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Genetical Analysis of the 1999 *C. macrocarpa* ProgenyTrial at Birch Hill

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EXECUTIVE SUMMARY

Cupressus macrocarpa has the ability to grow well in New Zealand, producing high quality timber at a growth rate only slightly slower than radiata pine. Unfortunately, most *C. macrocarpa* trees are susceptible to cypress canker. The use of chemicals has brought limited success due to the short impact of these remedies and their high cost. Selecting trees that are resistant to cypress canker, and propagating by means of grafting and controlled crossing has been proved the most efficient way to solve this problem in *Cupressus sempervirens*. Canker severity was scored in a second generation open pollinated progeny trial with 135 female parents in a single-tree-plot in a sets-in-replicates design at Birch Hill. The objectives of this analysis are 1) to explore the genetic variation of canker resistance, 2) to recognize families with canker resistance and select progeny from the families identified, and 3) to analyse the effects of canker disease on other traits.

Canker severity score (CNK06), total tree height (HT06), branch angle score (BRA06) and stem acceptability score (ACC06) were assessed at age 6 years. Canker severity score (CNK13), diameter-at-breast-height (DBH13), branch size (BRS13) and stem acceptability score (ACC13) were assessed at age 13 years. CNK06 had a scale of 1 to 6 (1 = no canker symptoms, 6 = killed by canker). CNK13 had a scale of 0 to 4 (0 = no canker symptom and 4 = killed by canker).

Variance components and heritability of canker severity and other traits were estimated using a linear mixed model. Fixed and random effect solutions were obtained by solving the mixed model equations. The residuals are assumed to be independent, and the analysis has a structure based on a decomposition of residual effects into spatially depended and spatially independent residuals. We used a covariance structure that assumes separable first-order autoregressive processes in rows and columns. A bivariate model was used to estimate genetic correlations among traits assessed, where a non-spatial analysis was carried out with assumptions that residual variance is consistent across rows and columns.

Highly significant genetic variation in canker severity was found at age 6 years and at age 13 years. Canker heritability was 0.07 at age 6 years and 0.26 at age 13 years. This may be because canker disease severity was not fully expressed at age 6 years. The narrow-sense heritabilities for DBH (0.65), height (0.35), branch angle (0.43), branch size (0.17) and stem acceptability at year 13 (0.22) were high to moderate. Visible spatial trend was found in canker severity and all other traits except ACC06.

Canker disease significantly reduced growth rate and stem acceptability at later age. The genetic correlations of canker severity with DBH (-0.55), tree height (-0.26) and (-0.89), were negatively strong to moderate, but it had no effect on branch angle and branch size.

Genetic correlation between canker severity at ages of 6 and 13 years was high and significant, which means that families showing canker resistance at age 6 years also show resistance at age 13 years. The genetic correlations of canker severity with DBH (-0.55), tree height (-0.26) and ACC13(-0.89), were strongly to moderately negative. Canker disease significantly decreased growth rate at both ages of 6 and 13 years. Canker disease affected tree diameter more than tree height. Canker disease had high and significant genetic correlations with tree acceptability at age 13 years (ACC13). No significant genetic correlation of canker severity was found with stem acceptability at age 6 years, branch angle or branch size.

A selection index was developed using canker severity, DBH and stem acceptability, with a heavy weight on canker severity. Twenty top open pollinated families were identified with a maximum of one family selected per grandparental family. Forty progeny were selected from the twenty families, with two top progeny from each family. A reduction of 0.59 in canker severity, an increase of 53.64 mm in DBH, and an increase of 0.25 in stem acceptability can be expected in the next generation, based on data at age of 13 years, if a forward selection scheme is applied.

INTRODUCTION

Cupressus macrocarpa is native to California, where it occupies one of the smallest areas of any forest tree species at 30 hectares[1]. It was brought to New Zealand in the 1860s and planted mainly as shelter belts, but occasionally as plantations[2]. It has the ability to grow well in New Zealand, producing high quality timber at a growth rate only slightly slower than radiata pine. The timber is a low to medium density softwood and has large amount of heartwood, which is a golden brown honey colour. Its timber is moderately stiff and strong. *Cupressus macrocarpa* is one of the most naturally durable exotic softwoods grown in New Zealand[2].

