Resistance to Ceratocystis Wilt (*Ceratocystis fimbriata*) in Parents and Progenies of *Eucalyptus grandis* x *E. urophylla*

By Carla Cristina Gonçalves Rosado[1)], Lúcio Mauro da Silva Guimarães[1)], Miranda Titon[1)], Douglas Lau[2)], Leonardo Rosse[3)], Marcos Deon Vilela de Resende[4)] and Acelino Couto Alfenas[1),*)]

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Abstract

Ceratocystis wilt, caused by *Ceratocystis fimbriata*, is one of the most damaging diseases in eucalyptus plantations worldwide. Although there are resistant genotypes, the genetic basis of resistance is still poorly understood. In this paper we studied the resistance level by a stem inoculation experiment of genotypes of *Eucalyptus grandis* and *E. urophylla* and estimated the heritability and gains of selection in families derived from controlled interspecific crosses. In both species, highly resistant as well as highly susceptible genotypes to Ceratocystis wilt were found. Out of 21 parents assessed, twelve were resistant and nine susceptible. Estimates of individual narrow (50%) and broad (59%) sense heritability suggested a high degree of genetic control and low allelic dominance of the trait. There was great genetic variation among and within families, a fact that contributes to high heritability and genetic gain. A genetic gain in lesion size of up to –74.4% was obtained from selection of the 50 best clones in the evaluated families, i.e., the mean lesion length in the progeny population can be reduced by 74,4%.

Key words: Genetic parameters, genetic resistance, forest pathology.

Introduction

Due to its lethal nature, broad range of hosts and wide geographical distribution, the Ceratocystis wilt caused by *Ceratocystis fimbriata* Ellis & Halsted is currently one of the most damaging diseases of eucalyptus plantations in Brazil (Alfenas et al., 2009). Besides eucalyptus, *C. fimbriata* also infects other crops of economic importance, such as mango (*Mangifera indica* L.) (Viegas, 1960), coffee (*Coffea arabica* L.) (Marin, 2003), black-wattle (*Acacia mearnsii* of Wild.) (Ribeiro and Ito, 1988; Santos and Ferreira, 2003), taro (*Colocasia esculenta*) (Harrington et al., 2005) and crotalaria (*Crotalaria juncea* L.) (Ribeiro et al., 1977). The main symptoms caused by *C. fimbriata* in eucalyptus are wilting, canker and wood darkening. The pathogen penetrates the plant through fresh wounds on the stem and root contact and attains the stem and trunk via medullar parenchyma. Dark stripes originates from the medulla spread through the radial parenchyma up to different heights and may reach in the stem. The fungus also kills partially of the vascular cambium, the phloem and pheloderm, resulting in longitudinal, continuous or interrupted lesions on the outer part of the trunk, which cause tree wilting and death (Ferreira et al., 2006).

Besides *C. fimbriata*, other species such as *C. eucalypti*, *C. moniliformis*, *C. moniliformopsis* and *C. pirilliformis* have been reported in eucalyptus (Barnes and Roux, 2003). Of these, only *C. fimbriata* is known to be pathogenic and was reported for the first time causing wilting and plant death of *Eucalyptus* spp. in the Southeast of the State of Bahia in 1997 (Ferreira et al., 1999). *Ceratocystis fimbriata* has also been reported in eucalyptus in Uruguay (Barnes et al., 2003), in the Republic of Congo (Roux et al., 2000), in Uganda (Roux et al., 2001) and in the South Africa (Roux et al., 2004).

Although genetic resistance is the best control strategy (Zauza et al., 2004), the inheritance pattern and mechanisms involved in resistance have not yet been determined. However this information is essential to guide the use of resistance sources in genetic breeding programs of eucalyptus. The purpose of this study was to determine the resistance level of *E. grandis* and *E. urophylla* genotypes and to estimate the possibility of resistance transferring through controlled interspecific crosses between these two species.

Material and Methods

Plant Material

The resistance to ceratocystis wilt of five elite genotypes of *E. grandis* (G39, G45, G58, G93 and G547) and 16 of *E. urophylla* (U1072, U1177, U1179, U1183, U1185, U1237, U1275, U1282, U1286, U1305, U1310, U1313, U1316, U1392, U1450, and U1455) was assessed. Segregation in 30 hybrid progenies derived from controlled *E. grandis* x *E. urophylla* crosses was also evaluated. Five replicates of each parent and 18 to 25 plants per progeny were inoculated, depending on the cross (*Table 2*). Not all parents of the evaluated progenies were inoculated, since they would not be propagated by rooted cuttings. However these progenies were used to estimate the genetic gains. The rooted cuttings

[1)] Departamento de Fitopatologia/BIOAGRO, Universidade Federal de Viçosa (UFV), 36570-000, Viçosa, MG, Brazil.

