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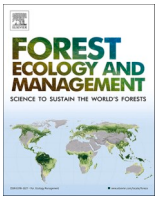

The inheritance of resistance to Scots pine blister rust in *Pinus sylvestris*

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ABSTRACT

Scots pine blister rust is a rust fungal pathogen that has become more noticeable in recent years and has increased in recorded incidence in Northern Scandinavia. This has prompted an initiation of resistance breeding to the fungus in the Northern Swedish pine breeding program. To accomplish this, it is necessary to evaluate the breeding potential for increased resistance and putative impact on other breeding objective traits that may have genetic correlations to resistance. To assess the potential of the trait to be included in the breeding program we examined half sibling families in four trials of which two had high incidence of Scots pine blister rust in a range of 25.4–42.1 %. We assessed vitality and height in one year and rust lesion frequency at two later time points. We found that rust resistance had a narrow sense heritability of 0.36–0.41, while vitality reached 0.17 and height 0.25. We found a high genetic correlation between sites in rust resistance and no genetic correlation between rust resistance and either vitality or height. This means that breeding for increased resistance in Scots pine can be done effectively without risking a negative impact on established breeding objective traits and that resistance is stable across environments.

1. Introduction

Blister rust on Scots pine (*Pinus sylvestris* L.) is caused by *Cronartium pini* (Willdenow) Jørstad, which until recently was considered as two closely related rust fungi *Cronartium flaccidum* (Alb. & Schwein) G. Winter and *Peridermium pini* (Pers.) Lév. (Hantula et al., 2002; Samils et al., 2011). Common names for the disease are Scots pine blister rust (abbreviated SPBR) or resin-top disease. The pathogen can be found on two-needled pines (Subgenus *Pinus*, Section *Pinus* like *P. tabuliformis, P. halapensis,* and *P. nigra.* see Samils and Stenlid, 2022 for references) in Europe and parts of Asia in the northern Hemisphere (CABI/EPPO, 1989). *C. pini* has two different forms which corresponds to the previous species names, a host-altering heteroecious form, where plants of *Melampyrum* spp. have been recognized as important alternate hosts in northern Fennoscandia (Kaitera et al., 2005), whereas the other form is an autoecious form that do not need an alternate host and where infection spreads between trees in a pine stand, (see Samils and Stenlid, 2022 for review). The fungus infects trees mainly through the stomata on the current year shoot. The mycelium of the parasitic fungus then grows in the phloem of infected trees and spreads both horizontally and vertically along branches and stems, eventually strangling the vascular transport and thereby killing the tissue above the lesion. Infected young seedlings generally die whereas older trees can withstand the fungus over several years, resulting in vertical wounds on the stem and reduction in stem volume production (Martinsson and Nilsson, 1987; Kaitera et al., 1994). The disease has historically been managed by excluding infected trees as seed trees in artificial seed tree regenerations. One significant difference between rust fungi and many other pathogens is that rust fungi also infect healthy, vigorously growing plants, because, as biotrophic parasitic fungi, they depend on the hosts continual production of nutrients (Callan and Carris, 2004). For comparison, another, non-biotrophic parasitic fungus which attacks conifers, *Gremmeniella abietina*, is known to have an increased incidence in stressed stands of *P. sylvestris*, e.g. densely planted or with unsuitable provenance materials (Niemelä et al., 1992; Romeralo et al., 2023). As stands of *Pinus sylvestris* ages in cold northern areas the yearly mortality rate decreases because most of the stressors only affect young seedlings that subsequently die-off (Persson 1994, Persson et al., 2010), but are still susceptible to SPBR.

In the beginning of the twenty-first century a widespread outbreak of Scots pine blister rust in Scots pine regenerations across large areas in northern Sweden were reported. According to Wulff et al. (2012) the

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total area of infected young pine forest stands in northern Sweden was estimated at 130,000 ha and on 33,000 ha of this area more than 10 % of the pine trees were infected. It has been shown that it is the heteroecious form that dominates the area (Samils et al., 2011). However, severe outbreaks of Scots pine blister rust in northern Fennoscandia are not a new phenomenon and there are reports from the beginning of the 20th century (Lagerberg, 1912).

