shown that the Californian and Japanese populations of *F. circinatum* are mostly likely to have derived from the populations present in the southeastern USA[30]. These three populations are genetically, significantly different from the Mexican and South African populations of *F. circinatum*, which were found to have a limited overlap indicating that the South African population may have originally come from Mexico[30]. These results, along with the high levels of resistance in most pine species from Florida and Mexico, would suggest that both regions are centres of origin for *F. circinatum*, instead of Mexico alone as originally was thought. As the sample size used from Mexico was small, further sampling of the Mexican population would be required to determine the degree of genetic overlap, if any, between the Florida and Mexican populations of *F. circinatum*. The genetic diversity of *F. circinatum* has also been measured using VCGs. All populations were found to have unique VCGs, with the exception of C7 which is shared between the Californian and Japanese populations, and for some of the populations worldwide the high number of VCGs present indicates that regular outcrossing is probably occurring[30-33]. However, the study of diversity with molecular markers have shown that VCGs are a more sensitive indicator of genetic diversity in *F. circinatum* and would be a more informative method of measuring the level of diversity within a population rather than among countries[30].

The genetic diversity of *F. circinatum* is only of immediate importance to the New Zealand forestry industry if the diversity is correlated with pathogen virulence or if some populations worldwide only contain one of the two mating types. At this stage, no direct correlation between

genetic diversity and virulence has been detected. Studies have shown that the level of virulence does not significantly differ between the populations of *F. circinatum* worldwide[8,13,34]. However, in the Californian population, there are two distinct virulence groups[8,35]. Thus, although variation in virulence among strains does exist within populations this has not appear to have been extrapolated between countries. Both mating types are known to be present among the populations of *F. circinatum* worldwide[36-38]. However, mating has never been observed in the field, although crosses have been successfully obtained in the laboratory[38] and analysis of VCGs and molecular marker results would suggest that mating is occurring, at least in some populations. Analysis of genetic diversity, virulence and mating has been restricted to populations of *F. circinatum* from the USA, Mexico, South Africa and Japan. In contrast, nothing is known about the Chilean and Spanish populations, although it is suspect that they were introduced through contaminated seed stock from Mexico.

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