[2)] EMBRAPA Trigo, Rodovia BR 285, Km 294, 99001-970, Passo Fundo, RS, Brazil.

[3)] VERACEL S.A. Rodovia BA-275, Km 24, 45820-970, Eunápolis, BA, Brazil.

[4)] EMBRAPA Florestas, Estrada da Ribeira, Km 111, 83411-000, Colombo, PR, Brazil.

[*)] Corresponding author: Acelino Couto Alfenas. Telephone: +55 (31) 3899-2939. E-Mail: aalfenas@ufv.br.

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and seedlings (90 days old) were transplanted to 2L pots, containing a mixture of soil:sand:manure (3:1:1). Ouro Verde® fertilizer (15-15-20 NPK and low quantities of Ca, S, Mg, Zn, B, Fe, and Mn) was applied twice a week (100 mL/plant at 7,5 g/L) until the plants reached an adequate development stage for inoculation, 60 days after transplanting (plants of 1.5 to 2.0 cm diameter and 60 cm hight). Daily mean temperature in the period was of 25 ± 3 °C. Irrigation was conducted 2–3 times a day according to the plant needs.

Inoculation

In the experiment, it was used a culture medium (SBS-1) of *C. fimbriata*, isolated from plants of a hybrid clone *E. grandis* x *E. urophylla* with wilting symptoms, from the region of Teixeira of Freitas (BA). This isolate has been routinely used in our laboratory for screening for host resistance. The fungus was grown in Petri dishes (9 cm diameter) containing MYEA (2% malt extract, 0.2% yeast extract, and 2% agar) for eight days at 28 ± 1°C and 12 h photoperiod under a light intensity of 40 µmol. $s^{-1}.m^{-2}$. For the spore collection (conidia + ascospores), sterilized distilled water was added to the dishes and, after scraping off the colony surface with Drigalski spatula, the spore suspension was filtered through double gaze and calibrated at 2.5 x 10^6 spores mL^{-1} (Zauza et al., 2004). A cut lengthwise approximately 2 cm length was made on the stem (about 3 cm above soil surface) and 0.5 mL of the spore suspension was deposited and the inoculation area was covered with plastic film. Ten replicates of each of the clones HRC2277 (natural hybrid of *E. grandis* x *E. urophylla*) and HGU1172 (hybrid *E. grandis* x *E. urophylla*) were used as resistant and susceptible controls, respectively.

Data evaluation and analysis

The lesion lenght on the xylem was determined 60 days after inoculation and the data were analyzed using the programs Statistica® version 5.0 (StatSoft), Genes® version 2007.0.0 (Cruz, 1997) and Selegen-Reml/Blup® (Resende, 2002; Dunlop et al., 2005). The Scott & Knott grouping test ($p \leq 0.01$) was used to classify the parents as resistant or susceptible.

The phenotypic, genetic and environmental variances were estimated for each family, based on the procedure for families with intercalated controls (Cruz, 1997), the adjusted analysis model and parameters estimated by the methodology of mixed linear models (REML – residual maximum likelihood and BLUP – best linear unbiased prediction). Five replications of each resistant and susceptible clone were employed as intercalated control. The REML/BLUP method was fitted based on the following mixed model: $y = Xf + Zg + e$, where: y, f, g and e are vectors of data, of fixed effects (means of controls and the progeny population), genotypic effects of families and parents (random) and random errors, respectively, and X and Z: incidence matrices for f and g, respectively.

From the fitting of the model for the experiment with families and also for the experiment with parents, estimates of variance components (via REML) and the heritability in the narrow and broad sense were obtained. Genotypic values for each individual of the hybrid families were also predicted and gains with genetic selection estimated within families and also in the hybrid population as a whole. The prediction of gains with selection (GS) was based on the selection of the 50 best plants of the progeny population (F1).

The first cross between two distinct populations is not in equilibrium and its total genetic variance is not linearly related to any of the parent populations. However, total genetic variance can be partitioned into additive and dominance components as follows (Hallauer and Miranda, 1988; Gallais, 1989; Comstock, 1996):

$$\sigma^2_{A(12)} = pq[a + (r-s)d]^2 + rs[a + (q-p)d]^2 = \sigma^2_{A12} + \sigma^2_{A21}$$

$$\sigma^2_{D12} = 4p(1-p)r(1-r)d^2 = 4pqrsd^2, \text{ where:}$$

$\sigma^2_{A(12)}$: total additive genetic variance in the crossed population.

