PITCH CANKER – RISK OF ESTABLISHMENT IN NEW ZEALAND BASED ON A GLOBAL PERSPECTIVE.

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Client Report No. 40217

PITCH CANKER – RISK OF ESTABLISHMENT IN NEW ZEALAND BASED ON A GLOBAL PERSPECTIVE.

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Date: July 2006
Client: Forest Biosecurity Research Council
Contract No: 40217

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EXECUTIVE SUMMARY

Objectives

- To review and evaluate the current global situation of the pine disease, pitch canker, in a variety of countries worldwide in both exotic and native pine stands.

- To predict how pitch canker could behave, spread and be controlled, if the fungus *Fusarium circinatum* were introduced into New Zealand, based on the establishment of this disease worldwide.

Key Results

Based on the environmental conditions and epidemiology of pitch canker in a variety of countries worldwide, the following summarises the risk status for establishment of this disease in New Zealand:

- Forests have wounds, wounding agents and vectors suitable for infection.
- In the absence of intricate insect-host systems (such as in California), wounding agents present are unlikely to play a significant role in disease establishment.
- Temperature and humidity levels would be sufficient in most areas for establishment of the disease.
- Pitch canker would be expected to be more severe in regions that are coastal or frequently are covered in fog.
- Colder or dryer regions would be expected to have a low incidence and severity of disease.
- Unlikely that moisture stress will be a problem as stands have low stock density and soil moisture levels are usually high.
- New Zealand has high nutrient levels but it is currently unknown whether the levels would facilitate disease establishment or severity.
- Additional fertilisation is not recommended.
- Swift and stringent eradication procedures could easily prevent the spread of pitch canker to adjacent forestlands or nurseries, if *Fusarium circinatum* were to be introduced.

The behaviour of pitch canker varies considerably between countries, depending on the host tree species, climate, wound agents, host resistance and silvicultural practices. Prediction of how *F. circinatum* would function if it were introduced into New Zealand is difficult as disease establishment and severity requires a combination of many environmental factors.

Application of Results

Findings and recommendations are applicable for revision of pitch canker risk assessments and current eradication/management procedures.

Further Work

Monitoring of the impact of this disease should be continued in countries where pitch canker is established and in countries where recent introductions of *F. circinatum* have been detected.

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Information for Ensis abstracting:

Contract number	
Client Report No.	
Products investigated	
Wood species worked on	*Pinus* species
Other materials used	*Fusarium circinatum*
Location	Chile, New Zealand, South Africa, Spain, USA

INTRODUCTION

Fusarium circinatum is the casual agent of the pine disease known as pitch canker. Pitch canker infections are characterised by the exudation of copious amounts of resin at the site of infection and can result in mortality of the tree, but most commonly suppress growth. The disease is present in a variety of locations globally.

The establishment of pitch canker in New Zealand could have devastating effects on the pine industry through mortality, growth suppression and stem deformation of the softwood species planted, specifically *Pinus radiata*. As a result, *F. circinatum*, is considered New Zealand's most undesirable and unwanted exotic forest pest.

This report reviews the epidemiology and current situation of pitch canker worldwide and discusses the risk and potential of establishment of pitch canker in New Zealand, along with the possible impact and preventative methods that have or could be employed.

RESULTS AND DISCUSSION

Worldwide distribution of pitch canker

Pitch canker was first observed in 1945 on *P. virginiana* in North Carolina, USA[1]. Since its original sighting, the disease is now known to be throughout the southeastern USA occurring from Florida to as far north as Virginia, and westward to Texas[2,3]. Early reports also identified the pathogen in native *P. occidentalis* in Haiti [4,5]. In 1986, the disease was first observed in California in Santa Cruz[6]. Although pitch canker now occurs in the three mainland native *P. radiata* stands, on costal planted *P. radiata* and in many Christmas tree plantations throughout California[7,8], it has not spread northward up to Canada, as was expected. Unlike the disease distribution in the southeast, pitch canker in California appears constrained to near-coastal regions[7], with the exception of one site in the Sierra Nevada[9].

