A Comparison of Host Susceptibilities to Native and Exotic Pathogens in Forest Tree Ecosystems

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ABSTRACT

Forest tree ecosystems are in danger of being disrupted by novel pathogens. It is important to examine factors that influence host susceptibility to these pathogens. There are three main components that contribute to host susceptibility: pathogen specific defense mechanisms, genetic bases, and pathogen generic defense mechanisms. Different susceptibilities can be found with varying tree and pathogen species and not every combination of host species and pathogen species will be influenced by the same factors. This review explores how different host tree species react to native and exotic diseases that affect their respective species. It has been determined that *Eucalyptus globulus* and *Corymbia citriodora* ssp. *variegate* show a form of pathogen-specific defense mechanisms to the native diseases *Teratospheaeria sp.* and *Quambalaria pitereka* respectively, with strong susceptibility to the exotic disease *Austropuccinia psisii. Alnus glutinosa,* however, shows that there is a genetic basis for pathogen resistance to *Phytopthora uniformis* and *Phytopthora x alni.* Lastly, *Pinus radiata* showed a generic form of pathogen defense towards *Fusarium circinatum.*

Introduction

Disease occurs when a pathogen comes into contact with a susceptible host within a favorable environment (Klopfenstein et al., 2009). A host's resistance to disease plays a large role in the chance of becoming infected (Klopfenstein et al., 2009). Globally, a variety of diseases and introduced pests threaten forest ecosystems, the most devastatingly of which are fungal or fungal-like organisms (Ahrens et al., 2018; Bjelke et al., 2016). It is critical that we examine what influences a tree's susceptibility to disease and pests in order to properly implement management strategies to maintain forest function and resilience (Ahrens et al., 2018). Additionally, due to the high socioeconomic value of forest ecosystems, which was globally estimated at $4.7 trillion in 2001, recognition and elimination of certain fungal pathogens in these ecosystems is of high importance (Krieger, 2001;Fisher et al., 2012). It has been documented that plants with complex pathogen detection systems and resistance are evolutionarily driven by selection pressures (Anderson et al., 2018) as well as migration and genetic drift (Burdon and Thrall, 2008). There are complex interactions between a pathogen and its host and understanding the ecological implications can be complicated (Hamilton et al., 2013). Typically, the immune system of plants functions by recognizing pathogens, carrying out signal transduction, and defensively responding through pathways involving genes and their products, however pathogens attempt to evade these pathways and systems, which can lead to successful infection (Andersen et al., 2018). The more a host has evolved resistance to a pathogen, the less severe the infection or the less likely they are to be infected (Klopfenstein et al., 2009; Hamilton et al., 2013). Current forest management strategies to mitigate pathogen infections include the use of fungicides, altering species composition and favoring forest species that are adaptive to changes in climatic conditions and/or resilient to specific diseases and pests (Klopfenstein et al., 2009; Fisher et al., 2012). This review will examine the factors that influence host susceptibility to novel pathogens in forest tree ecosystems and determine if these resistance factors can be passed on through generations.

Pathogen Specific Defense Mechanisms

Two tree species *Eucalyptus globulus* and *Corymbia citriodora* ssp. *variegata* (CCV) are species of trees found across southeastern Australia, Tasmania, and the Bass Straight Islands and are currently being plagued by a novel, exotic disease known as Myrtle rust, or *Austropuccini psidii*. Susceptibility to *A. psidii* was assessed using artificial inoculation of seedlings from open-pollinated families which derived from wild populations of *E. globulus* and CCV. Separately, both trees were inoculated with their native diseases; *E. globulus* was inoculated with Teratospheria Leaf Disease (TLD) caused by *Teratosphaeria spp.* and CCV was inoculated with Quambalaria shoot blight, caused by *Quambalaria pitereka*. This was performed to compare the susceptibilities of the trees to native diseases versus the exotic disease.