In an inoculation experiment, comprised of eight northern Finnish Scots pine populations (originating from 66°30' to 68°00' N), Kaitera (2003) examined the susceptibility and lesion development in Scots pine samplings infected with *C. pini*, but found no significant variation in disease resistance between the populations. However, genetic variance and parameter estimates for Scots pine blister rust resistance among and within naturally infected Scots pine populations has to our knowledge not been quantified. On the contrary, for the related, host-alternating, and endemic rust fungus *Cronartium quercuum* (Berk.) Miyabe ex Shirai f.sp. *fusiforme*, there is verified large and significant family variation in rust resistance on *Pinus taeda* L. and *Pinus elliottii* Engelm. var. *elliottii* (the two main economically important hosts in the southeastern United States) in both field (Hodge et al., 1993; Dieters et al., 1996; McKeand et al., 1999) and green house inoculation experiments (Isik et al., 2008; Quesada et al., 2014). In addition, there appears to be low levels of interactions between genotypes and environment in host-resistance among *P. taeda* and *P. elliottii* field experiments (Hodge et al., 1993; Dieters et al., 1996; McKeand et al., 2003).

The resistance to *C. quercuum* on *P. taeda* has previously been assumed to be polygenic inherited and commonly analyzed using quantitative inheritance models. However, it has been shown that the genetic basis of tree resistance cannot be explained exclusively by polygenic models. Instead, the disease resistance is assumed to involve major genes in the host (Wilcox et al., 1996; Li et al., 2006; Huber and Amerson, 2011) and gene-for-gene interactions between host resistance and pathogen virulence (Kubisiak et al., 2011). Li et al. (2006) demonstrated, using Bayesian complex segregation analyses on *P. taeda* progeny data, that an ordinary polygenic model was applicable but that a mixed inheritance model that included polygenic effects and a few major effects was better at explaining the inheritance of rust resistance. This genetic architecture of resistance has been verified in two recent studies showing several major QTLs and markers associated with resistance (Ence et al., 2021; Lauer and Isik, 2021).

In an effort to improve disease resistance at North Carolina State University – Tree Improvement Program, the identification and roguing of susceptible *P. taeda* seed trees and the deployment of families with enhanced resistance to *C. quercuum* has been a successful endeavor (Schmidt, 2003). Similarly, considerable genetic gain have been achieved in *P. elliottii* in the resistance breeding against *C. quercuum* infections (Hodge et al., 1990). Although we expect that *C. pini* resistance will have similar inheritance and weak genetic correlation patterns to other important traits in Scots pine as do resistance to *C. quercuum* and *C. ribicola* we need to verify that is the case. Thus, before implementing selection for resistance to Scots pine blister rust in the current breeding programs of Scots pine of Northern Scandinavia, the genetic variance in resistance and genetic correlation of resistance with commonly used breeding objective traits (Persson and Seppo 2013) need to be quantified.

The objective of this study was to establish the proportion of heritable variation in resistance and estimate other genetic parameters for Scots pine blister rust resistance, including the genetic associations with survival and height growth using rust infection scores from Scots pine field trials located in northern Sweden.

2. Material and methods

2.1. Genetic material

The study was based on field data from four open pollinated half-sib progeny trials of Scots pine (*P. sylvestris*) plus-trees, F356 and F357 planted 1983 and F442 and F423 planted in 1985. Plus-trees are trees selected in forest stands that were tall, healthy, had a slender crown, and thin branches that were perpendicular to the trunk of the tree. The plus-trees are the founders of a tree improvement program. The trials are part of the northern Swedish tree improvement program at Skogforsk, the Forestry Research Institute of Sweden. The field assessments analyzed were tree vitality, tree height and susceptibility to infections of the pathogenic fungi Scots Pine blister rust (caused by either the heteroecious or autoecious form). The parent plus-trees (in total 373 trees) were selected from natural or artificially regenerated (generally with seed of local origin) forest stands, with geographical location ranging from 66°46' to 68°28' N in northern Sweden.