Since pitch canker was first observed in the USA, it has also been found to occur on pine trees in a variety of locations worldwide (Figure 1). Other countries known to have *F. circinatum* include: Chile[10], Japan[11,12], Mexico[13-15], South Africa[16,17], and Spain[18,19] It has also been speculated that the pathogen occurs in several other countries, such as Italy, Iraq and South Korea, but these are considered unconfirmed in the absence of supporting or unambiguous reports. The centre of origin for *F. circinatum* is currently unknown. Originally, the pathogen was thought to have been introduced to the USA from Haiti, where it was considered abundant and endemic[4]. However, more recently Mexico has been considered another potential centre of origin based on the wide distribution but low disease levels; this has also been supported by results from molecular analyses[14,20]. The pathogen has been identified on 19 pine species in 13 states across central Mexico[15].

F. circinatum was initially identified in nurseries in Spain[18] and since then has also been reported in plantations[19]. Pitch canker has been found in plantations of *Pinus radiata* and in nurseries on *P. radiata* and *P. pinaster*. However, other susceptible species present in the nurseries, *P. nigra*, *P. sylvestris*, and *Pseudotsuga menziesii,* did not show disease symptoms. To date, there has been no evidence of the disease or the fungus in native or other pine species. Surveys of the distribution throughout Spain are not available. However, it is suspected that the disease is present across the entire northern part of Spain, with a southern limit to its spread. It is also likely that the fungus is present in northern Portugal and southern France.