Narrow-sense heritability estimates were created for *A. psidii* and TLD within *E. globulus* in multiple batches and trial sites. It was found that *E. globulus* had a high heritability of susceptibility for *A. psidii* compared to TLD. Heritability of susceptibility was on average over twofold greater for *A. psidii* (0.65) compared to TLD (0.23) (Table 1 from Freeman et al., 2019). This would imply that there was an increased genetic variation for host susceptibility to the exotic disease *A. psidii* compared to the native disease TLD, making hosts more susceptible to the exotic disease than the native disease.

Table 1. Narrow-sense heritability (h^2_{op}) estimates for *Austropuccini psidii* and Teratospheria leaf disease (TLD) damage in *Eucalyptus globulus* (Freeman et al., 2019).

Pathogen and assessment[a]	h^2_{op}	SE	p[b]
A. psidii			
Batch 1	0.63	0.09	< 0.001
Batch 2	0.70	0.10	< 0.001
Combined[c]	0.65	0.07	< 0.001
TLD site			
Tog05	0.22	0.05	< 0.001
SR05	0.17	0.04	< 0.001
Temma06	0.26	0.05	< 0.001
SR06	0.13	0.03	< 0.001
GC08	0.35	0.06	< 0.001
Average	0.23		

[a]Screening batch for *A. psidii* artificial inoculations and trial site for TLD natural infections (Freeman et al., 2019).
[b]Significance of the additive genetic variance from zero (Freeman et al., 2019).
[c]Combined data with the difference between batches included in the modelled replicate effect (Freeman et al., 2019).

Quantitative inbreeding coefficients were also analyzed for TLD and *A. psidii* within *E. globulus*. The average quantitative inbreeding coefficient for TLD damage was 0.14 while the quantitative inbreeding coefficient for *A. psidii* (Rust) was 0.06 (Table 2 from Freeman et al., 2019). If pathogen imposed selection had shaped the evolution of these host species, it would be expected that population differentiation (Q_{st}) to host susceptibility would exceed that expected through drift, seen using a neutral marker F_{st} measured at an average of 0.09 (Freeman et al., 2019). This implies that host populations have more differentiated susceptibility to the native pathogen than the exotic pathogen in cases exceeding that expected through drift, providing evidence for pathogen-imposed selection.

Table 2. Quantitative inbreeding coefficients (Q_{st}) for Teratospheria leaf disease (TLD) and *Austropuccini psidii* (rust) damage in *Eucalyptus globulus*, and correlations with susceptibility to *A. psidii* damage for TLD in each field trial (Freeman et al., 2019).

						Population correlations (r_p)			Additive genetic Correlations (r_a)		
Pathogen and trial	n_{farms}[a]	n	Q_{ST}	SE	$P\ (Q_{ST\ TLD} = Q_{ST\ rust})$	r_p	SE	$P\ (r_a = 0)$	r_g	SE	$P\ (r_a = 0)$
TLD											
Tog05	140	2295	0.05	(0.03)	0.655	−0.36	(0.41)	0.417	−0.10	(0.17)	0.610
SR05	146	2727	0.12	(0.06)	0.173	−0.38	(0.34)	0.313	0.08	(0.16)	0.624
Temma06	124	2771	0.25	(0.09)	0.005	0.06	(0.35)	0.888	0.21	(0.15)	0.182
SR06	140	2863	0.12	(0.06)	0.168	0.29	(0.35)	0.431	0.16	(0.17)	0.354
GC08	141	2236	0.16	(0.07)	0.057	0.49	(0.30)	0.164	−0.04	(0.15)	0.806
Average			0.14			0.02			0.06		
Rust	189	2597	0.06	(0.03)							

[a]n_{farms} indicate the number of families, and n the number of individuals, used for TLD damage estimates in each field trial (Freeman et al., 2019).