2.2. Experimental design

The progeny trials were planted with one-year-old seedlings randomized without restrictions according to a single-tree design (Wright and Freeland 1960; Loo-Dinkins and Tauer, 1987; Haapanen, 1992; Ericsson 1997). The seedlings were grown in cultivation cassettes in nursery, either from open-pollinated seed collected in the original forest stands (314 plus-trees) or from polycross seed produced by controlled crosses (59 plus-trees). The pollen mixture used for the polycross consisted of an equal proportion of pollen from 19 clones with average origin 66°50' N. Most of the 373 plus-trees were represented in all four field trials (Table 1).

2.3. Assessed traits

VIT: Reflects survival ability, tree vitality, and was scored visually for each individual tree in four ordered classes: healthy, slightly damaged, severely damaged but still alive, and dead (or missing). **HGT:** The height was measured on the living trees using a sectional wooden pool with markings. **RUST:** The number of lesions (both old and active) of Scots pine blister rust on stem and branches was counted on all trees that were alive in calendar year 2002. This was done visually by counting the number of lesions from several directions standing on the ground beside the trees. The age of shoot bearing the lesions (i.e., location of internode) was determined by counting the number of branch whorls from the top of the tree or the branch. The age of shoot bearing lesions was only noted on living trees and branches, and at most 3 lesion-bearing internodes per tree was recorded. Due to the substantial mean height in trial F422 (see Table 1) only lesions located on the stem were documented.

Because the trees were older, we did not analyze any other pathogens or pests on the trees as the frequencies of those damages were too small to evaluate. By the time the trees in cold northern site have reached the age of the trees in this study, most trees that suffered from other pests and fungi have usually been eliminated.

The field trials were monitored between the years 2002–2011 and the total age of the trees at the time of measurement ranged from 19 to 30 years. VIT, HGT and RUST were assessed in four, three and two field trials, respectively. Trial information, descriptive statistics of measured traits and calendar year of the assessments are summarized in Table 1.

2.4. Statistical analyses

Before analyses, the environmental variation within each trial was handled by bringing together observations from adjacent single-tree plots into blocks (Ericsson 1997). Furthermore, the observations were clustered in two groups, depending on the origin of the pollen source: *i*) open-pollinated seed and *ii*) polycross seed. VIT and RUST were linearized separately within each block through a "normal score" transformation (Gianola and Norton, 1981). Thus, both VIT and RUST were considered complex traits related to underlying normally distributed liability variables.

Table 1
Summary of the progeny trials, the genetic material and descriptive statistics of measured traits included in the study.

								Calendar year of assessment[d]					
	Lat.	Long.	Alt.	Temp.	Site	No.	Total no.	2002 Field survival	2006 Mean height	2009 Stem rust	2011 Stem rust	Stem rust	Stem rust
Trial[a]	°´N	°´E	(m)	sum.[b]	index[c]	Plus-trees	progenies	(%)	(cm)	(%)	(%)	(%)	(%)
F356	67°3´	20°18´	490	514	16	373	9401	12.8	NA	0	NA	25.4	42.1
F357	66°23´	18°22´	560	496	14	373	9097	20.1	335	0	NA	NA	NA
F422	66°26´	23°18´	200	794	18	358	7244	55.7	532	0	33.0	36.0	NA
F423	67°36´	23°00´	315	628	16	359	8958	11.8	372	0	NA	NA	NA

[a] Trial F356 and F357 were planted 1983 while trial F442 and F423 were planted1985.
[b] Expected temperature sum in day-degrees, threshold temperature +5°C (Morén & Perttu 1994).
[c] Expected dominant height (m) at age 100 years (Hägglund and Lundmark, 1977).
[d] Field survival = the percent of trees alive (vitality classes 1–3); Mean height = least square mean values of tree height (not measured in trial F356); Stem rust = the percent of the trees recorded alive 2002 with lesions of Scots pine blister rust on the stems at the year of assessment. NA not assesed

The statistical analyses consisted of a multivariate analysis of assessed traits, handling the data from different sites and assessment years as separate traits to provide additive genetic and phenotypic variance estimates, and estimates of genetic correlation between the traits (applying an unstructured correlation model form). We also included the latitudinal origin of the plus-trees as a covariate in the modelling to investigate the influence of origin in the material.

