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ORIGINAL PAPER

Genetic parameters for wood quality traits and resistance to the pathogens *Heterobasidion parviporum* and *Endoconidiophora polonica* in a Norway spruce breeding population

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Abstract The root rot pathogens in Norway spruce (*Picea abies*) *Heterobasidion* ssp. cause substantial loss in carbon sequestered in forest and economic revenue for forest owners. To facilitate strategic breeding planning for increased resistance against this pathogen in particular, the blue stain fungus *Endoconidiophora polonica*, growth and wood quality traits (wood density and spiral grain), we estimated additive genetic parameters, correlations and the potential response from selection. Parameters were estimated from a progeny trial series established at two sites (25 years from planting) and their parents in a seed orchard (43 years from grafting). A standard half-sib analysis based on progenies and a parent–offspring regression was used for estimation of heritabilities. Resistance against the pathogens was measured as lesion length under bark after inoculations in phloem. Heritability values varied with site and estimation procedure from 0.06 to 0.33, whereas the phenotypic variance (as CV_P) is high and fairly stable around 40–50 %. Heritability values for wood density and spiral grain in the same material varied from 0.32 to 0.63. The highest heritability values were generally obtained from parent–offspring regression. There is no evidence of resistance traits being genetically correlated with growth or wood quality traits. Wood density is negatively correlated with stem diameter. Implications for breeding are discussed.

Keywords *Picea abies* · Tree breeding · Heritability · Resistance · *Heterobasidion* · *Endoconidiophora* · Wood density · Spiral grain

Introduction

Tree breeding programs for Norway spruce (*Picea abies* (L.) Karst.) that aim to increase the productivity and added value from forest have in order to increase the volume production per unit area traditionally emphasized on selection for climatic adaptation, growth and to some extent external stem quality. Improvement of the quality of the wood harvested at the end of a rotation period would improve the economic output further. One of the most significant sources of loss in biomass and quality is due to discoloration and degradation of the stem wood caused by root rots (*Heterobasidion* ssp.) (Woodward et al. 1998). Additional variation in inherent wood properties influences usability of the wood and the proportion of high and low grades (Zobel and van Buijtenen 1989). There is evidence of genetic variation in both resistance and several wood quality traits (summarized in: Eriksson 2010; Swedjemark et al. 2012), but the practical implementation of these traits in breeding programs has not yet been fully accomplished due to uncertainties about efficient screening methods and the genetic gain that can be obtained.

At present, production of seed in seed orchards containing selected genotypes is the most cost-effective way of delivering genetic gain from a Norway spruce breeding program. It is the additive proportion of the genetic variance that can be utilized in open pollinated seed orchards. A successful breeding strategy therefore depends on reliable estimates of genetic parameters such as additive genetic and phenotypic variances, the narrow-sense

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heritability (h^2) and the additive genetic correlations between traits.

In Norway spruce, estimates of these genetic parameters have been calculated for wood density and spiral grain which seems to be among those traits that have the highest genetic correlations with end user product grades (Högberg et al. 2014). The narrow-sense heritability estimates are within the range from 0.40 to 0.60 for density and from 0.30 to 0.50 for spiral grain. Density is generally negatively correlated with growth traits, while spiral grain is not (Costa e Silva et al. 2000a, b; Grans et al. 2009; Hallingbäck et al. 2008; Hannrup et al. 2004; Hylen 1997; Karlsson and Högberg 1998; Rozenberg and VandeSype 1996; Steffenrem et al. 2009).

Several studies have demonstrated the presence of genetic variation in resistance against *Heterobasidion* ssp. after stem inoculation (Arnerup et al. 2010; Skrøppa et al. 2015b; Swedjemark and Stenlid 1997; Swedjemark et al. 1997, 2001, 2007; Swedjemark and Karlsson 2004b), natural infections (Karlsson and Swedjemark 2006; Swedjemark and Karlsson 2004a) and spread from inoculated neighboring stumps (Wellendorf and Thomsen 2008). Genetic variation in response to stem inoculations has also been observed in Sitka spruce [*Picea sitchensis* (Bong) Carr.] (Bodles et al. 2007; Woodward et al. 2007). There is evidence for presence of additive genetic variation although the heritability is low ($h^2 \approx 0.10$) and estimated with large standard errors (Skrøppa et al. 2015b; Swedjemark and Karlsson 2004a; Wellendorf and Thomsen 2008). Another pathogen much studied is the bark beetle vectored blue stain fungus *Endoconidiophora polonica* (Siem.) Z.W. de Beer, T.A. Duong and M.J. Wingf. The genetic component to the variation in resistance against this pathogen appears to be stronger in general and the reported heritability is around 0.20 (Christiansen and Berryman 1995; Skrøppa et al. 2015a, b). Despite the many studies made, the necessary genetic parameters for strategic planning and inclusion of resistance in breeding programs have not been sufficiently estimated so far.

