WESTERN GALL RUST — A THREAT TO *PINUS RADIATA* IN NEW ZEALAND

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ABSTRACT

Western gall rust (*Peridermium harknessii* J. P. Moore (syn. *Endocronartium harknessii* (J. P. Moore) Y. Hiratsuka) is potentially a serious threat to exotic *Pinus radiata* D. Don plantations of New Zealand although the pathogen has not been recorded here. Mechanisms that may have prevented invasion of the pathogen include geographic isolation, biological characteristics of the fungus, stand management, and regulatory mechanisms affecting transport and establishment. Major factors may include a low probability of importation of infected seedlings, unlikely spore transport in the atmosphere across the tropics, and asynchrony of rust sporulation and pine susceptibility in North America and New Zealand. The outbreak or "wave year" phenomenon in the native range of western gall rust demonstrates that both biological and microclimatic conditions must be suitable for establishment to occur. We conclude that the probability of invasion of New Zealand by western gall rust is very low; however, if the pathogen were to become established in New Zealand, the long-term effects may be large.

Keywords: invasion biology; *Peridermium (Endocronartium) harknessii*.

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INTRODUCTION

Western gall rust, caused by the fungus *Peridermium harknessii*, is a serious disease of most hard pines (subgenus *Pinus*), including *P. radiata* (Parmeter & Newhook 1967), in North America. As the rust is autoecious, it is considered to be a serious threat to *P. radiata* and other susceptible pines grown in the Southern Hemisphere (Old 1981; Old *et al*. 1986; van der Kamp 1989). In North America, the range of the disease extends from Mexico to the Yukon and from the Pacific to Atlantic coasts (CABI 2006); thus, the pathogen exists over broad climatic gradients. The mechanism of infection is by aeciospores that germinate and penetrate the epidermal layer of succulent elongating shoots, including the main leader (True 1938). Characteristic spherical galls are formed at the point of infection and sporulation typically occurs after 2 years (True 1938), although sporulation in the year of infection has been observed on inoculated juvenile *P. radiata* (Nelson 1971). Sporulation is annual, and it occurs in the Northern Hemisphere from early May until July (Moltzan *et al*. 2001). Galls on branches are important sources of inoculum for spread and intensification of the disease and they may cause a slight reduction in volume production; however, galls on the main stem cause serious damage to wood quality and lead to increased mortality due to stem breakage (Peterson 1961; van der Kamp 1989). Scientists in New Zealand have been cognisant of the potential risks to exotic plantation forestry posed by this pathogen for at least 50 years (Parmeter & Newhook 1967) as it is well known that *P. radiata* in California is extremely susceptible to the disease (Old 1981).

Successful biological invasions that result in the expansion of the range of a pathogen require conditions that allow transport, establishment, and spread of the pathogen (Liebhold *et al*. 1995). In this report, we describe (1) factors that influence the transport of *P. harknessii* to New Zealand and (2) factors that have prevented its establishment here.

FACTORS AFFECTING TRANSPORT OF *P. HARKNESSII* TO NEW ZEALAND

For an invasive organism to become established in a new geographic region, it must be transported to the new region and be in a viable state when it arrives (Liebhold *et al*. 1995). Several factors relating to the establishment of exotic pine plantations in New Zealand, as well as geographic, biological, and regulatory mechanisms, have prevented the pathogen from arriving in New Zealand.

Early Exotic Forest Establishment in New Zealand

Exotic forest plantations in New Zealand are currently composed of approximately 90% *P. radiata* (NZFOA 2005). This is the result of early growth and yield trials that were conducted to determine optimal species for softwood production in New

Zealand. The history of early *P. radiata* establishment in New Zealand is not known. One view is that mine workers introduced seed from California to Australia and then on to New Zealand. Another view is that seedlings were shipped from England, where *P. radiata* was introduced in the 1830s, to New Zealand by early settlers (Maddern Harris 1991). In addition to *P. radiata*, other hosts of western gall rust that were imported as seedlings included *P. ponderosa* P. Laws. ex C. Laws., *P. contorta* Dougl. ex. Loud., and *P. nigra* Arnold (Kirkland & Berg 1997).