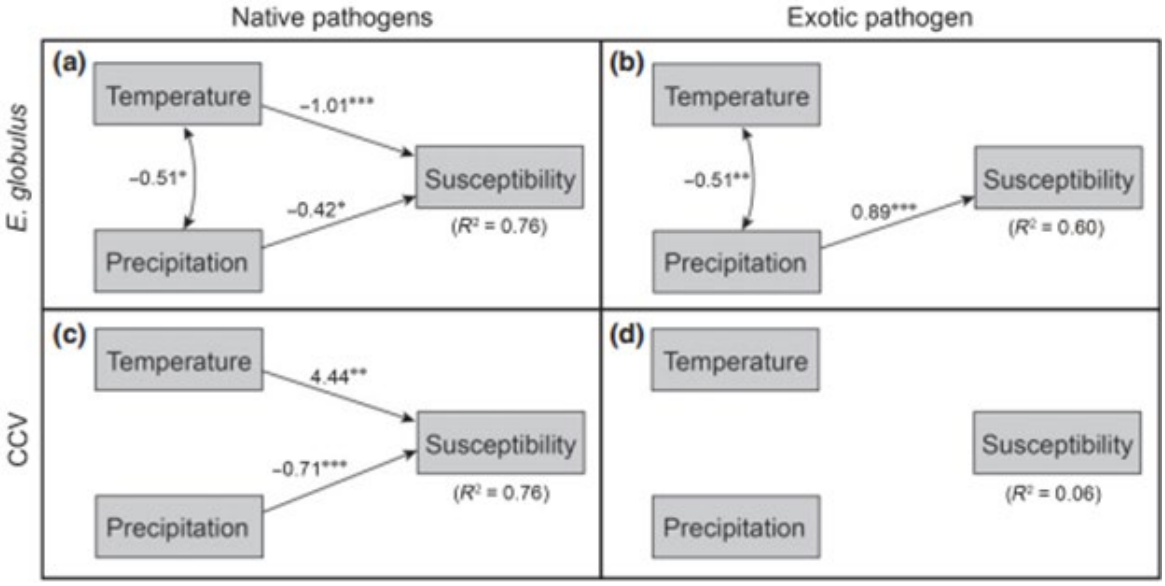

Figure 1. Path diagram depicting the effects of climate on host susceptibility within different pathosystems: (a) *Eucalyptus globulus/Teratosphaeria*; (b) *Eucalyptus globulus/Austropuccinia psidii*; (c) *Corymbia citriodora* ssp. *variegata* (CCV)/*Quambalaria pitereka*; (d) *Corymbia citriodora* ssp. *variegate/Austropuccini psidii* (Freeman et al., 2019).

Temperature and precipitation of home-site climate were compared for *E. globulus* and CCV for both their native and exotic diseases. An increase in temperature and precipitation led to a decrease in *E. globulus* susceptibility to the native pathogen, however increased precipitation led to an increase in *E. globus* susceptibility to the exotic pathogen while temperature had no impact (Figure 1 from Freeman et al., 2019). An increase in temperature led to an increase susceptibility and a decrease in precipitation led to a decrease in susceptibility for CCV to the native pathogen, while temperature and precipitation had no meaningful impact on CCV to the exotic pathogen (Figure 1 from Freeman et al., 2019). Areas that have increased temperature and precipitation are more prone to fungal diseases. Susceptibility

generally decreases where climate increased disease risk for the native pathogen but not the exotic pathogen (Freeman et al., 2019). This would also provide evidence for pathogen-imposed selection.

Genetic Basis for Population Pathogen Resistance

Two pathogen species commonly infect Alders (*Alnus glutinosa*); *Phytopthora uniformis* (PU) and *Phytopthora x alni* (PA). Seedling progenies of mother trees from both uninfected and infected areas were inoculated with either PA or PU. Their survival rate were studied 10 days post inoculation and recorded as either healthy (H), symptomatic (S), or dead (D).