σ^2_{A12}: additive genetic variance when plants of the population 1 are used as female parents.

σ^2_{A21}: additive genetic variance when plants of the population 2 are used as female parents.

σ^2_{D12}: dominance genetic variance in the crossed population.

The quantities p and q are the allelic frequencies in the population 1 and r and s are the allelic frequencies in the population 2. The quantities a and d are the effects of the genotypes homozygous favorable and heterozygous, respectively.

The components $\sigma^2_{A(12)}$ and σ^2_{D12} are called homologous of the additive and dominance variances as defined for one population, i.e., σ^2_A and σ^2_D. In fact they equal σ^2_A and σ^2_D when p = r, i.e., when both populations have the same allelic frequency.

When the crossed population is in a full-sib family structure, the genetic variance among full sib (FS) families is given by

$$\sigma^2_{FS(12)} = (1/2)pq[a + (1-2r)d]^2 + (1/2)rs[a + (1-2p)d]^2 + pqrsd^2$$

$$\sigma^2_{FS(12)} = (1/2)\sigma^2_{A(12)} + (1/4)\sigma^2_{D12}$$

which is analogous to $\sigma^2_{FS} = (1/2)\sigma^2_A + (1/4)\sigma^2_D$, the variance among full-sib families for one population.

The quantity $\sigma^2_{A(12)}$ can be used as the numerator of an inter-population narrow sense heritability and the components $\sigma^2_{A(12)}$ and σ^2_{D12} can be used as the numerator of an inter-population broad sense heritability. Such components $\sigma^2_{A(12)}$ and σ^2_{D12} are called inter-population additive and dominance variance and are routinely estimated in maize breeding programs (Hallauer and Miranda, 1988; Coors and Pandey, 1999), without assuming alleles at frequency of 0.5. Selection of F1 hybrid individuals for cloning can be predicted by using this defined inter-population broad sense heritability. Estimates of the heritabilities on full-sib family means were also obtained associated with family selection.

Considering separated analysis for the full-sib experiment the following variance structures and relations are obtained:

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Genetic variance among full-sib familes:

$\sigma^2_{FS} = (1/2)\sigma^2_{A(12)} + (1/4)\sigma^2_{D12}$.

Full-sib family mean heritability:

$h^2_{FSM} = \sigma^2_{FS}/(\sigma^2_{FS} + \sigma^2_{WFS}/18)$,

where σ^2_{WFS} is the within family individual phenotypic variation.

Accuracy of family selection: $(h^2_{FSM})^{1/2}$.

Coefficient of genotypic variation among progenies:
$CVg\% = 100 * (\sigma^2_{FS})^{1/2} / (GeneralMean)$

Coefficient of residual variation:
$CVe\% = 100 * (\sigma^2_{WFS})^{1/2} / (GeneralMean)$

Within full-sib family individual broad sense heritability: $h^2_{WSF} = \sigma^2_{FS}/\sigma^2_{WFS}$, assuming that between and within family genetic variances are approximately the same. That is correct for traits with low dominance.

Considering separated analysis for the cloned parents experiment the following variance structures and relations are obtained:

Genetic variance among cloned parents: $\sigma^2_{CP} = \sigma^2_A + \sigma^2_D$.

Individual broad sense heritability: $h^2_b = \sigma^2_{CP}/\sigma^2_{FCP}$, where σ^2_{FCP} is the individual phenotypic variation for parents.

Considering the joint analysis for both experiments (cloned parents and full-sib families) and assuming the average allelic frequency in each population as approximately the same, i.e., p close to r, it was possible to estimate the additive genetic variance (σ^2_A) by isolating it from the sum of itself and the dominance variance. The three types of covariance between relatives (full-sibs, cloned parents and parent-offspring) were used simultaneously for estimating σ^2_A, by using residual maximum likelihood (REML). This assumption (p close to r) was made only for estimation of this parameter σ^2_A. We consider and believe that such assumption approximately holds for a quantitative trait (controlled by many genes) in two non-improved populations. The following estimates were obtained:

Additive genetic variance from the joint analysis:
$\hat{\sigma}^2_A = 75.8103$.

Dominance genetic variance from the joint analysis:
$\hat{\sigma}^2_D = 11.2846$.