Unfortunately, most *C. macrocarpa* trees are susceptible to cypress canker, which has annihilated entire stands and removed it from the list of species worth trying by most forest growers. Cypress canker is caused by fungi from the genus *Seiridium*[3]. Cankers form on stems, branches and in branch axils, causing dieback of leading and lateral shoots. Very small trees may be killed from infection low on the stem. On older trees, stems with large cankers are prone to malformation and breakage in high winds, and death may eventually result from the combined effects of many branch cankers[3]. Cypress canker and its causal fungi are found throughout New Zealand[4]. Very dry soil and lack of nutrients are certainly a contributing factor to the outbreak of this disease.

Different methods have been used to control cypress canker spread. The use of chemicals, i.e. spraying with fungicides, and mechanical interventions, like cutting down of infected trees, have brought limited success due to the short impact of these remedies and their high cost[5]. Selecting trees that are resistant to cypress canker and propagating them by means of grafting and controlled crossing has been proved to be the most efficient solution to this problem in *Cupressus sempervirens*[5]. Four clones of *C. sempervirens*[6, 7]are already commercially available and patented for their resistance to the canker.

Scion started a *C. macrocarpa* breeding program in the early 1980s, with the selection of plus trees throughout New Zealand[8]. Seed was collected from these trees, and more was sourced from a rangewide seed collection in California[9]. Seedlings were raised and a progeny trial was planted on two sites (Strathallan and Gwavas) in 1985. These trials were assessed in 1993 and canker was evident at both sites, but it appeared that good stands would be formed at each site. Many trees at Strathallan had cones, but almost no trees at Gwavas were coning. A second assessment was made in 1996 when canker infection at Strathallan was similar to the 1993 assessment. However, most trees at Gwavas were heavily infected and many had been killed. Genetic resistance to canker was identified, and selections were made of well-formed trees in resistant families at Strathallan. Seed was collected from these selections at Strathallan in 1997, and some was used to raise seedlings for a second generation progeny trial, planted on two sites in 1999, Dunsdale forest and Birch Hill station. The Dunsdale site was also assessed at age six, but had relatively few canker symptoms. More canker symptoms had developed at Birch Hill, so canker severity was scored there at 13 years of age.

The objectives of this analysis are 1) to explore genetic variation of canker resistance,2) to recognize families with canker resistance and make forward selections in the progeny from the families identified, and 3) to analyse the effects of canker disease on other traits.

METHODS

Trial Design

The trial analysed was a second generation open pollinated *Cupressus macrocarpa* progeny trial planted in Birch Hill station. It had 135 female parents in a single-tree-plot in a sets-in-replicates design. There were thirty replicates and each family had 30 trees, one per replicate. Thirty trees per block were planted at six rows of five trees, the plot size being 13.7 meters by 21 metres. Stocking at planting was 1,235 stems per hectare (2.7 metres x 3 metres). The overall layout of the trial has 85 rows and 54 columns.

This ex-pasture site was ripped to alleviate compaction from stock and to induce deep rooting. The soil, called “Ruapuna soils”, was classed Yellow Grey to Yellow Brown Earths Intergrade. The parent material was greywacke loess and alluvium and was mainly shallow silt loams. As the soil was stony and bouldery, the soil was well drained. Being an ex-pasture site, this site was medium to high in fertility status (DSIR Soil Bulletin No.27, 1968).

There were two control seedlots and nine families of *C. pygmaea* (sometimes called *C. goveniana* var. *pygmaea*). Control seedlots were excluded from the genetic analysis.

Assessment

Canker severity score (CNK06), total tree height (HT06), branch angle score (BRA06) and acceptability score (ACC06) were assessed at age 6 years. Canker severity score (CNK13), diameter-at-breast-height (DBH13), branch size (BRS13) and acceptability (ACC13) were assessed at age 13 years. CNK06 had a scale of 1 to 6 (1 = none of canker symptom, 6 =killed by canker). CNK13 had a scale of 0 to 4 (0 = no canker symptom and 4=killed by canker). BRA06 were assessed subjectively in a scale of 1 to 5, where small numbers are steep angles. ACC06had a scale of 0 to 2 (0 = unacceptable based on size, health, malformation, 1 = acceptable and 2 = plus tree). BRS13 was the size of an average branch, subjectively assessed to the nearest centimetre. ACC13 was acceptability on a 0-1 scale (0=unacceptable and 1=acceptable). A summary of statistics of traits assessed and score distribution for score traits is presented in Table 1.

