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First evidence of genetic-based tolerance to red needle cast caused by *Phytophthora pluvialis* in radiata pine

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Abstract

Background: Red needle cast (RNC) is a new needle disease of *Pinus radiata* D. Don (radiata pine) in New Zealand that is causing significant, but as-yet un-quantified, loss of growth and productivity. This foliar disease has recently been attributed to the infection of the needles by *Phytophthora pluvialis* Reeser, Sutton & E Hansen. Genetic improvement is seen as a possible solution to mitigate the effects of this needle disease on forest productivity.

Findings: To quantify the ability of genetics to provide a solution, RNC was assessed on a single clones-within-families genetics trial using two methods: the percentage needle cast that was attributable to red needle cast symptoms; and the percentage needle cast where the causal agent was not clearly identifiable. Both needle cast assessment methods were found to be heritable ($\hat{h}^2$ 0.21-0.31).

Conclusions: Selecting for tolerance to RNC is likely to deliver healthier trees. More assessments across a number of sites and seasons are required to confirm this result.

Keywords: *Pinus radiata*; Forest health; Needle cast disease; Tree breeding; Genetics; Red needle cast; *Phytophthora pluvialis*

Introduction

Red needle cast (RNC) is a new needle disease caused by *Phytophthora pluvialis* Reeser, Sutton & E Hansen that causes defoliation of *Pinus radiata* D. Don (radiata pine) under conditions favourable to the development of the disease (Dick et al. 2014; Hansen et al. 2012; Hood et al. 2014; Reeser et al. 2013). The disease has become well established in certain areas of New Zealand and has the potential to cause production loss through needle shed and reduced growth. Infection appears to be limited to needles of infected trees, with no recoveries of *Phytophthora pluvialis* having been made from the roots, stems or branches (Dick et al. 2014). The risk of any transfer on logs appears to be negligible (Hood et al. 2014). Past experience with other needle diseases such as Dothistroma needle blight suggest that such diseases can be managed in the medium-to-long term with tree breeding and appropriate silviculture. This study aimed at giving an early indication of the potential to select for better tolerance to RNC.

Selective breeding for tolerance to *Phytophthora* spp. has been successful in several tree species with at least some differences in susceptibility and tolerance observed. These results demonstrate that there is scope for the expansion of breeding to additional species (Table 1). To date, the focus of such programmes has been on the development of tolerance to soil- borne pathogens, such as *P. cinnamomi* Rands, *P. drechsleri* Tucker, *P. lateralis* Tucker and Milbrath and *P. cactorum* (Lebert & Cohn) Shröt. There appears to be no long-term breeding programme focussed on the development of tolerance to pathogenetic *Phytophthora* spp. affecting leaves or needles of woody plants in forest ecosystems. This is primarily due to the relatively recent emergence of species such as *P. ramorum, P. pinifolia* and *P. pluvialis* as significant foliar pathogens within coniferous forest systems (Durán et al. 2008; Werres et al. 2001; Dick et al. 2014), and contemporary recognition of *Phytophthora* species as foliar pathogens of mature trees under appropriate climatic

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Table 1 Forest tree species that have shown some differential tolerance or susceptibility to infection by *Phytophthora* pathogens

Host		*Phytophthora* species	Reference
Common name	Latin name		
Port-Orford-Cedar	*Chamaecyparis lawsoniana* (A. Murray) Parl.	*P. lateralis* (Tucker & Milbrath)	Hansen et al. 1989
		P. cinnamomi Rands	Green et al. 2013
			Sniezko et al. 2000
			Bower et al. 2000
Shortleaf pine	*Pinus echinata* Mill.	*P. cinnamomi*	Zentmyer 1980
Jarrah	*Eucalyptus marginata* Donn ex Sm.	*P. cinnamomi*	McComb et al. 1991
			Stukely and Crane 1994
			Stukely et al. 2007
Mountain ash	*Eucalyptus regnans* F.Muell.	*P. cinnamomi*	Harris et al. 1985
Gully gum/blackbutt peppermint	*Eucalyptus smithii* R.T.Baker	*P. cinnamomi*	Brasier 2000
Radiata pine	*Pinus radiata* D. Don.	*P. cinnamomi*	Butcher et al. 1984
Fraser fir	*Abies fraseri* (Pursh) Poi	*P. cactorum* (Lebert & Cohn) Schröt.	Frampton et al. 2013
Canaan fir	*Abies balsamea* var. *phanerolepis* (Fern.)	*P. cinnamomi*	
Nordmann fir	*Abies nordmanniana* (Steven) Spach	*P. drechsleri* Tucker	Hoover 2013
Trojan fir	*subsp. equitrojani* (Asch. & Sint. *Ex* Boiss.)		
Turkish fir	*Abies bornmuelleriana* Mattf.		
Coast live oak	*Quercus agrifolia* Née	*P. ramorum* Werres, de Cock & Man in't Veld	Dodd et al. 2005

conditions (Dick et al. 2006; Hansen et al. 2003; Green et al. 2013).