The importation of diseased seedlings carries the highest risk of disease introduction, as is known to have occurred with the introduction of white pine blister rust (caused by *Cronartium ribicola* J. C. Fisch.) into North America. Unlike western gall rust, early infection by *C. ribicola* can be difficult to detect. In North America during the early 1900s, when diseased eastern white pine (*P. strobus* L.) seedlings were imported from Europe (the pathogen is thought to have originated in eastern Asia), seedlings with visible cankers were destroyed, but many seedlings with incipient cankers were nevertheless outplanted, and this established the pathogen (Hunt 2003). It is highly doubtful that trees infected with western gall rust would have been selected for export to New Zealand because of the visible symptoms of infection. Additionally, any seedlings sourced from England would have been rust-free as the fungus is not present there. Therefore, export hygiene practices, or the absence of the pathogen in England, may have prevented the establishment of western gall rust when the original *P. radiata* seedlings were imported into New Zealand.

Early settlers planted *P. radiata* in shelterbelts and seed collected from these shelterbelts was used to develop the exotic plantations in the 1920s and 1930s. Seed for improvement breeding was also obtained from native stands in California (Kirkland & Berg 1997). Although seed is an important carrier for fungi, western gall rust is not seed-borne and therefore could not be imported via this pathway.

Asynchronous Phenology

Another major factor contributing to the absence of the disease in New Zealand may be that the seasons in the Northern and Southern Hemispheres are out of phase. Rust sporulation coincides with the extension of the shoots and leaders of susceptible hosts in the spring (Moltzan *et al*. 2001). When sporulation is occurring on galls in the Northern Hemisphere, *P. radiata* in the Southern Hemisphere are entering autumn. The growing season of *P. radiata* in New Zealand is relatively long but the absolute amount of susceptible host material is less in the Southern Hemisphere autumn than in spring when the plants are growing vigorously. During the Southern Hemisphere spring, when host trees are particularly susceptible, no inoculum is being produced in the Northern Hemisphere, thereby reducing the chance of accidental introduction through transport of aeciospores.

Long-distance Dispersal

Long-distance dispersal is an important mechanism by which plant pathogens extend their range (Brown & Hovmøller 2002). As outlined by Aylor (1986), successful long-distance airborne dispersal requires a spore to escape the canopy, be transported both horizontally and vertically by the wind, survive the journey, be deposited on a susceptible host, and then encounter favourable environmental conditions for infection prior to death. Examples of successful long-distance dispersal include the introduction of sugarcane rust (*Puccinia melanocephala* H. and P. Syd.) from Cameroon into the Dominican Republic, establishment of coffee leaf rust (*Hemileia vastatrix* Berk. and Br.) from Angola into Brazil (Nagarajan & Singh 1990; Brown & Hovmøller 2002), and establishment of poplar rusts (*Melampsora larici-populina* Kleb. and *M. medusae* Thum.) from Australia into New Zealand (Spiers 1989). Air currents have been observed that would allow the passive east–west movement of these pathogens (Spiers 1989; Nagarajan & Singh 1990); however, no continental scale north–south migrations of pathogens have been documented (Ridley 2003). The likely explanation for prevention of passive north–south migration is the intertropical convergence zone (ITCZ), which effectively prevents spore movement between hemispheres at altitudes less than 6 km (Nagarajan & Singh 1990). Modelling the north–south dispersal of tobacco blue mould (*Peronospora tabacina* Adam) and wheat stem rust (*Puccinia graminis* Pers. f. sp. *tritici*) in the United States revealed the importance of the "green wave" of susceptible host material that allowed the pathogens to move northwards as host material became susceptible, as well as the importance of alternative dispersal mechanisms (Aylor 2003).

The *P. radiata* plantations of New Zealand are approximately 10 500 km from the southernmost extent of *P. harknessii* in North America. Given this long distance, the effect of spore dilution in the air, the lack of host material to bridge the distance, and the intertropical convergence zone, the probability of passive entry of live spores into New Zealand from aeciospores released in North America is extremely small. However, should western gall rust reach Australia, spores could reach New Zealand from that countrry's pine plantations, as did spores of the poplar rusts from Australian poplar plantations in the early 1970s (Spiers 1989).