The general linear mixed model equation used was [1] $\boldsymbol{y} = \boldsymbol{Xb} + \boldsymbol{Zf} + \boldsymbol{e}$,

with $\boldsymbol{y} = [y_1' \quad y_2' \ldots y_n']'$, $\boldsymbol{b} = [b_1' \quad b_2' \ldots b_n']'$, $\boldsymbol{f} = [f_1' \quad f_2' \ldots f_n']'$ and $\boldsymbol{e} = [e_1' \quad e_2' \ldots e_n']'$, where **y** is the observation vector of individual phenotypic values of trait 1 to n ($n = 11$, i.e. VIT, HGT and RUST data from 4, 3 and 2 trials, respectively, and for RUST 2 additional assessment years), and **b**, **f** and **e** are the corresponding vectors of the fixed effects (i.e. overall mean, block, pollen group and latitudinal origin), random family effects, and individual residual deviations, respectively. **X** and **Z** are the corresponding incidence matrices. The random effects were assumed to follow independent multivariate normal distributions with zero means and (co)variances $V\begin{bmatrix}\boldsymbol{f}\\ \boldsymbol{e}\end{bmatrix} = \begin{bmatrix}\boldsymbol{G}_f \otimes \boldsymbol{I} & \boldsymbol{0}\\ \boldsymbol{0} & \boldsymbol{R} \otimes \boldsymbol{I}\end{bmatrix}$, where **0** is a null matrix, **I** is an identity matrix, $\mathbf{G}_f = \{\sigma_{f_i f_j}\}$ and $\mathbf{R} = \{\sigma_{e_i e_j}\}$ are the family and residual variance-covariance matrices, respectively (i, $j = 1$ to trait n, denoting variance when $i = j$), and $\otimes$ is the Kronecker product. In **R**, all off-diagonal elements were assumed to be zero for combinations of traits that were measured in different trials.

The model was fitted with the software package ASReml (Gilmour *et al.* 2009), which uses an average information algorithm for residual maximum likelihood (REML) (co)variance component estimation (Gilmour *et al.* 1995). The variance estimates were constrained to be non-negative.

2.4.1. Genetic components

The estimated additive genetic and phenotypic variances, and individual-tree narrow-sense heritabilities were calculated as $\widehat{\sigma}_A^2 = 4\widehat{\sigma}_f^2$, $\widehat{\sigma}_P^2 = \widehat{\sigma}_f^2 + \widehat{\sigma}_e^2$ and $\widehat{h}^2 = \widehat{\sigma}_A^2/\widehat{\sigma}_P^2$, respectively. Standard error of h^2 were approximated by Taylor series expansion, based on the ASReml results. The additive genetic coefficients of variation (CV_A) were calculated from $CV_A = 100\sqrt{\widehat{\sigma}_A^2}/\overline{x}$ for HGT and $CV_A = 100\left(\left(\Phi(\sqrt{\widehat{\sigma}_A^2}) - 0.5\right)/0.5\right)$ for VIT and RUST at the 50 % incidence level, where $\overline{x}$ is the least square mean for tree height and Φ is the standard normal probability distribution function.

2.4.2. Correlations

The additive genetic correlations ($\hat{r}_A$) and their standard errors were estimated directly using ASReml in which $\mathbf{G}_f$ was specified in the form **DCD** with **D** (a diagonal matrix) containing the standard deviations and **C** (a symmetric matrix) the correlations. Additionally, a structure qualifier was applied to **C**, in which sixteen $\hat{r}_A$ between VIT and RUST and twelve $\hat{r}_A$ between HGT and RUST were combined into single correlation estimates, respectively (i.e., $16 + 12\ \hat{r}_A$ were reduced to $1 + 1$ combined estimates).

2.4.3. BLUPs

The BLUPs (Best Linear Unbiased Predictors) for the parent breeding values for VIT, HGT, and RUST were calculated as two times the derived model solution of the random family effects. We assume each parent contributes to half the family effect by transferring half of its genetic material to the offspring, therefore multiplying by two to get the parents breeding value. The breeding values were derived from a model identical to model [1], with the exclusion of the model parameter latitudinal origin. The BLUPs for RUST were thereafter transformed to 50 % incidence level following $100 * \Phi(\Phi^{-1}(0.5) + BLUP)$, where Φ is the standard normal probability distribution function.