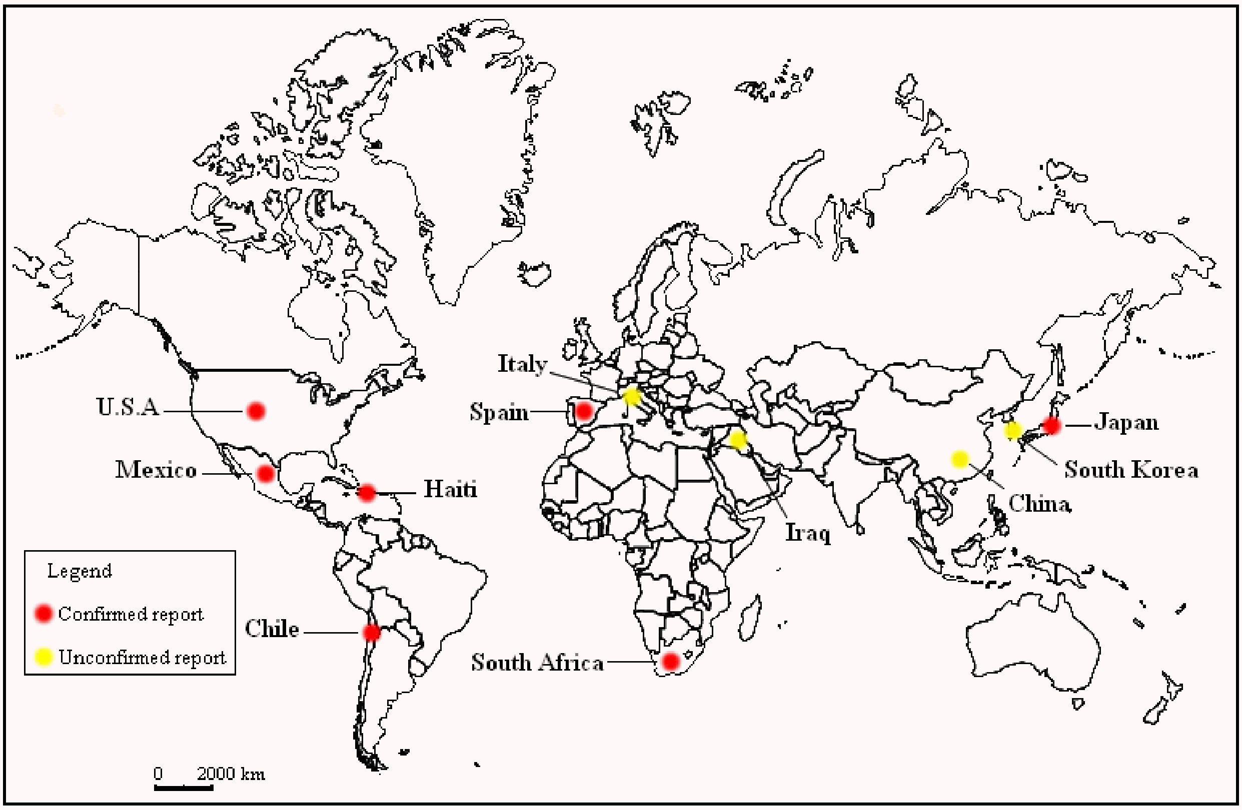

Figure 1. Worldwide distribution of pitch canker.

In Japan, pitch canker was first discovered in 1987 in native *P. luchensis* on Amamiooshima and the Okinawa Islands[11]. The disease causes branch and shoot dieback and resinous stem cankers but is not considered to be problematic[21]. *F. circinatum* has also been found in containerised stock and nurseries in Chile[10] and South Africa[16], and is believed to have been introduced from contaminated seed stock[22,23]. Although the fungus is considered to be well established in both countries, the pathogen has not yet been discovered in the field[10,24]. It is unknown why the disease has not spread to plantations, especially so when the disease is believed to have been present since the early 1990's and occurs in pine growing areas[16,22].

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*[8,9,25]. 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[25]. 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[26]. *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[27,28]*,* are also all susceptible to pitch canker[25].

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[29]; 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[7]. 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* from greenhouse trials it would be expected that 98% of *P. radiata* seedlings could be infected by *F. circinatum*, should the disease be introduced. However, 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[30]. 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*[31]*,* 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[32].

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*[27]. 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[33]. Heating wood products at 50°C for at least ten days can kill the pathogen but can be a costly process[33]. 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

F. circinatum can be disseminated vertically, through infected seed, or horizontally by spores that can be vectored by a variety of different agents, such as wind, rain, animals, insects or soil. For horizontal transmission, successful infection only occurs when vectoring of the spores is coupled with wounds or openings on the trees. In general, insects, weather or mechanical damage can cause wounds.

In California, vertical infection is very common with over 80% of the seed infested with *F. circinatum* due to infections by cone-related insects that can allow the fungus to spread to and infect nearby seeds[34]. Likewise, seedborne infections of pine seed in the southeast USA are common, although, the frequency varies between tree species and is probably related to specific insect relationships with the host. In contrast, very little evidence of seedborne infections has been detected in other countries with pitch canker, probably because of the lack of host specific cone-related insects in these regions. This limitation in vertical transmission of *F. circinatum* is a critical factor in restricting the spread of the pathogen to nurseries, and subsequently, back to the plantations.

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[3,35] and Spain and wounds of this type constitute a large number of the total infections observed. 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[36]. 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[33,36]. 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[37], 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[38,39], whereas, in other studies no difference in disease levels has been reported with varying spore loads[25,40]. 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[40]. 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[41]. In general, possums have little effect on trees over 14 years old[41]. 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 the colder or dry inland areas of New Zealand.

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[42,43]. Although a direct correlation between N and pitch canker has been demonstrated[42], other studies have found that fertilisers containing potassium (plus nutrients), but lacking in nitrogen, also increase disease severity[43]. In contrast to fertilisation, thinning has been found to reduce the effects of pitch canker[43]. 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[36]. 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[43].

