

# Altered Distribution of Susceptibility Phenotypes Implies Environmental Modulation of Genetic Resistance

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Resistance to disease is determined by the genetic capacity of a plant to recognize and respond to a pathogen, as modified to varying degrees by the environment in which the interaction occurs. Physical factors such as temperature and moisture can limit the ability of a pathogen to infect and cause disease, and may also influence the response of the host through effects on gene expression and/or by imposing constraints on physiological activities required to deliver an effective defense. Recent research has also drawn attention to the potential for the biotic environment to modulate susceptibility to disease. Thus, resistance may be enhanced by endophytic microbes and also by sub-lethal exposure to a plant pathogen. For example, studies under controlled conditions document that systemic-induced resistance (SIR) to pitch canker, caused by *Fusarium circinatum*, is operative in *Pinus radiata* D. Don (Monterey pine) (Bonello et al. 2001). Evidence for SIR in natural populations of *P. radiata* derives from studies showing that trees are more resistant to pitch canker in areas where the disease is of long residence than trees in areas where the disease is only recently established. Likewise, in a given stand, trees tend to become more resistant with time after establishment of pitch canker (Gordon et al. 2011). These observations suggest that susceptibility to a disease may be influenced as much by the history of exposure to a pathogen as by inherent genetic resistance.

To explore this possibility, we compared the distribution of virulence phenotypes in a native stand of *P. radiata* to a population of seedlings reared from seed collected from the same stand. Seedlings were maintained in a greenhouse and were not exposed to *F. circinatum*. At 1.5-years-of-age, each tree was inoculated once on the main stem, as described by Gordon et al. (1998). Three weeks later, the length of the lesion at the site of inoculation was measured. Lesion length was used as a proxy for susceptibility, with the more resistant trees sustaining shorter lesions than trees that are more susceptible. Lesion lengths on 614 trees were approximately normally distributed around a mean of 29.5 mm (fig. 1), which is consistent with resistance to pitch canker being a quantitatively inherited trait (Matheson et al. 2006). In contrast, lesion lengths in parent trees were not normally distributed, but instead were arrayed in a right skewed distribution (fig. 2).

The comparatively high proportion of short lesion lengths among parent trees cannot be attributed to a loss of susceptible trees due to pitch canker because the disease became established only after the sampled trees were mature, and no pitch canker caused mortality had occurred within that population. On the other hand, we cannot exclude the possibility that ontogenetic resistance contributed to the observed difference between progeny and parent trees. However, there is no evidence that susceptibility to pitch canker changes with the age of a tree. This is illustrated by results of an experiment in which 148 trees ranging in size from 3.5 to 77.5 cm were inoculated on each of three branches (fig. 3). Regression of lesion length on tree diameter (DBH) yields an  $R^2$  of 0.006, indicating that DBH (as a surrogate for age) accounts for a negligible proportion of the observed variation.

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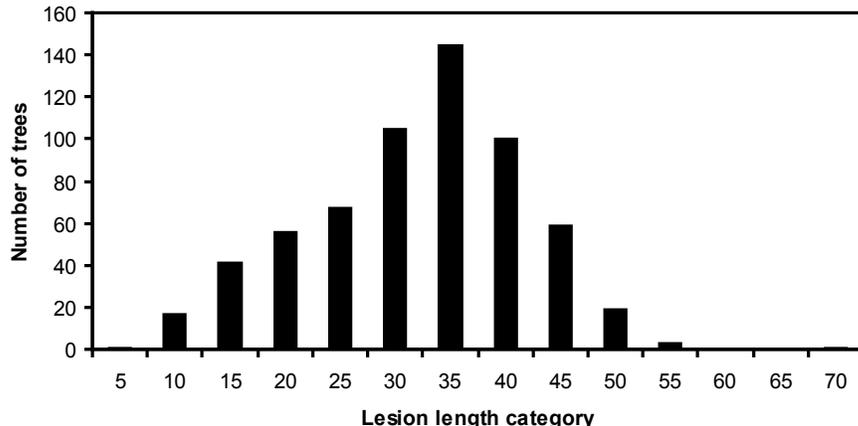


Figure 1—The number of trees corresponding to each lesion length category. Each category encompasses a range of 5mm, with the number on the X-axis representing the upper end of the range in mm.