In forest genetic studies, it is most common to estimate the narrow-sense heritability from analyses of observations from siblings planted in common-garden experiments. Estimates are then based on intra-class correlations and the covariance between related individuals of the same generation, and have usually large standard errors. An unbiased estimate of the heritability can also be obtained from the regression between parents and offspring (Falconer and Mackay 1996; Zas and Sampedro 2015). Additional estimates of this parameter from the latter estimation method would indeed increase the reliability needed for strategic planning of breeding operations.

The aim of the present study was to (a) characterize the additive genetic variation in a Norway spruce breeding population for wood quality traits (wood density and spiral grain) and resistance after artificial inoculations with two pathogens, *E. polonica* and *Heterobasidion parviporum* (Niemelä and Korhonen), using sib-analysis of progenies together with parent–offspring regression, (b) estimate genetic correlations between resistance, growth and wood quality traits and (c) discuss the response from selection for implementation of resistance breeding in seed orchards.

Materials and methods

Inoculation treatments and measurements were made on progenies in two trials, "Braset" and "Jord," and on their grafted maternal parents in "Møystad" seed orchard. An overview of the traits measured and basic statistics are given in Table 1.

The seed orchard at Møystad (N60°48, E011°11, 180 m elevation) was established with grafted clones in 1967. The scions were collected from selected plus trees in natural stands in the lowland of southeast Norway based on growth, branching habits and stem form. In the seed orchard, the clones were organized in rows in two sections (Sections 1 and 2). Our study involves 38 clones, of which 13 were present in two or more rows, and five of these in both sections of the orchard. The remaining 25 clones were present only in one row.

The progeny trials Braset (N60°53, E011°12, 400 m elevation) and Jord (N59°38, E010°43, 95 m elevation) were established in 1985 with the same set of families from polycross matings made in Møystad seed orchard in 1983. The mating was done by mixing pollen from 17 clones (fathers) and applying the polymix to isolated female flowers of 112 other clones (mothers). As the male parent of each individual progeny is unknown, we assume that the material consists of 112 half-sib families. The trials were established with 2-year-old container grown seedling at a spacing of 2 by 2 m in randomized single-tree plots replicated in 30 blocks. Soils at both sites are considered to be highly fertile and the expected average production is 10–12 m^3 ha^{-1} $year^{-1}$ through the normal rotation time. In our study of resistance and wood quality traits, a sub sample of 50–53 families was used (Table 1). Thirty-eight of the mothers to these families were available for the study at Møystad seed orchard.

Measurements

Spiral grain angle (SG) in the 2–3 outermost growth rings at breast height was measured on standing trees at Braset and Møystad in 2011. SG is the tracheids deviation from the vertical axis in degrees. It was measured using an instrument developed at Chalmers institute, Sweden, and

Table 1 Overview of traits measured at Braset, Jord and Møystad (Sections 1 and 2) presenting total number of observations (N), grand mean (μ), minimum (min) and maximum (max) values, lowest and highest mean value for family (min fam and max fam) and clone (min clone and max clone), and number of families and clones included in material (N fam and N clones)

Trait	Year	Unit	Acron.	N	μ	Min	Max	Min fam	Max fam	N fam
Progeny trials										
Braset										
Diameter	2001	cm	D	1569	9.2	2.2	18.2	7.3	10.8	104
Height	2001	m	H	1603	6.4	0.6	9.3	5.1	7.3	104
E. polonica	2011	mm	EP	498	81	16	310	55	139	50
H. parvipor.	2011	mm	HP	498	48	15	249	31	73	50
Density	2009	kg m^{-3}	DENS	519	388	298	511	355	432	50
Spiral gr.	2011	degrees	SG	1101	1.57	−1.5	5.3	0.7	2.5	104
Jord										
Diameter	2001	cm	D	2077	10.4	4.1	20.1	8.8	12.4	112
Height	2001	m	H	2399	8	0.7	12.7	6.8	9.4	112
E. polonica	2011	mm	EP	568	65	14	310	41	95	53
H. parvipor.	2011	mm	HP	569	37	14	157	25	57	53
Density	2009	kg m^{-3}	DENS	597	407	312	560	378	438	53

Trait	Year	Unit	Acron.	N	μ	Min	Max	Min clone	Max clone	N clones
Møystad seed orchard (parents)										
Section 1										
Diameter	2011	cm	D	164	26	14	35	20	31	25
E. polonica	2011	mm	EP	131	52	19	181	25	110	25
H. parvipor.	2011	mm	HP	132	27	13	91	18	60	25
Density	2009	kg m^{-3}	DENS	164	392	302	543	334	451	25
Spiral gr.	2011	deg	SG	166	2.6	−0.9	5.9	1.2	4.6	25
Section 2										
Diameter	2011	cm	D	211	24	9	38	17	29	22
E. polonica	2011	mm	EP	114	60	20	225	34	173	22
H. parvipor.	2011	mm	HP	112	36	13	101	20	78	22
Density	2009	kg m^{-3}	DENS	211	393	326	575	343	432	22
Spiral gr.	2011	deg	SG	211	1.7	−1.4	4.9	0.1	3.4	22

the method is described and evaluated by Hannrup et al. (2003). Positive values indicate left oriented grain angle.