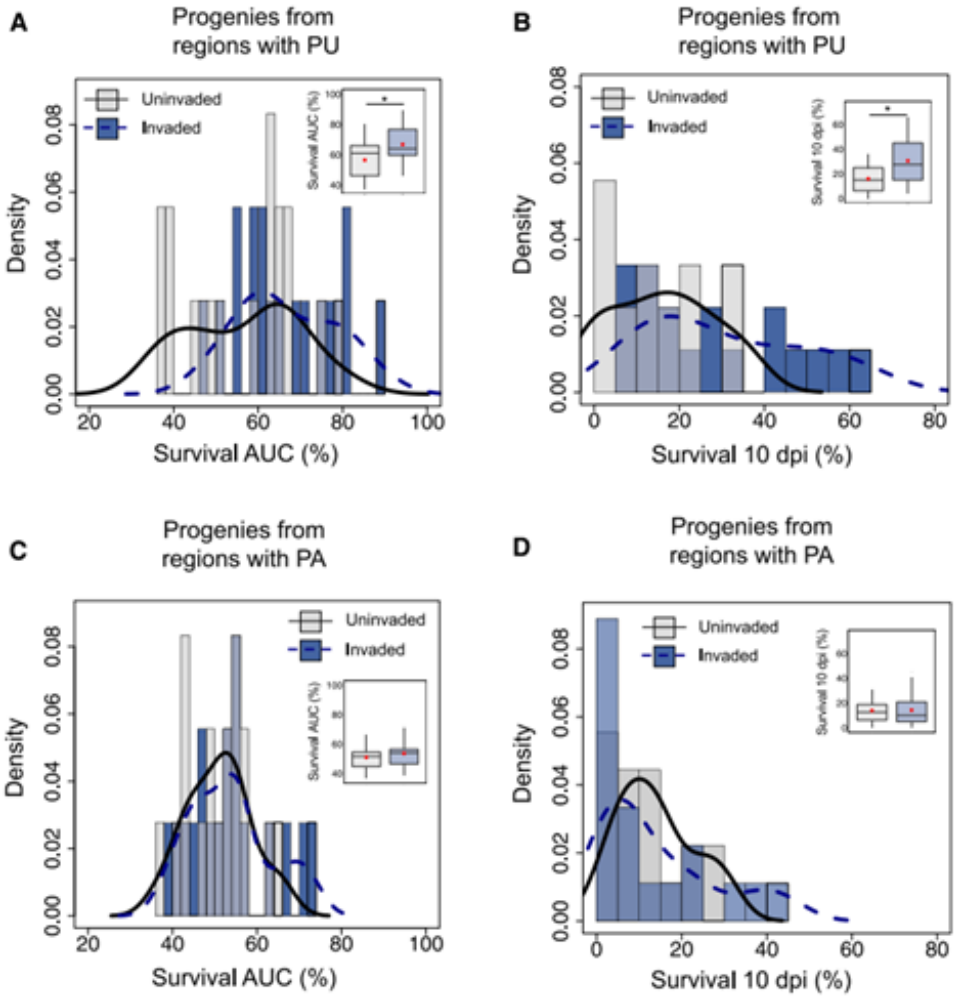

Figure 2. Distributions of the survival of progenies of alder mother trees from *Phytopthora uniformis* (PU) and *Phytopthora* x *alni*. **A and B,** The histogram, density lines, and box plot of the area under the curve (AUC) and survival 10 days post inoculation (dpi), respectively of alder progenies from regions with PU and inoculated with PU. **C and D,** The histogram, density lines, and box plot of the area under the curve (AUC) and survival 10 days post inoculation (dpi), respectively of alder progenies from regions with PU and inoculated with PA. Dots on the box plots depict the averages for each group. Asterisks indicate significant differences at $P < 0.05$ (Redondo et al., 2019).

Progenies from regions previously invaded by PU had a high survival rate compared to progenies that were uninvaded (Figure 2A and 2B from Redondo et al., 2019). Survival rate for seedlings from the invaded sites were almost twice that of the survival rate from the uninfected sites. Progenies from regions previously invaded by PA had a slightly higher survival rate compared to progenies that were uninvaded (Figure 2C and 2D from Redondo et al., 2019). A higher survival rate in progenies that had previously been exposed to the pathogen implies that there is a genetic component in pathogen resistance that the parent trees have passed on.