Water stress, generally caused from overstocking of stands, has been implicated in California where the suppression of fire has resulted in overstocking of native *P. radiata.* In addition, water stress from a four-year drought has been attributed to intensifying the disease over this time period, when the worst levels of disease were recorded. In other countries, observations of disease establishment, and consequently, severe levels of pitch canker in overstocked plantations at about the time of crown closure also suggests that water stress is an important factor in the progression of this disease. In some regions it is unusual to find young plantations or nurseries (open-root or container) with the disease, despite adequate local inoculum. In many cases, the young, disease-free plantations can be directly adjacent to, or surrounded by, heavily infected mature stands. It is unlikely that the lack of establishment of pitch canker in these plantations is due age, as multiple studies have shown that pitch canker is not an age-related disease. Instead, it is hypothesised that water stress, correlated from overstocking of stands, is directly related to both the establishment and severity of the disease in the stands as they mature.

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[42]. 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[44]. 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.

Chemical and Biological control

A variety of control methods have been investigated for preventing and/or reducing the effects of pitch canker in pines planted in urban settings, plantations or native stands. Once the disease is established, the most common form of control has been to prune initial branch infections when first detected and to remove heavily infected or dying trees[7,8,45,46]. Pruning of infected branches is unlikely to completely control the disease, as mortality or severe disease levels are usually a result of multiple infections causing extensive crown dieback rather than one canker that girdles the trees, but can help delay the development of pitch canker[8,47]. However, it is possible that pruning could create wounds suitable for infection by *F. circinatum*, unless the wounds are treated to prevent infections from occurring. Treatment of pruning wounds with thiabendazole in paint can prevent infections from occurring[45] but would be unlikely to be a feasible method for large-scale prevention.

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[48]. 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[48-50]. 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[34,51], 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.

In South Africa and Chile, where pitch canker is only problematic in the nurseries, sanitation procedures of soil, containers and infected material are used to help control the disease. While these control methods have prevented the spread of *F. circinatum* to other nurseries and into the plantations, they have not been effective in eradicating the disease. With the current low levels of *F. circinatum* inoculum and the apparent absence of horizontal transmission, both in the nurseries and the plantations, it is believed that this disease could be completely or very nearly eradicated if more stringent procedures were enforced.

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[20]. 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[20]. 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. A preliminary study of the Chilean and Spanish populations indicates that isolates from Chile have most likely come from Mexico, whereas those from Spain are from the southeast USA. No genetic diversity has currently been detected among the Spanish isolates tested, suggesting a clonal population in this country. 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[20,52-54]. 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[20].

The genetic diversity of *F. circinatum* is of 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[7,24,35]. However, in the Californian population, there are two distinct virulence groups[7,55]. 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* California, southeastern USA and South Africa[56-58]. However, mating has never been observed in the field, although crosses have been successfully obtained in the laboratory[58] and analysis of VCGs and molecular marker results would suggest that mating is occurring, at least in some populations. Conversely, only one mating type has been detected in both the Chilean and Spanish populations and is likely to be important in minimising the genetic diversity, which could potentially prevent the development of more virulent strains in these countries.

SUMMARY

Pitch canker is a varied and complex disease of *Pinus* species. Despite the establishment of this disease in multiple countries worldwide in a variety of hosts, predicting how this fungus would behave, if it were to be introduced to New Zealand, is difficult. Swift and stringent eradication of the fungus could certainly prevent establishment and spread of pitch canker. However, in the absence of such control the risk factors present in New Zealand that could increase the incidence and severity of this disease have been highlighted in this report.

Nevertheless, if pitch canker were to be established in New Zealand, there are positive reports from other countries of success in lowering or maintaining the level of disease incidence and severity. For instance, the proper management of pine plantations in the southeast USA, the apparent remission of pitch canker in California and the prevention of spread of pitch canker both between nurseries and to plantations in Chile and South Africa. Regardless, continued vigilance and monitoring for this disease is essential for prevention or early detection of this pathogen in the forestry sector.

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