Narrow-sense individual heritability: $h^2_n = \hat{\sigma}^2_A/\sigma^2_{FJA}$, where $\sigma^2_{FJA} = 151.3784$ is the individual phenotypic variation from the joint analysis.

Broad-sense individual heritability from the joint analysis: $h^2_{bj} = (\hat{\sigma}^2_A + \hat{\sigma}^2_D)/\sigma^2_{FJA}$, where $\sigma^2_{FJA} = 151.3784$ is the individual phenotypic variation from the joint analysis.

The standard errors for the estimated variance component were obtained through the inverse of the second derivatives matrix (Hessian matrix) of the residual likelihood function with respect to the variance components. The standard errors estimated for the heritabilities were obtained by the delta technique described by BULMER (1980).

Results

Out of the 21 parents assessed, 12 were resistant (R), seven were susceptible (S) and two were highly susceptible (HS) (*Figure 1*). Necrosis was observed on the bark of all five plants of the susceptible clone (HGU1172). Of the S and HS parents only for U1275 (S) we observed wilt. In the others S and HS parents external cankers and radial wood darkening were observed. In the resistant genotypes U1450, G547, G58, U1455 and HRC2277 (control) no wood lesions were observed.

The individual inheritability in the narrow and broad sense was estimated at 50% and 59%, respectively, and the heterosis value in progenies was –3% (*Table 1*). A

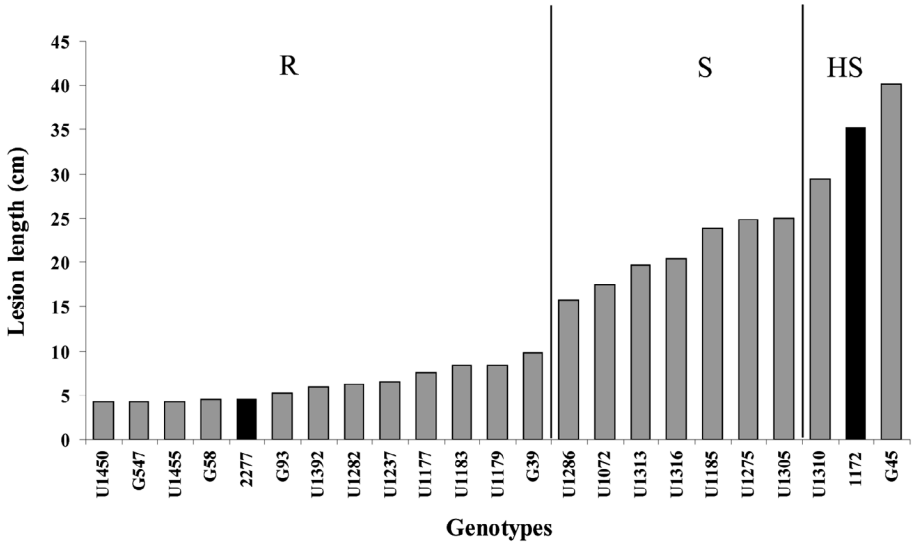

Figure 1. – Mean lesion length on the wood of *Eucalyptus grandis* (G) and *E. urophylla* (U) genotypes and in control clones HRC2277 and HGU1172 (highlighted columns), evaluated for resistance to *Ceratocystis fimbriata*. The genotypes were grouped in three classes (R = resistant; S = susceptible and HS = highly susceptible) based on the SCOTT-KNOTT test ($P \leq 0.01$).

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Table 1. – Estimates of variance components and genetic parameters (heritability and coefficients of genetic variation) associated with *Eucalyptus grandis* x *E. urophylla* families, evaluated for resistance to *Ceratocystis fimbriata* based on the lesion length on the wood (cm).

Genetic parameters analysis for the full-sib experiment	**Values**
Genotypic variance among full sib families	43.7642± 12.86
Individual phenotypic variation for progenies	158.8642
Individual phenotypic variation within family	115.0958± 17.32
Individual heritability within family	0.38
Heritability of family means	0.88
Accuracy of family selection	0.94
Coefficient of genotypic variation among progenies (CVg, in %)	39.33
Coefficient of residual variation (CVe, in %)	64.10
Coefficient of relative variation (CVg/CVe in %)	0.61
Genetic parameters analysis for the cloned parents experiment	**Values**
Genotypic variance among cloned parents	83.5509±28.37
Individual phenotypic variation for parents	139.6864
Residual variation	56.1355±9.48
Broad-sense individual heritability	0.59 ± 0.22
Genetic parameters from the joint analysis	**Values**
Additive genetic variance from the joint analyses	75.8103±21.53
Dominance genetic variance from the joint analysis	11.2846±4.18
Residual variance from the joint analysis	64.2835±10.22
Phenotypic variance from the joint analyses	151.3784
Narrow-sense individual heritability	0.50 ± 0.11
Broad-sense individual heritability from the joint analysis	0.58 ± 0.