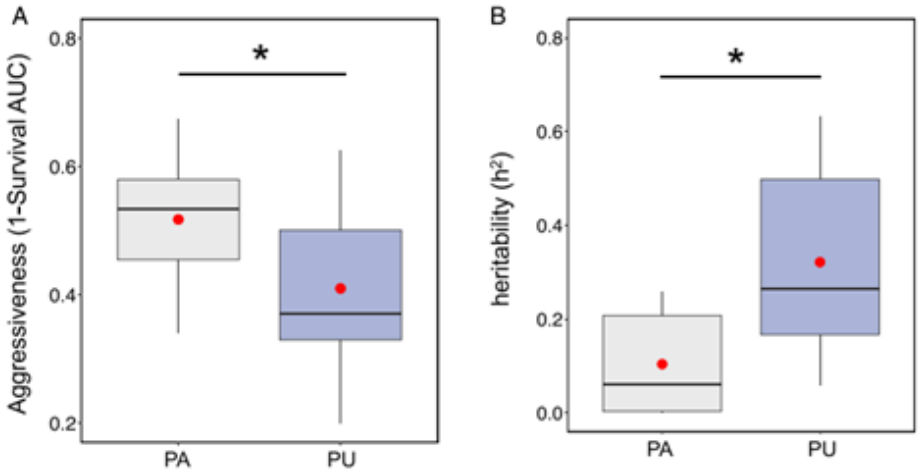

Figure 3. Response of families from uninvaded sites to *Phytopthoa* x *alni* (PA) and *P. uniformis* (PU). **A,** Differences in aggressiveness between PU and PA. **B,** Differences in narrow sense heritability (h^2) of the half-sibling populations (n=6) of alders from uninvaded sites when inoculated with PA and PU. AUC = area under the curve (Redondo et al., 2019).

Narrow-sense heritability and aggressiveness of PU and PA were different. Although PA was more aggressive than PU, the susceptibility to PU of uninvaded populations was much larger than that of PA (Figure 3 from Redondo et al., 2019). A higher narrow-sense heritability would imply that there is more genetic variation among susceptibility for PU, further proving evidence that genetics plays a role within pathogen resistance.

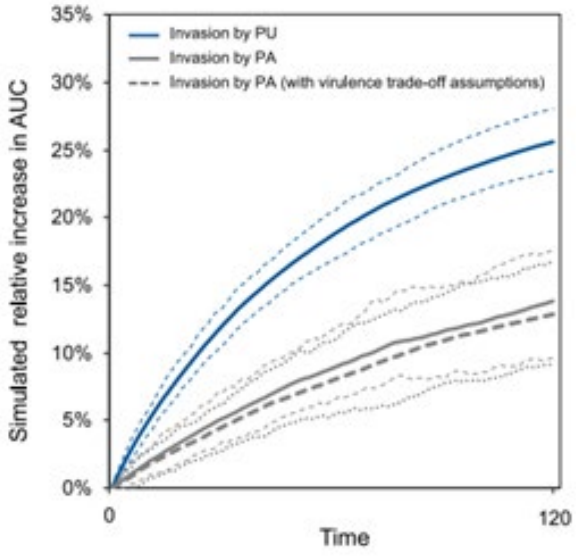

Figure 4. Simulations of a shift in the average survival of a naïve alder population over time as a consequence of invasion by either *Phytopthora* x *alni* (PA) or *P. uniformis* (PU) (Redondo et al., 2019).

The aggressiveness narrow-sense heritability factors were used as inputs for a simulated experiment which modeled how the survival rate of alder seedlings would shift over time as a consequence of invasion by PA or PU. The survival rate increased quicker for PU than PA (Figure 4 from Redondo et al., 2019). This would be due to the higher narrow-sense heritability of PU compared to PA, despite the higher aggressiveness of PA than PU. This implies that there is a strong genetic component that plays a role within ongoing pathogen-resistance mechanisms.