3. Results

The survival rates in the field trials 2002 varied from 12.8 % to 55.7 %. The low survival rates in three of the four trials investigated were associated with a low temperature sum (Table 1). Tree height was recorded in three of the trials, with average trial heights 2002 in the range of 335–532 cm. At the first assessment year of 2002 there were 8130 trees still alive of the 34700 planted progenies across all four trials

No infections of Scots pine blister rust were observed in any of the trials assessed in 2002 but started to be visible in measurable frequencies in 2006 and 2009, varying from 25.4 % to 42.1 %, in two of the trials (Table 1). In trial F356, 307 an additional 201 trees to a total of 508 trees (both dead and alive) were recorded as infected with Scots pine blister rust in the year 2009 and 2011, respectively, with lesion counts per tree in the range 1–27 (Table 2). The average number of lesions per infected tree in trial F356 increased from 2.3 to 3.5 between year 2009 and 2011. The mortality rate since 2002 had increased markedly in trial F422 year 2006 and 2009, with around 50 % of the dead trees having visible lesions of Scots pine blister rust (Table 2). In total 1332 and 1452 trees were recorded infected in trial F422 year 2006 and 2009, respectively, with lesion counts per tree varying from 1 to 15. The average number of lesions per infected tree in trial F422 was 2.8 in both years.

The year in which lesion-bearing shoots were formed varied from 1987 to 2002 in trial F356 (recorded in 2009) and from 1989 to 1999 in trial F422 (recorded in 2006, Fig. 1). In trial F356, two clear peaks in number of lesion-bearing shoots could be recognized for the years 1996 and 1998, where 1998 coincided with the highest number of recorded lesion-bearing shoots in trial F422. Because trees were much taller in F422, it was more difficult to estimate the timing of stem lesions, thus the lower numbers in Fig. 1. It is also worth mentioning that even though no trace of the fungus could be observed in the trials in 2002, many trees

Table 2
The number of lesions per tree of Scots pine blister rust on stem and branches (RUST) tabulated over trial, calendar year of assessment and survival.

	F356				F422			
	2009		2011		2006		2009	
RUST	Dead	Alive	Dead	Alive	Dead	Alive	Dead	Alive
0	20	881	33	667	243	2460	347	2236
1	3	151	4	175	130	436	178	436
2	1	63	1	97	32	197	51	209
3	.	34	.	60	8	156	26	130
4	.	26	.	37	6	122	18	111
5	.	6	.	36	12	78	33	72
6	.	5	.	19	6	46	11	55
7	.	2	.	24	5	31	11	29
8	.	4	.	13	2	15	5	20
9	.	5	.	13	7	16	8	16
10	.	3	.	7	2	8	2	9
11	.	.	.	3	1	3	1	6
12	.	2	.	6	.	6	2	6
13	.	1	.	4	1	3	1	1
14	.	.	.	2	.	1	.	3
15	.	1	.	.	1	1	2	.
16	.	.	.	2	.	.	.	.
17	.	.	.	1	.	.	.	.
18	.	.	.	2	.	.	.	.
24	.	.	.	1	.	.	.	.
27	.	.	.	1	.	.	.	.
Sum	24	1184	38	1170	456	3579	696	3339

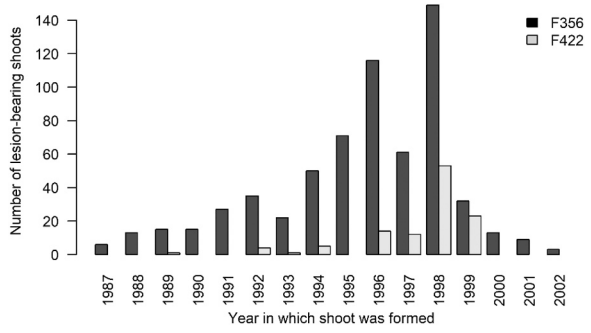

Fig. 1. Counts of shoot-formation-year for shoots bearing lesions in trial F356 and F422. Age of shoot was recorded calendar year 2006 and 2009 in trial F422 and F356, respectively.

at that time were in fact infected by Scots pine blister rust.