wide genotypic variation (39%) between families was found, which allowed high heritability (88%) and accuracy (94%) for the selection of families. The genetic variability for selection within families was also high, as showed by the high heritability estimate (38%) for selection within families (*Table 1*).

Results of the genetic means (predicted genotypic values) of the progenies and checks were compared (*Table 2*). Out of parents assessed, G39, classified as R, was involved in a considerable number of crosses [G39 x U1450 (R), x U1282 (R), x U1183 (R), x U1072 (S), x U1313 (S), X U1275 (S), x U1305 (S), x U1034 ex U1206]. The mean lesion length in progenies derived from parent G39 increased according to the increasing susceptibility of the parents used in the cross (*Figure 2*).

A genetic gain of –74.4% can be obtained with the selection of the 50 best potential clones in the families assessed. Six individuals (U1392 x G82, G91 x U1274, U1185 x G99, G47 x U1455, U1179 x G547 and G39 x U1450) of rather resistant families were used in this selection (*Table 3*). A genetic gain of –79.4% by selection of the 10 best individuals can be achieved, i.e., a reduction of 79.4% in mean lesion length, compared to the progeny population. In this case, the genotypic value or expected genetic mean for lesion length of the best clones would be 2.16 cm (*Table 3*), which is less than half the lesion length (4.5 cm) of the resistant control, in other words, a genetic gain of –52% (52% reduction in the lesion length) over the resistant control.

The analysis of the frequency distribution of disease severity (*Figure 3*) shows the presence of several intermediate resistance levels and transgressive segregation in the F_1 population, indicating the possibility that susceptible parents have other resistance genes than the resistant parents.

Discussion

The phenotypic analyses indicated the existence of a high genetic variability for resistance to *Ceratocystis fimbriata* wilt in *E. grandis* and *E. urophylla* as previously reported by Zauza et al. (2004) in others clones of the same species. Genetic variability for resistence to *Ceratocystis* wilt has also been described in other species as *Mangifera indica* (Ribeiro et al., 1984; Ribeiro et al., 1986; Ribeiro et al., 1995; Rossetto et al., 1997), *Coffea arabica* (Castilla, 1982) and *Crotalaria* spp. (Ribeiro et al., 1977). Our results indicate that it is possible to select *Eucalyptus* spp. genotypes resistant to *Ceratocystis* wilt for planting.

In some pathosystems, the genetic basis of disease resistance formerly considered polygenic or unknown, proved to have a simple inheritance model when detailed studied (Kinloch et al., 1970; Newcombe et al., 1996; Villar et al., 1996; Wilcox et al., 1996; Junghans et al., 2003). In the present work, the genetic parameters estimated allowed the following conclusions: (i) due to the high inheritability (50 and 59% in the narrow and broad sense, respectively) the degree of genetic control of the trait is high and probably controlled by a small number of genes, (ii) the allelic dominance of the trait is low, since the two values of heritability are very close. The determination coefficient of additive genetic resistance is therefore 50% and the determination degree of genetic dominance is only 9%. The strong additive

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Table 2. – Estimates of the genotypic values (GV or genotypic means) of the family, of the mean family heritability (MFH_2), phenotypic variation within each family (PVF), coefficient of phenotypic variation within each family (CPVF) and the phenotypic value (PV or phenotypic mean) of each family of *Eucalyptuas grandis* (G) x *E. urophylla* (U), evaluated for resistance to *Ceratocystis fimbriata* based on the lesion length on the wood (cm).