Aeciospore Viability

It has been observed that *P. harknessii* aeciospore survival is favoured by low temperatures and low relative humidity (Hiratsuka *et al.* 1987; Chang & Blenis 1989) and for experimental purposes the spores are dried and stored at 0°C (van der Kamp 1988a) or in liquid nitrogen (Moltzan *et al.* 2001). In his analysis of New Zealand's susceptibility to invasion by exotic pathogens, Ridley (2003) suggested that in the past, the equatorial region has acted as a "filter" that reduced the survival

chances of any pathogens during transport from the Northern Hemisphere to the Southern Hemisphere. This equatorial filter was a result of the warm and moist environment of the tropics combined with the long period of transport in the early days of shipping. With increasing globalisation and decreased transit times of both containerised cargo and passengers on aircraft in conditions that are more favourable for spore survival, the risk of the importation of exotic pathogens, including *P. harknessii*, has increased (Ridley 2003).

New Zealand Biosecurity

Both the populace and the Government of New Zealand recognise the importance of biosecurity (defined as the exclusion, eradication, or effective management of risks posed by pests and diseases to the economy, environment, and human health) as evidenced by the Biosecurity Act (Biosecurity Council 2003). The Act resulted in the formation of Biosecurity New Zealand, an agency of the Ministry of Agriculture and Forestry that is responsible for biosecurity issues. Further evidence of the Government's commitment to biosecurity is the $320M budgeted by the Government in 2003–04 for the delivery of biosecurity services (Biosecurity New Zealand 2005). All passenger baggage entering New Zealand is screened by both dogs and X-ray devices to detect the presence of prohibited material, including plant material. Similarly, cargo is screened for prohibited items at the port of entry prior to release. These practices reduce the probability of an inadvertent introduction of *P. harknessii* into New Zealand.

FACTORS AFFECTING ESTABLISHMENT OF *P. HARKNESSII* IN NEW ZEALAND

As the pathogen has not been recorded in New Zealand, we are sure it has not been able to establish and spread here even if it managed to arrive. Although infection by one aeciospore is enough to initiate disease on a susceptible host, this pathogen is a typical fungus that produces abundant spores in order to ensure survival; therefore, it is likely biological factors have interacted with climatic factors to reduce the probability of establishment in New Zealand. In addition to biological factors of the pathogen, stand management practices in New Zealand have promoted conditions that are not favourable for pathogen establishment.

Aeciospore Production

Peridermium harknessii infection occurs primarily in the region of the elongating shoot prior to periderm formation (True 1938) and this region of susceptibility moves, keeping pace with the growing tip (Nelson 1971). As the number of aeciospores increases, the probability of successful disease initiation increases because there is a higher chance that one aeciospore or more will land on the small

region of susceptible tissue. The likelihood of infection is also increased as aeciospores are released over time, therefore increasing the probability that aeciospores are present when microclimatic conditions are suitable. When the inoculum level is high and microclimatic conditions are suitable for infection while host tissue is susceptible, the result is an outbreak or "wave year" (Peterson 1971). Wave years occur because, even in areas where western gall rust and the host have been present together for a very long time, abundant infection every year is not guaranteed. The wave year phenomenon in the endemic range of western gall rust suggests that establishment in New Zealand due to the arrival of a small number of aeciospores is extremely unlikely, although not impossible as a successful infection is initiated by a single aeciospore.

Fungicide Application

In the early 1960s, *Dothistroma pini* Hulbary, a serious foliar pathogen of *P. radiata*, was detected in New Zealand (Gilmour 1967). Epidemiology of this pathogen was studied and management practices were devised to reduce its impact (Bradshaw 2004). One of the major management strategies is aerial fungicide application. Since the season of 1966–67, an average of 66 600 ha of forest have been sprayed annually with copper-based fungicides (Bulman *et al.* 2004). The sensitivity of *P. harknessii* to copper-based fungicides has not specifically been tested; however, Mueller *et al*. (2005) found that broad-spectrum copper sulphate pentahydrate killed urediniospores of five species of *Puccinia* and one species of *Pucciniastrum*. In Minnesota western gall rust was controlled in high-value stands through the application of Maneb™, a manganese-based fungicide (Merrill & Kistler 1976). Fungicide application in New Zealand is not expected to protect trees from other exotic pathogens but there may have been a serendipitous, prophylactic effect that has protected *P. radiata* from western gall rust.