3.1. Genetic components

Derived narrow sense heritabilities varied from 0.12 to 0.17 for VIT, 0.12–0.25 for HGT, and 0.36–0.41 for RUST (Table 3). Standard errors for VIT and HGT were in the range 0.02–0.04 and varied from 0.05 to 0.11 for RUST. The $\widehat{h}^2$ for RUST decreased from 0.41 to 0.36 between year 2009 and 2011 in trial F356. The coefficient of additive genetic variance ranged between 11.1 % and 28.6 % for VIT and HGT, and 38.0–42.1 % for RUST. The CV_A estimates for RUST had increased by around 2 % at the later assessment in both trial F356 and F422.

3.2. Correlations

Derived inter-site genetic correlations for VIT and HGT, respectively (i.e. type B genetic correlations between environments), were generally positive with small standard errors and varied from 0.59 to 0.99 and 0.55–0.93, respectively (data not shown). Also, the inter- and intra-site $\hat{r}_A$ between VIT and HGT were all positive and significant, and ranged between 0.36 and 0.93.

Large variation was observed in sign, magnitude and significance for the $\hat{r}_A$ provided by the unstructured correlation matrix, both for the $\hat{r}_A$ between VIT and RUST as well as for the $\hat{r}_A$ between HGT and RUST, with generally non-significant values ranging between –0.35 and 0.26 (data not shown). The combined correlation estimates provided by the constrained correlation structure showed the same pattern as for the unstructured model, with derived non-significant values between VIT and RUST, and between HGT and RUST of –0.08±0.07 and 0.02±0.07, respectively, i.e., in both cases not significantly different from zero. The change in Log-likelihood between the unstructured and the constrained correlation structures were found to be non-significant based on a two-sided likelihood-ratio test (thereby verifying the usefulness of the combined $\hat{r}_A$).

The two inter-site $\hat{r}_A$ between the RUST assessments from trial F356 (year 2009 and 2011) and year 2006 in trial F422 (type B genetic cor-

Table 3
Estimates of narrow-sense individual heritabilities ($\widehat{h}^2$) with standard errors and additive genetic coefficients of variation (CV_A) in percent for evaluated traits (tabulated over year of assessment).

	VIT		HGT		RUST					
	2002		2002		2006		2009		2011	
Trial	$\widehat{h}^2$	CV_A[a]	$\widehat{h}^2$	CV_A	$\widehat{h}^2$	CV_A[a]	$\widehat{h}^2$	CV_A[a]	$\widehat{h}^2$	CV_A[a]
F356	0.12 ± 0.02	17.1	.	.	.	.	0.41 ± 0.11	38.0	0.36 ± 0.10	42.1
F357	0.17 ± 0.02	22.4	0.25 ± 0.04	28.4	.	.	.	.	.	.
F422	0.04 ± 0.02	13.7	0.12 ± 0.03	11.1	0.37 ± 0.05	39.0	0.39 ± 0.05	40.5	.	.
F423	0.17 ± 0.02	19.9	0.20 ± 0.03	28.6	.	.	.	.	.	.

[a] At 50 % incidence level.

relations) were computed to 0.58±0.14 and 0.55±0.14. Corresponding figures for the inter-site $\hat{r}_A$ between the RUST assessments in trial F356 (again year 2009 and 2011) and year 2009 in trial F422 were calculated to 0.86±0.14 and 0.85±0.14. The latter estimates were associated with the largest CV_A estimates (Table 3) and illustrated a low genotype by environmental interaction. The two intra-site $\hat{r}_A$ between assessment-years (age-age genetic correlations) for RUST in trial F356 and F422 were calculated to 0.91±0.07 and 0.99±0.00, respectively.

3.3. BLUPs

In trial F356 the estimated coefficients for the covariate latitudinal origin with RUST as dependent variables were computed to 0.136 ±0.082 and 0.225±0.092 in the year 2009 and 2011, respectively. Corresponding parameter estimates in trial F422 were 0.075±0.055 and 0.081±0.057 in the year 2006 and 2009, respectively. A positive parameter estimate indicates an increased liability to Scots pine blister rust for the most northern plus-trees included in the analysis, which was clearly significant in trial F356 year 2009. Still, a large variation in the BLUPs for RUST, expressed at 50 % incidence level, between trees with equal latitudinal origin could be observed in both trials (Fig. 2). To elucidate the plots in Fig. 2, a simple linear regression of the BLUPs against latitude of origin was performed, which followed the same pattern as the modelled covariate with significant and non-significant parameter estimates for the linear function fitted to the plotted data in trials F356 and F422, respectively (Fig. 2). In addition, the low r-square values of 0.034 (F356) and 0.007 (F422) reflect the large dispersion of the BLUPs illustrated in both trials. Similarly, we find no correlation between breeding values for RUST with HGT nor VIT while there is a strong correlation between HGT and VIT (Fig. 3).