In 2010, 5.2-mm increment cores were collected at breast height, at north facing side of stem, at Braset, Jord and Møystad for determination of wood density (DENS). The cores were mounted in a cassette, acclimatized to 12 % relative humidity (RH) and scanned with a standard medical X-ray computer tomograph (Siemens Somaton Emotion single slice CT scanner with Syngo software) to obtain X-ray images. CT settings were: mAs = 150, Kv = 110, scantime = 1.0 s, filter = B50 s, slice width = 1 mm. The complete procedure is described in detail by Steffenrem et al. (2014). Wood density at 12 % RH was calculated from measured CT values as described by Steffenrem et al. (2009) and expressed in kg m^{-3}. The data were manually inspected and corrected for errors, and the individual tree values for wood density were calculated as a basal area weighted mean (Steffenrem et al. 2009) to obtain values representative for a stem disk. The ring from 2010 was not completely formed, and its values were therefore removed from the data set.

The trees in the progeny trials and grafts in the seed orchard were inoculated using our standard method (Skrøppa et al. 2015a, b). A 5- or 8-mm cork borer was used to remove the bark before insertion of fungal inoculum. The bark plug was then replaced. On each tree, three inoculations were done with *H. parviporum* (8 mm) and three with *E. polonica* (5 mm). The inoculations with *H. parviporum* were done on the east side of the trunk, while *E. polonica* were inoculated on the west side. The inoculations were done at breast height, and for each fungal species the inoculation spots were ca. 3 cm apart. On each side, the inoculation points were 2–3 cm from each other. The inoculation at Møystad was done June 27–28, 2011, at Jord July 3–7th, 2011 and at Braset July 9–11, 2011. Two and a half to three months after inoculation, the reaction

zones were made visible by peeling off the outer bark and the full lesion length at both sides of the inoculation point were measured. Values used in the analyses were means of the three inoculations, and the acronyms used through the text for the *traits* "lesion length after inoculation with *H. parviporum*" and "lesion length …. *E. polonica,*" are HP and EP, respectively. In total about 1720 inoculations with each of HP and EP were made at Braset and 1970 at Jord and 740 at Møystad. A low number of observations at Braset (23) and Jord (5) were discarded due to small cracks or wounds that caused extremely long lesions. However, never more than one of the three inoculations were affected and the mean of the two remaining was used further. The strains used in this experiment were *E. polonica* NFLI 93-208/115 and *H. parviporum* NFLI 87-257/1.

Tree height (H) and stem diameter at breast height (D) were measured on all living trees in the progeny trials in 2001. The survival at that time was 50 % at Braset and 70 % at Jord. Sample trees for inoculations, wood density and spiral grain measurements were all living trees in the 15 blocks with the highest survival. Hence, the data were unbalanced and the number of trees measured per family varied between 6 and 15 at each site with a mean of 11. Values from families with less than 6 observations at a given site were removed from the data. Between 5 and 14 ramets from each clone were included at Møystad depending on their presence in rows: 5–7 ramets were included for clones that were present in only one row and 7–14 ramets for clones present in two or more rows.

Statistical analyses

Plots of the residuals after preliminary analyses of variance of lesion lengths showed deviations from normal distributions and increase in value with increasing lesion lengths. Applying the Box-Cox method (in: Sakia 1992) to find a transformation to obtain normality showed that a "lambda" close to 0 gave the best distribution of the response variable which implies transforming by the natural logarithm: $y_t = \ln(y)$. Then variance estimation and hypothesis testing were performed on the transformed values for EP and HP according to the models given below. The variances were then back-transformed to original scale by the formula $\mathrm{Var}[Y] = \mathrm{e}^{2\mu} w(w-1)$, where $w = \mathrm{e}^{\sigma 2}$ (Proc GLIMMIX in: SAS Institute Inc. 2011) before they were used for parameter estimation.

Univariate analyses of variance were made by using the restricted maximum likelihood (REML) method in SAS PROC MIXED (SAS Institute Inc. 2003), and variances were estimated for the random effects in the models. Observations from each of the two progeny trials were analyzed by Model 1, and by Model 2 when the trials were analyzed together. Variances were presented with standard errors.

Model 1: $\gamma_{jk} = \mu + B_j + F_k + E_{jk}$

Here γ_{jk} is the observed value for the member of family k in block j, μ is the grand mean, B is the effect of block, F is the effect of family, and E_{kj} is the residual error. All terms, except μ, are considered to be random effects that are normal and independently distributed with mean = 0 and respective variances σ^2.