Table 1. Summary statistics and score distribution of traits assessed in second generation of *Cupressus macrocarpa* progeny trial

Trait	Unit	N	Mean	SD	Min	Max	Score distribution						
							0	1	2	3	4	5	6
CNK06		3903	1.42	1.23	1	6	-	3370	146	66	104	20	197
CNK13		3723	1.41	1.1	0	4	975	1044	987	653	64	-	-
DBH13	mm	3659	148.2	47.6	30	338	-	-	-	-	-	-	-
HTD06	dm	3706	33.98	0.82	10	6.7	-	-	-	-	-	-	-
BRA06		3652	3.28	0.82	1	5	-	14	622	1536	1296	184	-
BRS13	cm	3657	1.9	0.5	1	3	-	670	2856	382	-	-	-
ACC06		3546	0.99	0.18	0	2	85	3427	34	-	-	-	-
ACC13		4147	0.27	0.45	0	1	3017	1130	-	-	-	-	-

StatisticalAnalysis

Univariate Analysis – Spatial Analysis

The data were analysed using an individual tree linear mixed model, implemented with ASREML [10]. The general form of linear mixed model was

$$\boldsymbol{Y = Xb + Zu + e}$$

where ***y*** is the vector of data, ***b*** is a vector of fixed effects with its design matrix **X**, ***u*** is a vector of random effects with its design matrix **Z**, and ***e*** is a vector of residuals. Fixed and random effect solutions are obtained by solving the mixed model equations [11]:

$$\begin{bmatrix} \mathbf{X'R^{-1}X} & \mathbf{X'R^{-1}Z} \\ \mathbf{Z'R^{-1}X} & \mathbf{Z'R^{-1}Z + G^{-1}} \end{bmatrix} \begin{bmatrix} \hat{\mathbf{b}} \\ \hat{\mathbf{u}} \end{bmatrix} = \begin{bmatrix} \mathbf{X'R^{-1}y} \\ \mathbf{Z'R^{-1}y} \end{bmatrix}$$

where **R** is the variance-covariance matrix of the residuals and **G** is the direct sum of the variance-covariance matrices of each of the random effects. Where residuals are assumed to be independent, **R** has a structure based on a decomposition of ***e*** into spatially depended (ξ) and spatially independent (Π) residuals. We used a covariance structure that assumes separable first-order autoregressive processes in rows and columns. The **R** matrix is

$$\mathrm{R} = \sigma_{\xi}^{2}\left[\mathrm{ARI}(\rho_{\mathrm{c}}) \otimes \mathrm{ARI}(\rho_{\mathrm{r}})\right] + \sigma_{\eta}^{2}\mathrm{I}$$

where σ_{ξ}^{2} is the spatial residual variance, σ_{η}^{2} is the independent residual variance, ρ_{c} is autocorrelation parameter of column, ρ_{r} is autocorrelation parameter of row, I is an identity matrix, and ARI(ρ) represents a first-order autoregressive correlation matrix which, for ordered spatial coordinates of size n, has the form:

$$\mathrm{ARI}(\rho) = \begin{bmatrix} 1 & \rho & \rho^{2} & \cdots & \rho^{\mathrm{n}} \\ \rho & 1 & \rho & \cdots & \vdots \\ \rho^{2} & \rho & 1 & \cdots & \vdots \\ \vdots & \vdots & \vdots & \ddots & \vdots \\ \rho^{\mathrm{n}} & \cdots & \cdots & \cdots & 1 \end{bmatrix}$$

where ρ is the autocorrelation parameter.

Heritability of a trait assessed is calculated as

$$\mathrm{h}^{2} = \frac{\sigma_{\mathrm{a}}^{2}}{\sigma_{\mathrm{a}}^{2} + \sigma_{\xi}^{2} + \sigma_{\eta}^{2}}$$

Bivariate Analysis – Non-spatial Analysis

A bivariate individual tree model was used to estimate genetic correlations among traits assessed, implemented with ASREML [10]. A non-spatial analysis was carried out with assumptions that **R** is defined as $\sigma_e^2 I$. The joint distribution of the random effects was assumed to be multivariate normal, with means and (co)variances:

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

where **0** is a null matrix and **G** and **R** are variance-covariance matrices corresponding to ***u*** and ***e***, respectively. Genetic correlation between trait *i* and trait *j* ($r_{g_{ij}}$) is calculated as

$$r_{g_{ij}} = \frac{\sigma_{a_{ij}}}{\sqrt{\sigma_{a_i}^2 \sigma_{a_j}^2}}$$

where $\sigma_{a_{ij}}$is genetic covariance between trait *i* and trait *j*, $\sigma_{a_i}^2$ is genetic variance of trait *i* and $\sigma_{a_j}^2$ is genetic variance of trait *j*.