This study aimed to provide the first estimates of heritabilities from a clones-within-families genetics trial in order to determine whether tree breeding would help to mitigate the long-term effects of red needle cast disease on radiata pine in New Zealand.

Materials and methods

Trial design and establishment

One clones-within-families genetics trial was assessed at Wharerata forest on the east coast of New Zealand's North Island (38°55'11.43"S 177°50'42.99"E).

The trial design comprised 15 clones from each of 100 families, with up to 6 ramets per clone. The trial was an incomplete block design, with 4 replicates and 32 incomplete blocks per replicate (128 blocks). The trial was established to test a wide range of families across the New Zealand radiata pine breeding population (Dungey et al. 2009). Tolerance to Dothistroma needle blight or other needle diseases was not considered at selection.

Allocation of genotypes to incomplete blocks was undertaken using software package ALPHA + V.2.3 (CSIRO Australia and Biometrics & Statistics Scotland). Incomplete blocks were 24 m × 24 m (6 trees × 8 rows) and genotypes were planted randomly in a single-tree plot layout within incomplete blocks. Initial stocking was 833 stems per hectare, equating to tree spacing of 4 m × 3 m.

Cuttings were set in 2004 from stool-beds established from seed sown in 2002. Three controls were incorporated: GF14 seedlot 94/10; GF19 seedlot 03/652; and GF25 comprising an equal mix of seedlots 99/62, 99/378, 99/188, 97/67 and 99/318.

Sites were surveyed and pegged during September 2005. Plants were lifted between 14/9/05 to 16/9/05. Planting was carried out from 20/9/2005 to 22/9/2005.

Assessment

The trial was inspected for infection by RNC when the trees were six years of age from planting, in September 2011.

One week after inspection, the trial was subsequently assessed for two traits relating to damage to the crown caused by red needle cast. These were:

RNC – the percentage of crown that was clearly affected by red needle cast and distinguishable from other needle diseases such as Dothistroma needle blight (Carson 1989) and Cyclaneusma needle cast (Beets 1997; Dungey et al. 2006). This assessment was, therefore, based on the needles that were alive at that time.

NC – the percentage of crown that was lost overall due to needle cast, where the cause (i.e. type of disease) was no longer discernible. This assessment, therefore, included dead needles.

It is important to note that this trial had not yet reached crown closure, so this did not affect the scores. The trial was not assessed for growth rate, and there were no previous growth data available.

Analysis

Analysis was undertaken using ASReml software (Gilmour et al. 2010) using a univariate model of the following form:

$$\mathbf{y} = \mathbf{Xb} + \mathbf{Zu} + \mathbf{e} \quad (1)$$

where **y** is a vector of individual tree observations for a trait, **b** is a vector of fixed effects, **u** is a vector of random effects, **e** is a vector of random residuals, and **X** and **Z** correspond to design matrices relating the observations in **y** to the fixed and random effects in **b** and **u**, respectively. The joint distribution of the random terms was assumed to be multivariate normal, with means and (co)variances defined as:

$$\begin{bmatrix} \mathbf{u} \\ \mathbf{e} \end{bmatrix} \sim N\left(\begin{bmatrix} 0 \\ 0 \end{bmatrix}, \begin{bmatrix} \mathbf{G} & 0 \\ 0 & \mathbf{R} \end{bmatrix} \right) \quad (2)$$

where **0** is a null matrix, and **G** and **R** are (co)variance matrices for effects in **u** and **e**, respectively.

Fixed terms in vector **b** included the overall mean, mu, and a factor with two levels to account for the effects of control seedlots versus pedigreed (genetic) material. Random terms in vector **u** included additive and non-additive genetic effects of individual genotypes within the 'genetic' material, the effects of replicates and the effects of incomplete blocks within replicates. All the effects in **u** were assumed to be mutually independent. Missing values were also fitted as a fixed effect.

The error process in vector **e** was partitioned into spatially correlated (ξ) and uncorrelated (η) residuals. The spatially correlated error (ξ) was modelled using a first-order separable autoregressive process in the row and column directions, as suggested by Gilmour et al. (1997) for agricultural trials, as well as by Costa e Silva et al. (2001), Dungey et al. (2012) and Dutkowski et al. (2002) for forest genetic trials.

Significant differences between the fit of models (spatial and non-spatial) were estimated using a two-tailed likelihood ratio test (LRT) by comparing twice the difference between the log likelihood of the two models against the chi-square distribution with two degrees of freedom.