4. Discussion

In this study, Scots pine blister rust scores from naturally infected trees were used, with average incidence levels ranging between 25–42 %. It is known that derived variance and correlation estimates may be dependent on the mean incidence in the studied material (Gianola and Norton, 1981). The average infection levels in our study were all within the span ~20–80 % mean rust incidence that has been shown to provide reasonable reliable estimates (e.g. Dieters et al., 1996). Fresh aecia do appear in 3–20-year-old shoots, although most commonly in shoots at the ages of 5–10 years (Kaitera, 2000). We found aecia in similarly aged shoots and like previous studies on *Cronartium pini,* find clear indication that infection waves have occurred (van der Kamp, 1988; Kaitera et al., 2000) (Fig. 1). It is however likely that older lesions are harder to detect and our count estimates of infection ages closer to 20 are downward biased. It should also be noted that the infections of SPBR were naturally occurring and that means that infection pressure likely varied within sites. However, the design using complete randomization with single tree plots should be able to account for that variation.

Derived heritability estimates for Scots pine blister rust resistance are consistent with field estimates for the related rust fungus *Cronartium quercuum* (Dieters et al., 1996; Isik et al., 2008). In both trials of the current study, the additive variance increased at the later assessment year. However, in trial F356 the heritability decreased, which can be attributed to an increased environmental variance and more unbalanced data after mortality rate increase in some families. Also, the generally high age-age correlation and low GxE interaction for Scots pine blister rust resistance are in accordance with Dieters et al. (1996) figures on *Pinus elliottii*, illustrating that variation in site conditions and age seems to be of little importance for blister rust resistance in pine.

Kaitera (2003) proposed that northern Scots pine provenances have the same resistance despite latitudinal origin. In our study we found a significant latitudinal trend of 8 percentage point increased rust incidence with each increase in latitude in trial F356, with less resistant northern populations (Fig. 2). One hypothesis to this pattern could be that the most northern populations have not developed the same resistance due to lack of alternate hosts in their original environment. However, the trend was low and not detected in trial F422 which includes the same families as F356, and most of the variation was observed within populations, which also has been shown to be the normal pattern for several fungal pathogens (Tack et al., 2012).

The general pattern for the $\hat{r}_A$ between VIT and RUST as well as for the genetic association between HGT and RUST, provided by both the unstructured and constrained correlation structures, was that they were weak and not significantly different from zero. Thus, it is reasonable to assume that selection of rust resistant trees for breeding will not affect survival and growth traits. However, in a clonally replicated test of *P. taeda* (Isik et al., 2003), both positive and negative significant genetic correlation between resistance to *C. quercuum* and growth traits on 4–6-year-old rooted cuttings was found. Comparing the breeding values for vitality, height and rust resistance among the tested 373 trees also show low correlation between rust and the other two traits while vitality and height covary extensively in these northern trials (Fig. 3). This implies that there could be weak detectable correlations in *Pinus sylvestris* once more data is analyzed, however not to a degree where it significantly impacts other breeding objectives.