Progeny	No. of plants	Genetic parameters				
		PV	GV	MFH_2	PVF	CPVF (%)
U1392 (R*) x G82	23	2.34	3.19	0.87	0.52	30.82
U1274 x G91	19	2.47	3.46	0.85	4.51	85.98
U1185 (S) x G99	19	3.05	3.97	0.85	0.89	30.93
G47 x U1455 (R)	18	3.33	4.26	0.84	4.44	63.28
U1179 (R) x G547 (R)	19	3.51	4.37	0.85	3.70	54.80
U1185 (S) x G51	21	3.62	4.39	0.86	1.97	38.77
G39 (R) x U1450 (R)	23	4.02	4.69	0.87	0.31	13.85
U1185(S) x G47	20	4.34	5.06	0.85	5.18	52.44
Resistant control	10	-	**4.50**	-	-	-
G45 (HS) x U1450 (R)	24	4.67	5.25	0.87	9.12	64.67
U1310 (HS) x G51	20	5.20	5.82	0.85	30.22	105.72
U1237 (R) x G93 (R)	20	5.48	6.07	0.85	19.09	79.73
U1286 (S) x G99	24	7.85	8.12	0.87	26.51	65.59
U1310 (HS) x G58 (R)	19	8.37	8.63	0.85	186.44	163.13
G99 x U1316 (S)	20	8.80	9.00	0.85	86.91	105.94
U1179 (R) x G549	23	8.87	9.04	0.87	98.30	111.78
G39 (R) x U1183(R)	20	9.10	9.27	0.85	187.12	150.32
G45 (HS) x U1177 (R)	24	10.33	10.35	0.87	88.71	91.18
G39 (R) x U1034	19	10.55	10.55	0.85	93.41	91.61
G39 (R) x U1206	18	11.67	11.52	0.84	131.00	98.08
U1310 (HS) x G91	19	12.76	12.49	0.85	170.37	102.29
U1185 (S) x G83	20	13.13	12.82	0.85	82.02	68.98
G39 (R) x U1282 (R)	18	14.17	13.70	0.84	550.09	165.52
U1310 (HS) x G93 (R)	24	14.63	14.21	0.87	146.68	82.78
U1310 (HS) x G547 (R)	20	15.33	14.76	0.85	143.80	78.22
G39 (R) x U1313 (S)	20	15.55	14.96	0.85	99.10	64.02
U1286 (S) x G504	25	15.56	15.07	0.87	170.40	83.89
U1412 x G549	25	20.86	19.87	0.87	488.28	105.93
G39 (R) x U1275 (S)	19	21.11	19.81	0.85	191.32	65.52
G39 (R) x U1072 (S)	20	27.15	25.20	0.85	190.98	50.90
G39 (R) x U1305 (S)	19	27.47	25.39	0.85	283.29	61.27
Susceptible control	10	-	**35.20**	-	-	-

* Parents evaluated and classified as R = resistant; S = susceptible and HS = highly susceptible based on the grouping test of SCOTT and KNOTT ($P \leq 0.01$).

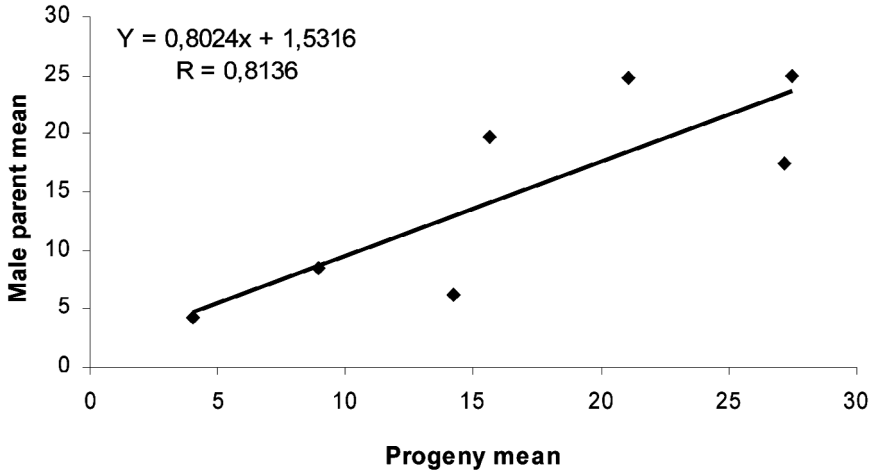

Figure 2. – Correlation of the lesion length on the wood (cm), observed between the progenies and the different male parents of *Eucalyptus urophylla*, involved in the crosses with the female parent G39 of *E. grandis*.

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Table 3. – Estimates of genotypic values and genetic gains involving trees of families of *Eucalyptus grandis* (G) x *E. urophylla* (U), evaluated for resistance to *Ceratocystis fimbriata*, based on the lesion length on the wood (cm).