Pathogen Generic Mechanisms

The Monterey Pine (*Pinus radiata*) is highly susceptible to the disease Pitch canker caused by *Fusarium circinatum.* It has been previously documented that the Monterey Pine has at least some heritable variation for resistance (Reynolds et al., 2019). Multiple sets of trees were observed: standing trees from native forests, seedlings from breeding populations, seedlings established from native forests, seedlings grown in a greenhouse, and seedlings grown in the field.

Seedling progeny from previously established trees were compared. Seedlings from a Chilean breeding population and seedlings from a native forest were inoculated with *F. circinatum* by applying a liquid suspension into small wounds on the main stem, and lesion lengths were measured to the nearest millimeter. These lesion lengths were used to display Phenotypic Susceptibility. Graphs were created to display these findings. Both seedling graphs showed normal distribution for susceptibility (Figures 5 and 6 from Reynolds et al., 2019).

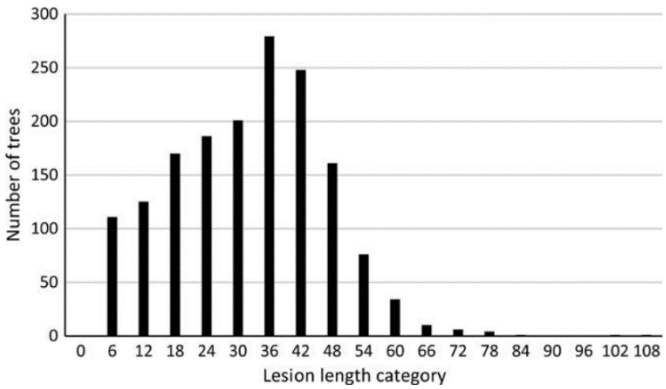

Figure 5. Histogram of lesion length data for seedlings representative of families in a Chilean breeding population of *Pinus radiata*. The number on the x-axis represents the upper end of the range of lesion lengths (in mm) for that category (Reynolds et al., 2019).

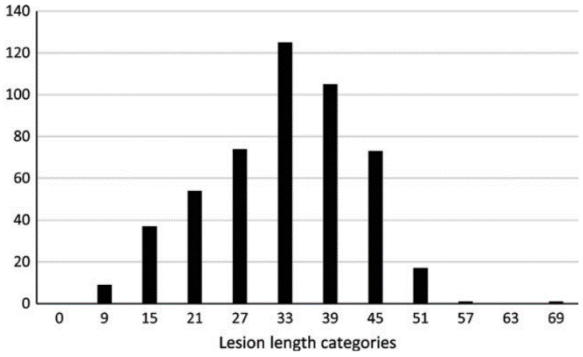

Figure 6. Histogram of lesion length data for seedling progeny of 62 randomly selected mature *Pinus radiata* trees in a native forest. The number on the x-axis represents the upper end of the range of lesion lengths (in mm) for that category (Reynolds et al., 2019).

The same procedure was performed for seedling progeny of trees native to Hatton Canyon and compared to lesion lengths of the parent trees. The data was positively skewed for the parent trees (Figure 7 from Reynolds et al., 2019) while there was a relatively normal distribution for their progeny (Figure 8 from Reynolds et al., 2019).

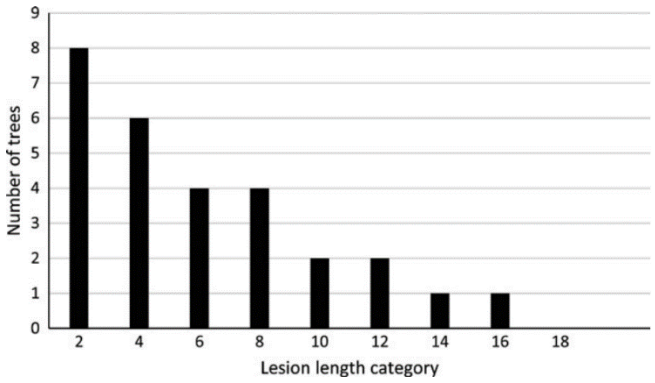

Figure 7. Histogram of lesion length data for parent trees in the Hatton Canyon stand in a native forest of *Pinus radiata*. The number on the x-axis represents the upper end of the range of lesion lengths (in mm) for that category (Reynolds et al., 2019).