Individual narrow-sense heritability ($\hat{h}^2$) was estimated as the additive genetic variance divided by the sum of the additive genetic variance and the error variance. The clonal repeatability ($\hat{H}^2$) was estimated as the sum of the additive and non-additive genetic variance divided by the sum of the additive, non-additive and the error variance. Dominance variance ($\hat{d}^2$) was estimated as the non-additive variance divided by the sum of the additive, non-additive and the error variance.

The percentage of genetic gain available was estimated as the difference between the average of the top selections (20 or 100) and the average of the population, divided by the average of the population, multiplied by 100. Gains for clonal deployment assumed direct deployment of selections and that juvenile material was available for this to occur. Gains based on open-pollination assumed equal representation of selections established in grafted clonal seed orchards and that pollen contributions were unknown (i.e. gains were multiplied by 0.5). All gains were estimated using predicted breeding values of individual traits (i.e. RNC or NC) using spatial analyses.

Findings

There was a high level of infection in the area and a high level of infection throughout the trial. However, there was some variation among the experimental blocks, with some blocks higher up the slope having higher infection levels than those further down the slope.

Basic statistics

The mean of infection or damage levels for RNC and NC were remarkably similar (Table 2). There was a large amount of variation, with a coefficient of variation (CV) for RNC of 86% and for NC of 72%.

Variance components, heritability and breeding values

Needle loss due to red needle cast was found to be moderately heritable ($\widehat{h}^2$ 0.21–0.31). Fitting an additional spatial component to the residuals increased the estimates of additive genetic variance and heritability for both traits (Table 3). Clonal repeatability estimates ($\widehat{H}^2$) were between 0.23 and 0.59 (Table 3), indicating that selecting and deploying tolerant clones will give additional gains.

The spatial model also increased the estimate of dominance ($\widehat{d}^2$, Table 3) from a negligible range to a moderate one (0.31-0.41) with spatial partitioning of the residual variance. This was not what was expected, and may mean that clonal selection and deployment will deliver the best gains. Again, more evidence will be needed before embarking on such a strategy.

The spatial model was a significantly better fit to the data for both the traits, RNC and NC ($P < 0.0001$, LRT). Fitting a spatial component to the error variance, as we have done here, has been found to give better fitting models for many forestry trials and traits than when modelling only replicate and block effects (Dutkowski et al. 2002, 2006; Dungey et al. 2012). Modelling spatial

Table 2 Basic statistics for percentage needle loss for red needle cast (RNC) and for needle cast (NC) estimated from individual trees at the clones-within-families trial

Variable	Maximum (%)	Mean (%)	Minimum (%)	SE	CV (%)
RNC	95	18.4	0	0.25	86.0
NC	90	18.7	0	0.22	71.6

Table 3 Variance component estimates, narrow sense heritability and clonal repeatability estimates for RNC and NC

Trait	Variance component	Non-spatial estimate	Spatial estimate
RNC	Replicate	30.6	59.1
	Replicate.Block	13.3	32.7
	Spatial residual variance (units)	-	385
	AR1 (row)	-	0.75
	AR1 (column)	-	0.75
	Additive variance	47.2	54.6
	Non-additive variance	5.19	49.8
	Residual	171	72.4
	Log Likelihood	-3177.29	-3139.51
	Narrow-sense heritability $\hat{h}^2$	0.21 ± 0.04	0.31 ± 0.04
	Clonal repeatability $\hat{H}^2$	0.23 ± 0.02	0.59 ± 0.02
	Dominance $\hat{d}^2$	0.02 ± 0.03	0.41 ± 0.04
NC	Replicate	4.60	20.3
	Replicate.Block	16.2	29.4
	Spatial residual variance (units)	-	275
	AR1 (row)	-	0.77
	AR1 (column)	-	0.76
	Additive variance	39.6	38.6
	Non-additive variance	0.00	33.8
	Residual	125	50.3
	Log Likelihood	-11636.0	-11599.9
	Narrow-sense heritability $\hat{h}^2$	0.24 ± 0.02	0.31 ± 0.04
	Clonal repeatability $\hat{H}^2$	0.24 ± 0.02	0.59 ± 0.02
	Dominance $\hat{d}^2$	0.00 ± 0.00	0.42 ± 0.04

error variance has not been found to be useful for traits that do not have strong auto-correlated spatial structures, for example stem counts, form or branching in various species (*Eucalyptus globulus* Labill., *Picea abies* (L.) Karst., *Picea mariana* (Mill.) Britton, *Pinus pinaster* Aiton, *Pinus radiata, Picea sitchensis* (Bong.) Carrière) and cypress canker in C*upressus lusitanica* (Dungey et al. 2012).