Model 2: $\gamma_{ijk} = \mu + S_i + B_{j(i)} + F_k + \mathrm{FS}_{ik} + E_{ijk}$

Here γ_{ijk} is the observed value for the member of family k in block j within site i. μ and F are the same effects as in Model 1, S is the effect of site, B is the effect of block within site, FS is the interaction between site and family, and E is the residual error. All terms, except μ and S, are considered to be random effects.

Data for the parents at Møystad were analyzed with two different models: Model 3, which is a mixed two-way analysis of variance considering the class variable section (T) as a fixed effect and clone (C) as a random effect. Model 4, which is a covariate analysis based on Model 3 but an additional continuous regression term (X_1) as fixed effect (covariate). The covariate was stem diameter (D) and the intention was to adjust for environmental variance not accounted for by the sections and rows in the original layout of the seed orchard.

Model 3: $\gamma_{ijn} = \mu + T_i + C_j + E_{ijn}$

Model 4: $\gamma_{ijn} = \mu + \beta D + T_i + C_j + E_{ijn}$

Here γ_{ijn} is the observed value for ramet n of clone (C) j in section of seed orchard (1 or 2) (T) i. μ is the grand mean and E_{ijn} is the residual error. βD in Model 4 is the regression term (stem diameter), β being the regression coefficient. All terms, except D, T and μ, are considered to be random effects (NID 0, σ^2). Twelve of the genotypes were replicated in two rows, one in three rows, and five were represented in both of the two "sections" of Møystad. The interaction between C and T was tested but found insignificant for all traits ($p > 0.10$) and not included in further analysis.

Phenotypic and genetic parameters from progeny trial data were estimated from variances for the respective effects in Models 1 and 2. Parameters estimated were the phenotypic variance ($\sigma^2_{\mathrm{P}} = \sigma^2_{\mathrm{F}} + \sigma^2_{\mathrm{FS}} + \sigma^2_{\mathrm{E}}$), the additive genetic variance ($\sigma^2_{\mathrm{A}} = 4\sigma^2_{\mathrm{F}}$), the narrow-sense heritability from half-sib analysis ($h^2_{\mathrm{hs}}=\sigma^2_{\mathrm{A}}/\sigma^2_{\mathrm{P}}$), coefficients of phenotypic variation ($\mathrm{CV}_P = 100(\sqrt{\sigma^2_{\mathrm{P}}}/\mu)$, additive genetic variation ($\mathrm{CV}_A = 100(\sqrt{\sigma^2_{\mathrm{A}}}/\mu)$, and the type-b genetic correlation $r_{\mathrm{b}} = \sigma^2_{\mathrm{F}}/\left(\sigma^2_{\mathrm{F}} + \sigma^2_{\mathrm{FS}}\right)$ (Yamada 1962). The

standard errors reported for h^2_{hs} and r_b were estimated from the Taylor expansion for variances of ratios (Lynch and Walsh 1998).

Narrow-sense heritability was estimated from parent–offspring regression analyses (h^2_{reg}) on basis of the linear regression model (Model 5): $y = \beta_0 + \beta_1 x + E$. Here, the regressor x is the clonal (female parent) mean and the response variable y is the family (progeny) mean. Both type of means were estimated as *least square means* (LS-means) from Models 1 to 4. LS-means for families were calculated across both sites for D, EP, HP and DENS, while only data from Braset were available for SG. The parameter h^2_{reg} was estimated as $2\beta_1$, with standard error from formula 10.6 in Falconer and Mackay (1996). All genetic effects in Models 1–4 were considered as fixed effects for estimation of LS-means.

Phenotypic (r_p) and genetic (r_g) correlations and their standard errors were estimated in multivariate analyses between pairs of traits in ASReml (Gilmour et al. 2009) from progeny trial data. The phenotypic correlations were estimated as $r_p = \left(\mathrm{cov}_E^{(12)} + \mathrm{cov}_F^{(12)}\right) / \sqrt{\left[\left(\sigma^{2\ (1)}_E + \sigma^{2\ (1)}_F\right)\left(\sigma^{2\ (2)}_E + \sigma^{2\ (2)}_F\right)\right]}$, where $\mathrm{cov}_E^{(12)}$ is the within-family (E) covariance between traits 1 and 2; $\mathrm{cov}_F^{(12)}$ is the family covariance between traits 1 and 2; $\sigma^{2\ (1)}_E$ and $\sigma^{2\ (2)}_E$ are the residual variance (E) for traits 1 and 2, respectively; $\sigma^{2\ (1)}_F$ and $\sigma^{2\ (2)}_F$ are the family variance for traits 1 and 2, respectively. Genetic correlations were estimated as $r_g = \left(\mathrm{cov}_F^{(12)}\right) / \sqrt{\left[\sigma^{2\ (1)}_F \cdot \sigma^{2\ (2)}_F\right]}$. All correlations were estimated across both sites, except those for SG that were estimated with data from Braset only.