Family	Plant	Phenotypic value	Genotypic vaue	Genotypic effect below the mean	Genetic gain (%)
U1274 x G91	1	1	2.16	-8.3457	-79.42
U1274 x G91	3	1	2.16	-8.3457	-79.42
U1274 x G91	5	1	2.16	-8.3457	-79.42
U1274 x G91	8	1	2.16	-8.3457	-79.42
U1274 x G91	9	1	2.16	-8.3457	-79.42
U1274 x G91	10	1	2.16	-8.3457	-79.42
U1274 x G91	14	1	2.16	-8.3457	-79.42
U1274 x G91	15	1	2.16	-8.3457	-79.42
U1274 x G91	18	1	2.16	-8.3457	-79.42
U1274 x G91	19	1	2.16	-8.3457	-79.42
U1392 x G82	3	1.5	2.30	-8.2119	-78.14
U1392 x G82	13	1.5	2.30	-8.2119	-78.14
U1274 x G91	7	1.5	2.43	-8.0822	-76.91
U1392 x G82	8	1.8	2.46	-8.0538	-76.64
U1392 x G82	1	2	2.56	-7.9484	-75.63
U1392 x G82	4	2	2.56	-7.9484	-75.63
U1392 x G82	6	2	2.56	-7.9484	-75.63
U1392 x G82	7	2	2.56	-7.9484	-75.63
U1392 x G82	12	2	2.56	-7.9484	-75.63
U1392 x G82	16	2	2.56	-7.9484	-75.63
U1392 x G82	17	2	2.56	-7.9484	-75.63
U1392 x G82	18	2	2.56	-7.9484	-75.63
U1392 x G82	19	2	2.56	-7.9484	-75.63
U1392 x G82	21	2	2.56	-7.9484	-75.63
U1392 x G82	5	2.5	2.82	-7.6849	-73.13
U1392 x G82	9	2.5	2.82	-7.6849	-73.13
U1392 x G82	10	2.5	2.82	-7.6849	-73.13
U1392 x G82	11	2.5	2.82	-7.6849	-73.13
U1392 x G82	22	2.5	2.82	-7.6849	-73.13
U1392 x G82	23	2.5	2.82	-7.6849	-73.13
U1185 x G51	16	1.5	2.87	-7.6407	-72.71
U1185 x G99	3	2	2.93	-7.5784	-72.11
U1185 x G99	10	2	2.93	-7.5784	-72.11
U1185 x G99	17	2	2.93	-7.5784	-72.11
U1274 x G91	2	2.5	2.95	-7.5552	-71.89
G47 x U1455	3	2	3.07	-7.4418	-70.81
G47 x U1455	6	2	3.07	-7.4418	-70.81
G47 x U1455	8	2	3.07	-7.4418	-70.81
G47 x U1455	9	2	3.07	-7.4418	-70.81
G47 x U1455	11	2	3.07	-7.4418	-70.81
G47 x U1455	14	2	3.07	-7.4418	-70.81
U1392 x G82	2	3	3.09	-7.4214	-70.62
U1392 x G82	14	3	3.09	-7.4214	-70.62
U1392 x G82	20	3	3.09	-7.4214	-70.62
U1179 x G547	1	2	3.12	-7.3884	-70.31
U1179 x G547	9	2	3.12	-7.3884	-70.31
U1179 x G547	14	2	3.12	-7.3884	-70.31
U1179 x G547	17	2	3.12	-7.3884	-70.31
U1179 x G547	19	2	3.12	-7.3884	-70.31
U1185 x G51	20	2	3.13	-7.3772	-70.20
Overall gain	-	-	-	-7.82	-74.37

genetic control and low dominance of the trait were confirmed by virtually nil heterosis (–3%), estimated when comparing the progeny performance with the mean performance of their parents.

The additive effect can be defined as a linear relationship between the genotypic values of individuals of a population and the number of favorable alleles they have (CRUZ and REGAZZI, 1997). The prevalence of additive effects in the genetic control of Ceratocystis wilt resistance is therefore an indication of an easy identification of superior genotypes with a high concentration of favorable alleles, suitable for breeding. High narrow sense (65–77%) heritability values were also found by BORGES and BRUNE (1981) in the *E. grandis* x *Chrysoporthe cubensis* pathosystem.