Selection Index

A selection index was developed to select 40 superior individuals with key traits canker severity, DBH and stem acceptability from 20 top OP families, two from each family. The selection index applied equal index weights on EBVs of DBH and stem acceptability, and doubled the index weight on EBV of canker severity due to the importance of the trait.

$$I = \frac{a_d}{s_d} + \frac{a_a}{s_a} - \frac{2a_c}{s_c}$$

where a_d,a_a and a_c are EBVs for DBH, acceptability and canker resistance, and s_d, s_a and s_c are the standard deviations of EBVs for DBH, acceptability and canker resistance. Two best individuals from 20 families with the highest selection index were selected. Only one family was selected if two or more families shared the same grandparents. Apart from selection index, a threshold was set to phenotypes of trees selected. Phenotype of canker severity score was zero, that of DBH was equal or above 150 mm, and that of stem acceptability was one.

RESULTS

Canker Phenotype in Progeny and Control Trees

Table 2 presents the average performance in progeny and control trees, and also in the best performance families. The heavy infection of cypress canker (*Seiridium cardinale*, *S. unicorne*, *S. cupressi*) compromised the growth of most families, allowing some canker-resistant families to dominate in spectacular fashion. Growth traits were significantly affected by canker disease. The average diameter of all trees at age of 13 years is 150 mm, but the best family averages 250 mm, with some trees over 300 mm. This contrasts with permanent sample plots installed into the more fertile sister trial at Dunsdale in May 2012, where trees in plots on the area of best growth averaged 230 mm in diameter and 170 mm on the poorest part. However the Dunsdale trial had been thinned to around 400 stems per hectare shortly before measurement.

Table 2. Means of family groupings

Grouping	N	N06	N13	Surv13	CNK06	CNK13	DBH13 (mm)	HTD06 (dm)	BRA06	BRS13 (cm)	ACC06	ACC13
Californian families[1]	858	764	768	90	1.41	1.48	141	33	3.23	1.91	0.98	0.27
Control 200	150	89	79	53	1.33	1.19	133	32	3.37	1.75	0.98	0.27
Control 300	90	83	82	91	1.52	1.60	150	36	3.19	2.04	0.97	0.24
NZ select families[2]	3076	2742	2704	88	1.43	1.32	150	34	3.29	1.90	0.99	0.35
Pygmy cypress	268	227	230	86	1.31	1.50	165	37	3.69	2.30	0.91	0.31
Best macro family 44	30	27	27	90	1.34	0.96	249	39	3.89	2.48	1.00	0.65
Second best family 139[3]	30	30	30	100	1.00	0.43	230	42	3.50	2.20	0.93	1.00
Best pygmy family 510	28	23	23	82	1.17	1.48	188	38	4.04	2.26	0.91	0.45

[1] Californian families were raised from seed collected from seed-bearing individuals (not selected for growth or form) across the natural range of the species in California[12].
[2]New Zealand select families were from seed collected from individual trees selected for superior growth and form in what were usually smallish stands scattered around the countryside by John Millerin 1982[8].
[3]Families 44 and 139 were recognized as the most canker resistant and had been used later on.

Two very good families (139& 50) shared 297 as a grandparent – a family where some trees were putatively identified as being a hybrid between *C. macrocarpa* and *C. sempervirens* on the basis of different foliage and bark characteristics. The selected trees that gave seed for family 50 also supplied the seed for two highly ranked clones identified in the recent assessment of the 2003 *C. macrocarpa* clonal trial on Scion grounds (report submitted to FFR but not finalised). Interestingly there were other descendants of 297 on this site and in the clonal trial that were not growing at all well.

The *C. pygmaea* appeared slightly better adapted to this site than the *C. macrocarpa*. It was not a surprise that the *C. Pygmaea* was able to handle soils of lesser fertility, as the parents were growing on podzol soils in California, where the cypresses were small, but dominated redwoods and Douglas-fir. The difference was most noticeable in the poorer grown eastern half of the trial, where the *C. macrocarpa* looked quite sick in some places. The foliage of the *C. pygmaea* emitted an extremely pungent smell, so strong that the assessors could smell the trees from 10 metres away. Unfortunately, the *C. pygmaea* was no more resistant to cypress canker than the *C. macrocarpa*.

Cupressus macrocarpa trees in the eastern part of the trial had an unthrifty look about them. There was noticeably more canker damage in this part of the trial, but some trees had very little live foliage and appeared to be moribund, often without canker symptoms.