Results

Variances and genetic parameters from the progeny trials

Estimates of parameters from the progeny trials are presented in Tables 2 and 3. All variance components for the family effect, except for D at Braset and EP at Jord, were two times or higher than their associated standard error (Table 2). They can therefore be considered as significant. The heritability estimates (h^2_{hs}) varied between sites, being higher at Braset for EP, HP and DENS. For SG, which was measured only at Braset, h^2_{hs} was estimated to 0.32. Analysis across two sites showed significant family components for all traits except EP (Table 3). The family by site interaction was considerable only for EP. Estimates of h^2_{hs} were 0.06 for EP, 0.14 for HP and 0.44 for DENS across sites. The coefficients of additive genetic (CV_A) and phenotypic (CV_P) variance for EP and HP were higher than those found for growth traits and DENS.

Parent–offspring regression

There was significant variation among the clones in seed orchard for all traits ($p < 0.001$, data not shown), and the clonal variance was in many cases similar or larger than the residual variances (Table 4). Estimates of h^2_{reg} from parent–offspring regression without covariate adjustment varied from zero for D, 0.16 for HP, 0.33 for EP, 0.54 for DENS and 0.63 for SG (Table 4, Mod. 3). All standard errors, except for D and HP, were lower than ½ the parameter estimates, suggesting significant additive genetic variance for EP, DENS and SG. The heritability for DENS increased

Table 2 Genetic parameters estimated for each progeny trial

Trait	σ^2_F	σ^2_E	σ^2_P	σ^2_A	h^2_{hs}	CV_A %	CV_P %
Braset							
D	0.10 (0.07)	5.66	5.76	0.38	0.07 (0.05)	7	26
H	0.04 (0.02)	1.6	1.64	0.14	0.09 (0.05)	6	20
EP	63 (36)	1675	1738	252	0.14 (0.12)	20	52
HP	27 (12)	389	416	109	0.26 (0.13)	22	43
DENS	141 (49)	926	1067	564	0.53 (0.17)	6	8
SG	0.082 (0.024)	0.94	1.02	0.328	0.32 (0.09)	38	67
Jord							
D	0.27 (0.08)	5.7	5.97	1.08	0.18 (0.05)	10	23
H	0.14 (0.04)	3.08	3.22	0.55	0.17 (0.05)	9	22
EP	17 (13)	783	799	67	0.08 (0.10)	13	44
HP	9 (5)	215	224	37	0.17 (0.11)	17	41
DENS	109 (44)	1225	1333	435	0.33 (0.13)	5	9

Standard errors of family variance components and heritabilities are given in parenthesis

Table 3 Genetic parameters estimated across both trials

Trait	σ^2_F	σ^2_{FS}	σ^2_E	σ^2_P	σ^2_A	h^2_{hs}(se)	CV_A %	CV_P %	R_b
D	0.14 (0.06)	0.06 (0.06)	5.7 (0.14)	0.54	5.87	0.09 (0.04)	7	25	0.68 (0.25)
H	0.08 (0.03)	0.01 (0.02)	2.5 (0.06)	0.33	2.6	0.13 (0.04)	8	22	0.86 (0.21)
EP	16 (16.4)	21 (20.06)	1099 (41)	65.4	1136	0.06 (0.08)	11	47	0.42 (0.45)
HP	10 (5.8)	4.8 (5.68)	272 (15)	41.0	287	0.14 (0.09)	15	41	0.68 (0.31)
DENS	134 (37)	0	1079 (48)	535	1213	0.44 (0.11)	6	9	1 (<0.01)

Standard errors of family and interaction variance components, heritabilities and the type-b genetic correlation between sites are given in parenthesis

Table 4 Parameters estimated from regression analyses between LS-means of parent and offspring

Trait	N fam	σ^2_{Clone}	σ^2_e	R^2	β_1	h^2_{reg}
Model 3						
D	38	642	1403	0	0.001	0 (0.08)
EP	38	0.102	0.070	0.16	0.165	0.33 (0.09)
HP	38	0.104	0.073	0.05	0.079	0.16 (0.10)
DENS	38	530	974	0.23	0.269	0.54 (0.12)
SG	38	0.729	1.016	0.38	0.315	0.63 (0.13)
Model 4						
EP	38	3.932	0.096	0.14	0.163	0.33 (0.09)
HP	38	3.322	0.100	0.06	0.081	0.16 (0.10)
DENS	38	392	284	0.27	0.375	0.75 (0.12)
SG	38	2.072	0.697	0.38	0.327	0.65 (0.13)

Number of families (N fam), clonal (σ^2_{Clone}) and residual (σ^2_e) variances for the parental clones in Møystad; coefficient of determination (R^2), regression coefficient (β_1) and narrow-sense heritability (h^2_{reg}) with standard error. Models 3 and 4 were two statistical models used to estimate LS-means for the parents (see description in materials and methods)

to 0.72 for when stem diameter (D) were used as covariate (Table 4, Mod. 4). The covariate did not have any effect on parameters for EP, HP and SG.