In conclusion, the existence of substantial levels of additive variance and significant moderate inheritance in disease resistance to Scots pine blister rust in northern Scots pine populations has been verified. In addition, we find no evidence of a shared genetic component between

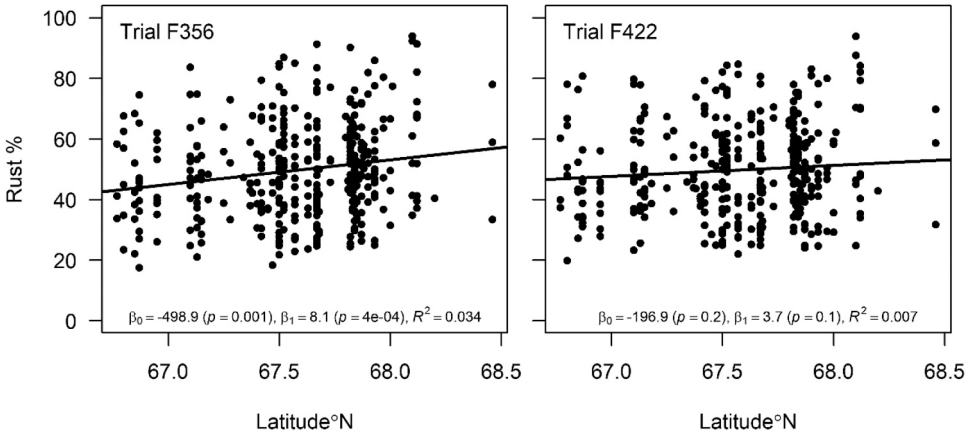

Fig. 2. Breeding values (BLUP's) for RUST (at 50 % incidence level) plotted over latitude of origin, for the 373 parent plus-trees studied in trial F356 and F422, year 2011 and 2009, respectively. The regression line and the parameter estimate for the linear function fitted to the plotted data are presented in the figures.

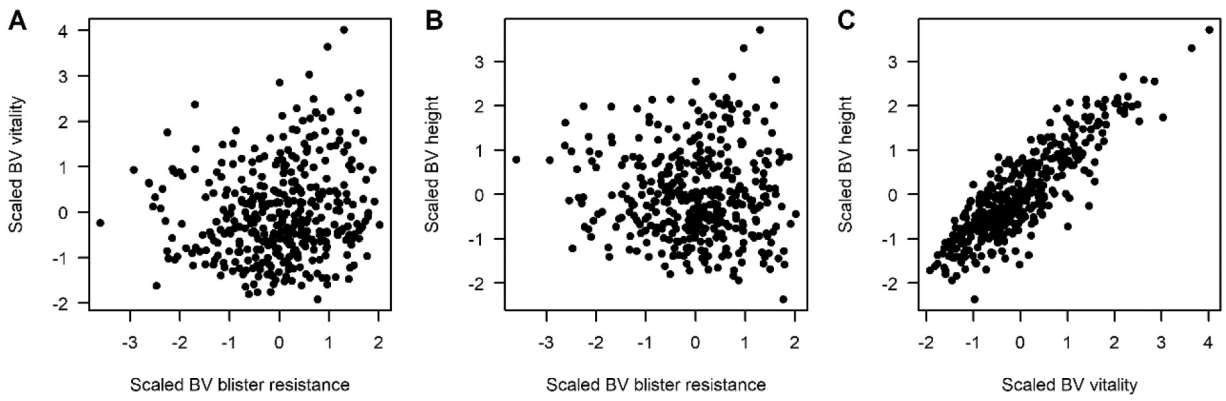

Fig. 3. Scatter plots of the scaled (to a unit variance) breeding values of the three traits examined and their relationship to each other. 0 equals the average breeding value and 1 is one standard deviation larger than the mean. **A)** Resistance to Scots pine blister (SPBR) rust vs. Vitality, **B)** Resistance to SPBR vs height and, **C)** Vitality vs height.

blister rust and field survival or tree growth. This conclusion is also strengthened by the independent assessment of vitality and height several years before detection of lesions in the trial. We find that implementing selection for resistance to Scots pine blister rust in current Swedish breeding programs for Scots pine could be performed without compromising genetic gain in other important breeding objective traits.

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CRediT authorship contribution statement

Torgny Persson: Writing – review & editing, Writing – original draft, Visualization, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Pia Barklund:** Writing – review & editing, Methodology, Conceptualization. **David Hall:** Writing – review & editing, Writing – original draft, Visualization. **Bengt Andersson Gull:** Writing – review & editing, Supervision, Methodology, Funding acquisition, Conceptualization. **Berit Samils:** Writing – review & editing, Methodology.

Declaration of Generative AI and AI-assisted technologies in the writing process

We did not use generative AI to write this manuscript.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data Availability

Data will be made available on request.

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