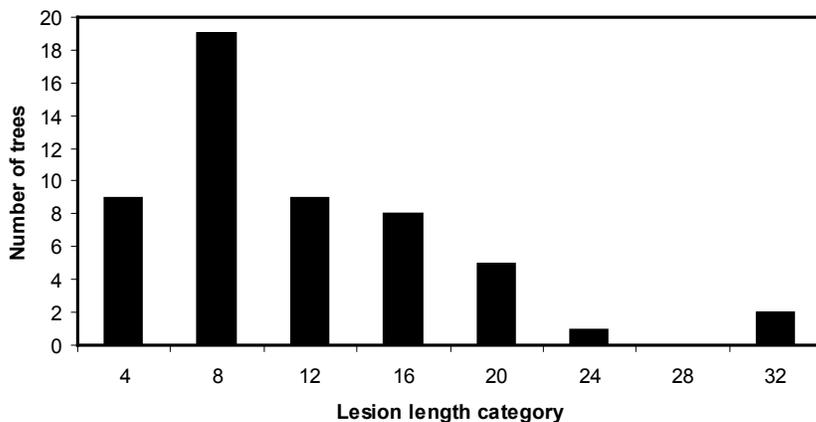


Figure 2—The number of trees corresponding to each lesion length category. Each category encompasses a range of 5mm, with the number on the X-axis representing the upper end of the range in mm.

An alternative explanation for a different distribution of lesion lengths between parent trees and their progeny is that environmental factors are influencing susceptibility, and more specifically, that exposure to parasitic microbes leads to a shift in the distribution of susceptibility phenotypes toward greater resistance. To test the merits of this proposition, we developed a simple model to simulate the transition from a normal distribution of lesion lengths (as occurs in unexposed seedlings) to the right-skewed distribution observed in the parent population. The model includes a stochastic contagious contact distribution between trees and the pathogen, which models exposure, and a host phenotypic response function that determines the impact of exposure events on lesion length. An outline of the model is shown in the schematic in fig. 4.

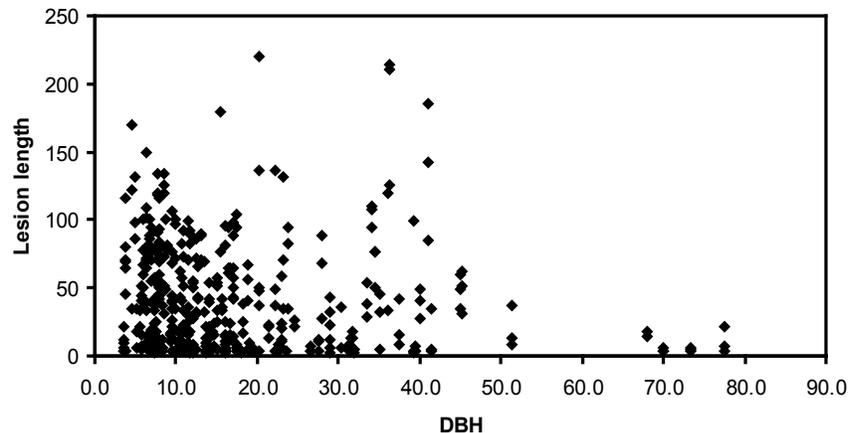


Figure 3—Each point corresponds to the length of a lesion on an inoculated branch (Y-axis) and the DBH (X-axis) of the tree. Each tree was inoculated on three branches and so is represented by three data points in this graph.

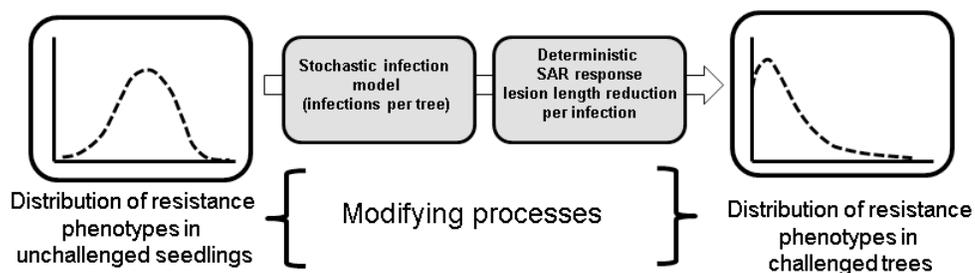


Figure 4—A schematic of the model used to simulate the transforming effect of induced resistance on the distribution of susceptibility phenotypes. (SAR = systemic acquired resistance).

Using parameters that are consistent with what we know about the epidemiology of pitch canker, the model effectively converts a normal distribution to one that resembles the pattern observed in nature. Experimental support for the operation of this process will be sought by establishing a population from seed and documenting that changes over time are associated with exposure to the pitch canker pathogen.

## Literature Cited

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