The two methods for estimating narrow-sense heritability produced the same results for HP (0.16), while for EP the estimate varied from 0.06 in the half-sib analysis to 0.33 in the parent–offspring regression. Estimates for DENS and SG were considerably higher when estimated with parent–offspring regression.

Correlation analysis

The genetic correlations were in many cases weak and were estimated with a considerable standard error that in most cases exceeded the parameter estimate (Table 5). The exception was the negative correlation between D and DENS (−0.65) and a strong positive correlation between D and H (0.86). Moderate correlations were found between D and EP (−0.41) and between HP and SG (−0.42). The genetic correlation between EP and HP was weak (0.30) and probably not significant.

Phenotypic correlations between growth traits and DENS were negative (<−0.30) (Table 5). Positive but weak phenotypic correlations were found between H and EP (0.35) and H and HP (0.24), although the genetic correlations between the same pair of traits were not significantly different from 0. The phenotypic correlation between HP and EP was 0.38.

Discussion

We made comparable estimates of the additive genetic variation and narrow-sense heritability using data from parents and offspring in a parent–offspring regression analysis, and data from the offspring only in an analysis of variance of the half-sib families. Only the latter method has been used in earlier studies of these traits. In order to estimate genetic parameters, there are two assumptions that should be discussed.

As the male parent of each individual offspring in a polycross is unknown, the first important assumption is that all progeny of a female parent are true half-sibs with an equal contribution of the male parents. Hence, the family variance (σ^2_F) estimates 1/4th of the additive genetic variance, and the average additive relatedness coefficient is 0.25. Departure from this assumption could result in upward biased genetic variances both from the analysis of the progeny trials (Tables 2 and 3) and the parent–offspring regression (Table 4) as relatedness would be underestimated. The broader consequence would be biased estimates of the response to selection. In open pollinated families, when the paternal contribution is unknown and highly unbalanced (each mother is likely to fertilized by a different set of fathers in addition to an unknown proportion of selfings) one often assumes a higher average relatedness and that the family variance estimate 1/3 of the additive genetic variance (e.g., Bridgwater 1992; Steffenrem et al. 2008). This is probably too conservative even for open pollinations since Gaspar et al. (2008) found an

Table 5 Genetic correlations (r_g) above diagonal and phenotypic correlation (r_e) below diagonal with standard errors in brackets

	H	D	DENS	SG	EP	HP
H		**0.86** (0.05)	−0.20 (0.23)	−0.23 (0.30)	−0.06 (0.35)	0.18 (0.26)
D	0.89 (<0.01)		**−0.65** (0.24)	−0.22 (0.34)	−0.41 (0.40)	−0.08 (0.31)
DENS	−0.32 (0.03)	−0.41 (0.03)		−0.15 (0.27)	0.11 (0.33)	−0.15 (0.24)
SG	0.09 (0.03)	0.14 (0.03)	−0.13 (0.05)		−0.25 (0.35)	**−0.42** (0.28)
EP	0.35 (0.03)	0.18 (0.03)	−0.14 (0.03)	−0.03 (0.05)		0.30 (0.32)
HP	0.24 (0.03)	0.09 (0.03)	−0.12 (0.03)	0	0.38 (0.03)	

All correlations are estimated across the two progeny trial sites Braset and Jord, except those involving SG that was estimated with data from Braset only

Genetic correlations similar or larger than two standard error are considered significantly larger than zero ($p < 0.05$) and indicated in bold

average relatedness coefficient of 0.26 from paternity analysis in maritime pine (*Pinus pinaster* Ait.) For the same species, Vidal et al. (2015) found the same value of the coefficient for polycross families. They observed that assuming true half-sibs instead of the actual pedigree gave nonsignificant overestimation of the heritability. Kumar et al. (2007) reported that GCA estimates obtained from polycrosses were similar to those from female-tester mating designs (true half-sibs). In their study, unequal paternal contribution was detected only in some of the 15 families. Since the larger deviations from equal paternal contribution have been detected when using only three or four pollen donors for radiata pine (*Pinus radiata* D. Don) (Moran and Griffin 1985) and Norway spruce (Skrøppa and Lindgren 1994), Kumar et al. suggested that polycrosses should make use of a larger number of pollen donors (>15) to decrease the level and effect of unequal paternal contribution. In our study 17 parents were involved as donors, and the polycross did not allow for self-pollinations. Calculations show that heritability estimates could be upwards biased by about 4–5 % if we assume an average relatedness of 0.26 instead of 0.25.