Pruning

Pruning of branches from the lower crown is conducted to increase recovery of clearwood. Between 1998 and 2003, approximately 70% of the 1.6 million ha of *P. radiata* growing in New Zealand was under a management regime that included pruning (NZFOA 2005). Pruning is normally conducted in three stages, in each of which all branches up to a specified stem diameter are removed, and the final pruning is normally to a height of 6.5 m; this can be as late as 10 years after planting. After pruning, 3–4 m of green crown is retained (Maclaren 1993). As *P. harknessii* infects only the elongating shoot tip and branches of host trees, removal of a large proportion of lateral branches significantly reduces the susceptible surface area available for infection. Removal of the lower branches also has the effect of opening up the canopy, thus reducing the relative humidity in the stand. It has been observed

in British Columbia that infection rate, expressed as galls per metre, decreases rapidly with height above ground and it is hypothesised that this is a result of higher relative humidity and shade, and moderate temperatures in the lower crown (van der Kamp 1988b). Thus, removal of the lower branches through pruning reduces the surface area available for infection and induces conditions less favourable for infection by the pathogen.

DISCUSSION

Successful invasion of New Zealand by *P. harknessii* requires suitable long-distance transport, and successful establishment requires optimal microclimatic conditions at a time when *P. radiata* is susceptible to infection. Passive transport of the pathogen into New Zealand appears extremely unlikely. Therefore, the most likely way that an invasion could occur is via the inadvertent importation of live infected plant material from North America.

Although the probability of accidental introduction of *P. harknessii* is low, it is not zero, and if the pathogen were to become established here, many factors would favour its development. It does not need an alternate host to complete its life cycle, as required by rust fungi in the closely related genus *Cronartium*, and so no protection is afforded by the absence of an alternate host and the pathogen could spread from pine to pine. Additionally, population genetics analysis (Vogler *et al*. 1991) and the observation of aecia on galls that had not produced spermogonia (Crane *et al*. 1995) indicate that the rust may be homothallic and thus able to produce aeciospores without fertilisation. Both of these factors suggest that a serious outbreak could be initiated by a small number of infection events if the "wave year" criteria were met. One of the major assumptions underlying the perceived risk of *P. harknessii* in New Zealand is that the climate would be suitable for establishment and spread of the pathogen (Old 1981; van der Kamp 1989). This assumption is the focus of a climate matching project that we are currently undertaking (Ramsfield *et al*. in press). We hope to improve our understanding of the climatic limitations of this pathogen in North America as well as to determine which portions of the Southern Hemisphere are at the highest risk of establishment of *P. harknessii*.

Live *P. radiata* seedlings are prohibited from being imported into New Zealand (M.Ormsby, Biosecurity New Zealand, pers. comm.), legally closing the major pathway through which the disease could become established here. Nevertheless, however small the risk of accidental introduction of the pathogen, regulatory authorities and foresters view western gall rust as a serious threat to the exotic forest industry of New Zealand. This has led to the development of DNA-based identification methods that can detect the pathogen within non-sporulating galls (Ramsfield & Vogler 2004). If the disease were detected on non-sporulating galls,

a response could be mounted before sporulation and spread of the pathogen. Because it would be very difficult to contain western gall rust in New Zealand if it was first detected after aeciospores were produced on galls, forest health officers are trained to identify and report any symptoms of western gall rust that they observe. Western gall rust has not been recorded in New Zealand, and the probability of establishment is low; however, we cannot be complacent and assume that the disease will never arrive here. We must ensure that all necessary steps remain in place to prevent establishment of this pathogen and be fully prepared to act if it is introduced.

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