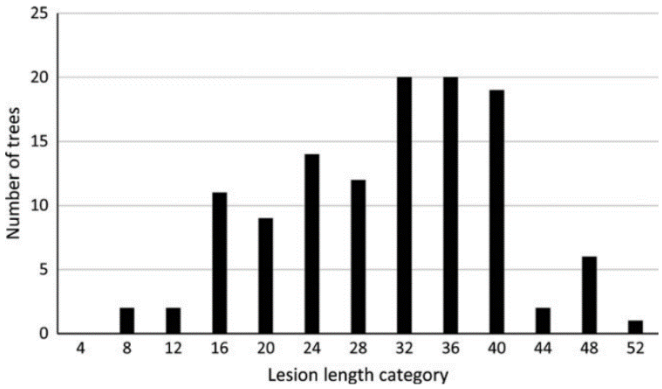

Figure 8. Histogram of lesion length data for naïve seedling progeny of trees in the Hatton Canyon stand in a native forest of *Pinus radiata*. The number on the x-axis represents the upper end of the range of lesion lengths (in mm) for that category (Reynolds et al., 2019).

Seedlings from parent trees within native forests were grown in different conditions; one set was grown in greenhouses and the other was grown in the field. The field-grown trees had a longer incubation time and experienced nearly six times more precipitation than those grown in greenhouses. Additionally, the field-grown trees had experienced other fungal infections such as Diplodia dieback and gall rust caused by *Endocranartium harknessii*. Both sets of seedlings were inoculated with *F. circinatum* and their lesion lengths were recorded. There was a larger median range for the field-grown trees compared to the greenhouse-grown trees (Table 3 from Reynolds et al., 2019). It is most likely that shifts towards higher levels of resistance are due to the frequency of challenges by microorganisms, such as the ones the field-grown trees experienced.

Table 3 Lesion length data for progeny of native trees that were grown in the greenhouse of in the forest (Reynolds et al, 2019).

Site[a]	Replication	Trees	Mean	Median	Range
Greenhouse	1	177	46.3 ± 1.22 mm	48 mm	6-84 mm
Greenhouse	2	187	47.6 ± 1.12 mm	48 mm	10-100 mm
Field	1	149	41.2 ± 3.0 mm	25 mm	1-132 mm
Field	2	124	70.5 ± 4.3 mm	59 mm	1-183 mm

[a]Where trees were grown prior to inoculation. [b]Number of trees on which means, medians, and ranges are based. (Reynolds et al, 2019).

Graphs of these trees were created. The Greenhouse-grown trees showed a normal distribution while the data for the field-grown trees was positively skewed (Figures 9, 10, 11 from Reynolds et al., 2019). This would overall imply that the proportion of trees that appear resistant to pitch canker is greater than what would be predicted by their progeny.

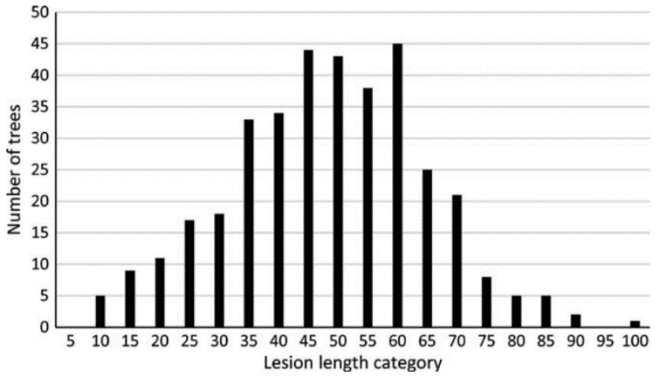

Figure 9 Histogram of lesion length data for naïve (greenhouse-grown) seedling progeny of trees in the native stand of *Pinus radiata* at the old quarry site. The number on the x-axis represents the upper end of the range of lesion lengths (in mm) for that category (Reynolds et al., 2019).