The second important assumption for the validity of parent–offspring regression for heritability estimation is that the variances are equal in the two sexes (Falconer and Mackay 1996). This has to be questioned since Skrøppa et al. (2015a, b) show that the genetic variation for both pathogens studied might be larger on the paternal side. Skrøppa et al. (2015a) discuss the possibilities for the presence of nonadditive genetic effects caused by some uniparental inheritance mechanism controlled by, i.e., paternally inherited chloroplasts. However, they also discuss the limitations with their own materials. The differences between the sexes could have been caused by sampling effects since very few parents were involved. However, also our data indicate presence of nonadditive genetic variation since the very strong clone variances at Møystad [equivalent to broad-sense heritabilities (H^2) of 0.60] contrasts the weak or moderate family variances in the progeny trials. These are, however, only indications without unconditional support from previous studies (Arnerup et al. 2010; Karlsson and Swedjemark 2006; Skrøppa et al. 2015a, b; Swedjemark et al. 1997; Swedjemark and Karlsson 2004a,b) as broad- and narrow-sense heritabilities vary within the same range (0–0.35).

EP was the only trait showing some family by site interaction (GxE) across the progeny trials Braset and Jord. The heritability estimates were also lower than expected from an earlier study (Skrøppa et al. 2015b). The interaction could be due to additive gene effects acting different in the two environments. Further, there are often sometimes poor correlations when same genetic materials are tested with different inoculums, at different test sites or in different years (Karlsson et al. 2008; Skrøppa and Solheim, unpubl. data). The seasonal growth rhythm stage at the time of inoculation, indicated by the phenology of apical meristems, is known to influence the results and could cause differences in observed variances and GxE interactions (Krokene et al. 2012; Skrøppa et al. 2015b; Swedjemark and Stenlid 1996). This vulnerability to environmental factors acting on the inoculum batch itself, or the trees tested, makes it important to design studies that include more than one test site, both parents and offspring, or different inoculum, to obtain an estimate of the repeatability of the results. Inoculations at Jord, Braset and Møystad were made in the end of June and early July when all trees were in stages of active shoot growth and the results should according to Skrøppa et al. (2015a) be little affected by variation in phenology.

The parental clones were planted in rows at Møystad seed orchard, and not all clones were replicated in more than one row. Unlike the progeny trials, Møystad is therefore not a well-designed genetic trial as genetic and environmental effects are confounded. We used stem diameter (D) as a covariate in Model 4 as a simplified approach to adjust for environmental variation in the seed orchard. However, the confounded effects of environment and genetics are difficult to correct for by any statistical

analyses, and genetic parameter estimates are vulnerable. We believe that the parameters estimated from the parent–offspring regression are more likely to be underestimated than overestimated since the confounding effects would introduce "noise" in the parental data. Anyway, its heritability estimates are made without the use of covariates that are interesting in terms of tree breeding.

Although, parent–offspring estimates of the narrow-sense heritability for resistance traits were generally higher than estimates from the sib-analysis, they were fairly well in line with those found in by Skrøppa et al. (2015b), taking the large standard errors into consideration. The general trend seems to be that resistance against *H. parviporum* and *E. polonica*, characterized by lesion lengths after inoculations, is under weak-to-moderate additive genetic control with narrow-sense heritability between 0.05 and 0.30. Wood density and spiral grain in Norway spruce are under much stronger additive genetic control as the reported heritability here and in literature seems to be quite consistent within the range of 0.30–0.60 for both traits (Costa e Silva et al. 2000a; Hallingbäck et al. 2008; Hannrup et al. 2004; Hylen 1997; Steffenrem et al. 2009).

Lesion lengths for EP and HP seem to be very weakly or not genetically correlated at all (Table 5), giving little hope of increased resistance against one of the pathogens when selecting for the other. This confirms the results in Skrøppa et al. (2015a, b), where they suggest that the genetic basis of phloem resistance differ between the two pathogens. Furthermore, a negative correlation between EP and D of −0.41, with a standard error of the same size, was the only correlation of any magnitude found between lesion lengths and growth or wood quality traits. This implies that breeding efforts for faster growth or higher wood quality are unlikely to affect phloem resistance. The negative genetic correlations between diameter growth and wood density confirm many earlier studies (Costa e Silva et al. 2000a; Grans et al. 2009; Hannrup et al. 2004; Hylen 1997; Karlsson and Högberg 1998; Rozenberg and VandeSype 1996; Steffenrem et al. 2009), emphasizing the importance of a balanced selection for growth and wood density (e.g., Lee 1999) in order to avoid reduced wood quality from intensive breeding.