The linear adjustment of the response found in the crosses involving the resistant parent G39 and several *E. urophylla* parents along with the high correlation coefficient of 81% between the progeny and parent performances confirmed the strong additive genetic control

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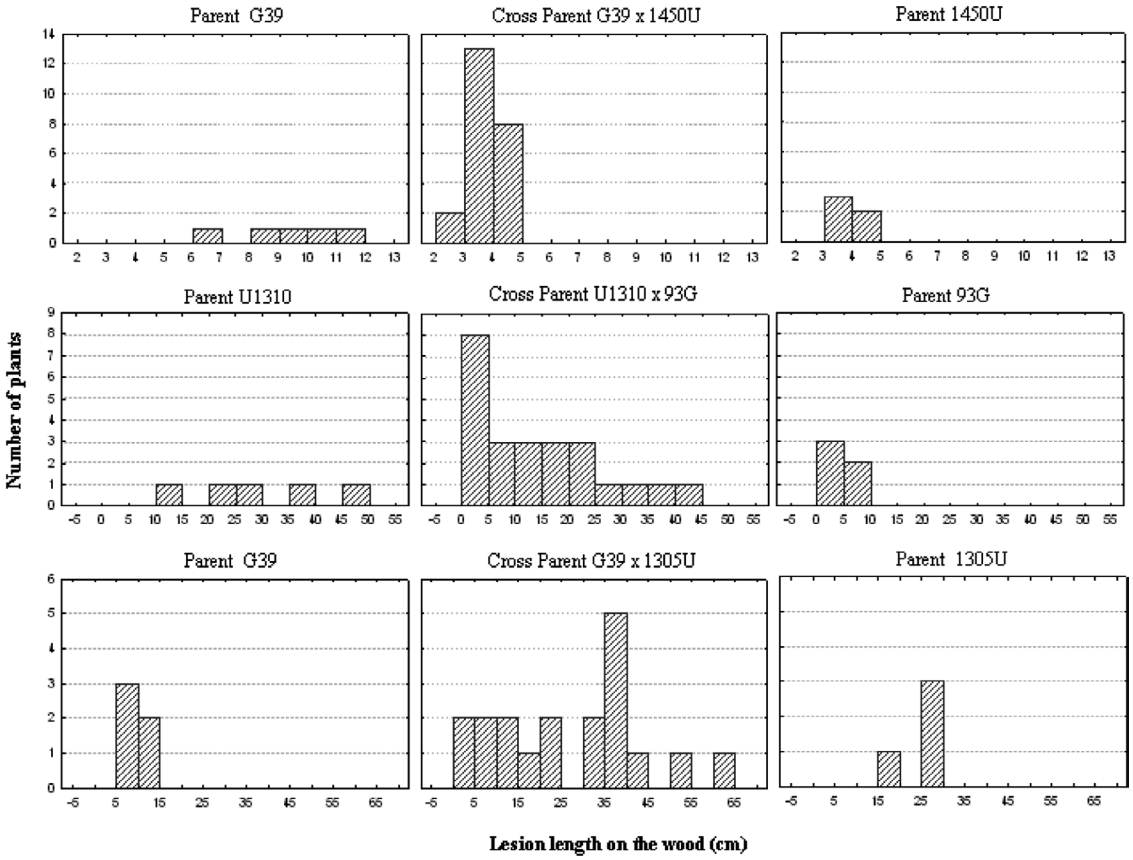

Figure 3. – Frequency distribution based on the lesion length on the wood of parents and three progenies of *Eucalyptus grandis* (G) x *E. urophylla* (U) evaluated for resistance to Ceratocystis wilt, caused by *Ceratocystis fimbriata*. Different intermediate resistance levels and transgressive segregations were observed in the F_1 population.

of the trait. This correlation estimate is strongly associated with inheritability in the narrow sense. The analysis of the other progenies showed that none was more susceptible than the susceptible control and that eight of them were more resistant than the resistant control, which is favorable for selection. In addition, the coefficients of phenotypic variation within families were also high (except cross G39 x U1450) indicating a great potential for selection within families as well.

In fact, the combination of the variability among and within progenies makes possible the selection of highly resistant plants. High genetic gains (up to 79%) can be obtained through selection of transgressive individuals in the F_1 generation as well as in the controlled crosses. It is worth highlighting that the controlled inoculation conditions are crucial for an efficient implementation of selection. Moreover, one should consider the possibility of genetic variability in the pathogen population and its interaction with differential host genotypes. A *C. fimbriata* isolate obtained from the areas chosen for commercial clone planting must therefore be used in resistance evaluations.

The evaluations carried out here were effective tools in the selection of superior parents for breeding programs as well as of promising hybrids; after evaluation of growth, adaptability, wood quality and resistance to other pathogens these may be indicated for planting at a commercial scale.

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