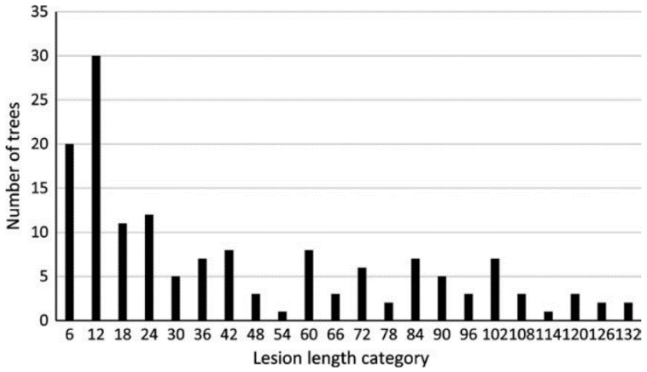

Figure 10 Histogram of lesion length data for the first replication of field-grown seedling progeny of trees in the native stand of *Pinus radiata* at the old quarry site. The number on the x-axis represents the upper end of the range of lesion lengths (in mm) for that category (Reynolds et al., 2019).

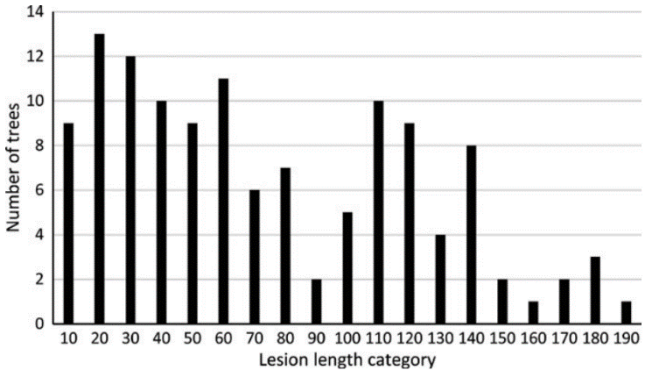

Figure 11 Histogram of lesion length data for the second replication of field-grown seedling progeny of trees in the native stand of *Pinus radiata* at the old quarry site. The number on the x-axis represents the upper end of the range of lesion lengths (in mm) for that category (Reynolds et al., 2019)

Conclusion

These three situations provide evidence for three different factors that affect host susceptibility to pathogens in forest tree ecosystems; pathogen specific defense mechanisms, genetic basis, and pathogen generic defense systems. In the case of *Eucalyptus globulus* and *Corymbia citriodora* ssp. *variegate* infections by *Austropuccini psidii*, studies showed that host susceptibilities to the native and exotic pathogens were uncorrelated, and rather it was pathogen-specific mechanisms that played a major role in resistance. The case of *Phytopthora alni* proved that there is a genetic basis for ongoing genetic resistance which can be passed on to their progeny. Despite this, there are other studies that show that interactions between *P. alni* and its host are likely impacted by additional biotic and abiotic environmental conditions which could affect the spread of the pathogen (Bjelke et al., 2016). Additionally, the case of *Pinus radiata* and *Fusarium circinatum* provided evidence for a generic form of pathogen resistance and showed that susceptibility of greenhouse-grown seedling progeny are not representative of standing communities. Alternatively, studies of other pine species have shown that susceptibility of greenhouse-grown seedlings are correlated with disease incidence observed in the field (Wingfield et al., 2008).

Since every tree species is different, there could be an indefinite number of factors that play a role in host susceptibility to forest pathogens. Pathogen growth and disease expression are strongly influenced by other environmental conditions (Soewarto et al., 2020). Abiotic conditions were not covered within this review and could prove to be worth studying in the future, as climate change increasingly threatens the health of forests globally by affecting pathogen-host interactions (Klopfenstein et al., 2009). Other future studies that could prove useful could include examining defense mechanisms against multiple pathogens in coinfected trees.

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