Expected response from selection

Estimates and observations indicate that seed from the first-generation seed orchards for Norway spruce have given 10–15 % gain in volume production at those sites applied since the mid-1970s (summarized by: Haapanen et al. 2015, Jansson et al. submitted manuscr.). This is a result from the plus-tree selection for larger trees, with beneficial quality characteristics, and some other seed orchard effects (Haapanen et al. 2015). It is unlikely that the gain obtained for growth has had any implications on the resistance to root rot since these traits seem to be uncorrelated. Trees were in many cases felled during plus-tree selections. Those with visible decay or rot in the stump were discarded, but the effect of this selection is probably marginal since the natural infection rate, which is only 20–30 %, allows very low selection intensity.

A relevant scenario for implementation of these results and data into breeding is to select the best first-generation parents based on progeny testing (backward selection) for establishing tested seed orchards. The typical selection intensity in a Nordic breeding scenario would be 25 genotypes out of 250 tested (top 10 %). The expected response from this selection can be estimated from the functions in Rosvall et al. (2001) and Lindgren (2002) with genetic parameters from Table 3. Selection solely for height growth after 15 years (20 % of rotation time) yields an expected gain at rotation age of 70 years of 6 %. If we assume an increased site index by 6 % that will be equivalent to a 12–13 % increase in the volume at harvest or harvest of the same volume 9 years earlier. A similar estimation for HP and wood density, when selection is made after 25 years (one-third of rotation time), produces an expected response of 16 % (7 mm) decrease in lesion length or 6–7 % increase in wood density (26 kg/m^3).

Most Norway spruce breeding programs keep the candidate genotypes in breeding arboretums. The presence of parent–offspring relationships provided for the option is to rank the parents for wood density and spiral grain instead of waiting for results from progeny trials.

Height, diameter, volume and wood density are direct measurements of traits with strong effect on the value of the stand or forest property and can easily be prioritized as breeding targets. It is more difficult to translate the expected response in lesion length to the value of the forest stand. Even if Swedjemark et al. (2012) and Skrøppa et al. (2015b) state that the genetic variance is sufficient for breeding for higher resistance, it might be argued that taking advantage of full selection intensity to obtain maximum gain in growth is more effective in terms of added value than selection for reduced lesion length that has a more uncertain result for the end user. Another important factor in that respect is the questioned repeatability of inoculation studies (Swedjemark et al. 2012; Skrøppa et al. 2015a, b).

On average, in Norwegian Norway spruce stands, 27 % of the logs are affected by rot at harvest, and mainly 70–80 % of these due to *Heterobasidion* (Huse et al. 1994). *Heterobasidion* rot usually infects in roots and grows in the heart wood to 3–6 m above ground, and in extreme cases to 10–15 m above ground (Woodward et al. 1998). In a "normal stand," 10 % of the volume will be reduced from saw timber to pulp wood of half the value. The total value

of the stand will be then reduced by ca. 5 %. However, the frequency of rot varies from stand to stand, and more than 50 % of the trees may be affected in extreme cases (E. Loe, Allskog AS, pers. comm., 2015; Hietala et al. 2015). Then, 15 % of the value, or even more, is lost. In addition, since biomass is lost, also the value as pulp wood is reduced causing losses later in the chain of value. The stands are destabilized against wind throw which force earlier harvest with higher costs and lower benefits. The decay recycle the carbon sequestered in the stems back to the atmosphere (Hietala et al. 2015), at the same time as the wood becomes less suitable in products that has higher substitution effects when used instead of concrete and steel in constructions. In addition, it is expected that climate change causes a *Heterobasidion* ssp. spores to spread during a longer season and infects new trees (Lindner et al. 2010), and offers more favorable conditions for fungal growth (La Porta et al. 2008; Lindner et al. 2010; Müller et al. 2011). It can easily be argued that the economic loss of 5 % in "normal" stands can be accounted for by more effective selection for faster growth rather than for increased rot resistance. But the losses in wider sense, and under climate change projections, argue for stronger implementation of resistance in the breeding strategies.

Conclusions

There is additive genetic variation for phloem resistance to the two pathogens the blue stain fungus *E. polonica* and the root rot fungus *H. parviporum* measured as lesion length after inoculations in Norway spruce. Heritability values vary with site and estimation procedure from 0.06 to 0.33, whereas the coefficient of phenotypic variance is high and fairly stable around 40–50 %. The latter is important making solid potentials available for response from selection. Heritability values for wood density and spiral grain in the same material varied from 0.32 to 0.63. The highest values were obtained from parent—offspring regression. There is no evidence of resistance being genetically correlated with growth or wood quality traits. Wood density is negatively correlated with stem diameter.

Regardless, successful breeding for increased resistance requires better knowledge of the relationship between lesion length after inoculations, which is an effective method for screening of large breeding populations, and the actual resistance to the pathogens and reduced frequency or growth of rot.

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