Breeding values were estimated for individual trees and their parents. From these breeding values, it was clear that there were no individual tree breeding values that had very little damage resulting from the disease. This was in contradiction to the fact that assessment did find trees with very little damage (minimum of 0%, Table 2), but is likely to be a direct result of the BLUP methodology shrinking predictions towards the mean, particularly where there are only a few individual trees in this category. Clear differences were, however, evident between the worst affected (~40% damage) and the least affected trees (~7% damage; data not shown).

The heritability estimated here for red needle cast was comparable with heritability estimates for other diseases on radiata pine. These include examples of Dothistroma needle blight in New Zealand (0.17-0.40; Carson 1989; and Wilcox 1982 family-mean heritability of 0.32). Dothistroma needle blight heritabilities in Australia have ranged from non-significant to 0.69 (median 0.35); (Ivković et al. 2010). Cyclaneusma needle cast heritabilties (0.44-0.68; from Beets (1997)) were slightly higher than the RNC heritabilities estimated here although estimates of individual narrow-sense heritabilities vary considerably between trial sites and are most frequently between 0.1 and 0.4 (Dungey et al. 2006).

Repeatable heritability estimates rely not only on a robust genetic trial, but on a level of infection that ensures differentiation between tolerant or resistant and susceptible genotypes (Dungey et al. 2006). Differences between sites can be due to genotype x environment interaction, real differences in infection levels and different stages of infection. The moderate-to-high infection rates in the

well-designed clones-in-families trial assessed here should provide a reasonably robust heritability estimate. Long term, however, infection trials to screen a large number of genotypes under controlled conditions will provide the most reliable method of repeating damage assessments.

Genetic gain

Gains of up to 48% were estimated from the top 20 selections using breeding values for RNC, and 39% for the top 100 assuming clonal deployment. Gains for NC were not as large (20-25% for the top 100 or 20 selections), again assuming clonal deployment. Gains assuming clonal deployment with selections limited to only one clone per family gave 40.5% for RNC and 25% for NC for the top 20 selections, and 27% and 16% for the top 100 selections respectively. Gains from open-pollination within a clonal seed orchard were half those where direct deployment was possible from selected clones.

Even when obtaining planting stock from open-pollination in a clonal seed orchard, these gains appear to be very good when compared with predicted gains for Dothistroma needle cast in the Dothistroma Resistant breed in New Zealand (6-7%; H. S. Dungey unpublished data) and other estimates in Australia. Using selection criteria, Ivković et al. (2010) predicted that damage caused by *Dothistroma septosporum* (Dorog.) M. Morelet may be reduced by up to 7.7% while Ades and Simpson (1990) estimated that selecting the best 10% of radiata pine clones would reduce infection levels by 12%.

The figures obtained here for predicted gain are high compared with the estimates for needle loss from Dothistroma needle cast, outlined above. This result is encouraging, and implies that breeding will be a useful tool to mitigate future damage to New Zealand *P. radiata* forests.

Long-term tolerance to *Phytophthora* spp.

This paper represents only one component of the approach we are taking to mitigate the effects of red needle cast. We intend to select for tolerance within a broad-based genetic collection within the current New Zealand radiata pine breeding population (Nari Williams pers. comm.). We will also be investigating the diversity and genetics of the pathogen (personal communication R. McDougal 2014) and taking a systems-biology approach to identify tolerant germplasm. Multiple approaches will be used, including field and *in vitro* screening, screening for several *Phytophthora* species and across several tree species. Putative resistant and susceptible trees will be genotyped in an attempt to identify genes, gene families or alleles that confer broad-spectrum tolerance to these pathogens in the host. In this way we hope to reduce the possibility of any tolerance being overcome.

Conclusions

Tolerance to needle loss due to red needle cast is heritable ($\hat{h}^2$ 0.21-0.31) and selection for this trait will result in improved tree health. These genetic parameters are the first estimated for this trait in New Zealand, from one site at one time. These results must, therefore, be treated with caution, as rank changes with more information across more sites may occur. Nevertheless, genetic gain estimates imply that significant progress can be made through selection, up to 48%. Breeding for tolerance to *Phytophthora pluvialis,* therefore, appears to be an appropriate response to mitigate the effects of the introduction of RNC in New Zealand forests.

Future plans for this research include confirming these results with further field assessments and through future RNC inoculations under more controlled environmental conditions.

Competing interests

The authors declare that they have no competing interests.

Authors' contributions

HD wrote the paper and analysed the data, NW helped revise the manuscript and greatly improved it, CL helped with data checking and preliminary analysis, GS lead the field team in obtaining the data, helped devise the assessment methodology and helped revise the manuscript. All authors read and approved the final manuscript.

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