Taking the mean of strips of blocks that run the length of the trial showed that the trees in the western-most strip had a diameter of 180mm. The next two strips were also quite good at 170 mm diameter, then the mean trended down over the next three strips to be 130 mm for the rest of the

trial, although growth was just over 140 mm in the easternmost strip itself. Height at age six similarly trended down from four metres to three metres and canker scores (on a 0-3 scale) increased from 1.15 to 1.67.

Just over 1000 trees had a canker score of 0 (no canker symptoms) and a further 1100 had a canker score of 1 (one small symptom). There are 3900 trees still alive out of 4500 planted. A Google Earth view of the trial is shown in Figure 1, and Figure 2 shows trees with foliage problems alongside a healthy tree.

Looking at *C. pygmaea* separately, the diameters on the western side were also 180 mm and were about 170 mm for the next four strips, trending down to 150 mm for the eastern strips. Details of various groups of seedlots are shown in Table 2.

Figure 1. Google Earth view of the trial in 2009. Note poorer growth on the right (eastern side)

Figure 2. Unhealthy *C. macrocarpa* tree on left, healthy tree on right, age 13 years

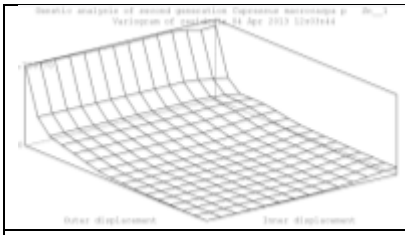
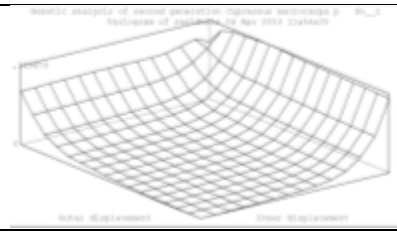
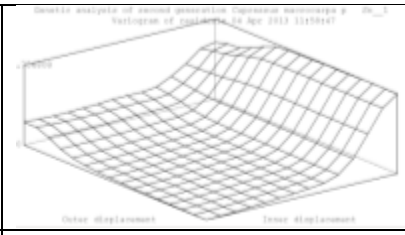
Variance Component and Heritability

Variance components and heritabilities of traits analysed are presented in Table 3. High spatial dependent residual variances were found in CNK13, DBH13, HTD06 and BRA06. High auto-correlations for row and column were found in all traits except ACC06. Highly significant heritabilities were observed in all traits except ACC06. Canker heritability at age 6 years (0.07) was lower than that at age 13 years (0.26). This may be because canker disease severity was not fully expressed at age 6 years. A visible spatial trend of residuals was found in all traits except ACC06 (Figure 3).

Table 3. Additive genetic variance (σ_α^2), spatial independent residual variance (σ_η^2), spatial dependent residual variance (σ_ξ^2), autocorrelations for row (ρ_r) and column (ρ_c) and heritabilities for traits assessed from a spatial analysis

Trait	σ_α^2	σ_η^2	σ_ξ^2	ρ_r	ρ_c	h^2	se
CNK06	0.10	1.40	0.03	0.99	0.99	0.07***	0.03
CNK13	0.33	0.81	0.15	0.99	0.99	0.26***	0.05
DBH13	1423	253	525	0.98	0.96	0.65***	0.09
HTD06	20.57	17.77	16.67	0.97	0.91	0.35***	0.06
BRA06	0.32	0.32	0.11	0.99	0.95	0.43***	0.08
BRS13	0.04	0.20	0.01	0.90	0.87	0.17***	0.03
ACC06	0.001	0.03	0.002	-0.46	0.34	0.02 ns	0.02
ACC13	0.05	0.14	0.03	1.00	1.00	0.22***	0.04

Note: ns: not significant, *** P<0.001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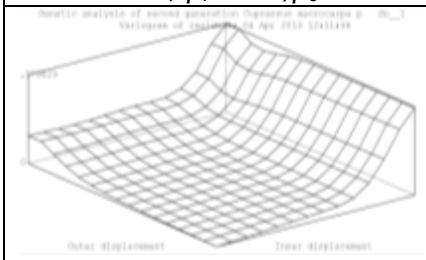
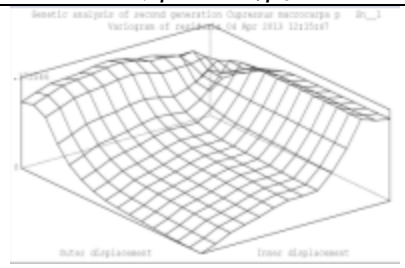
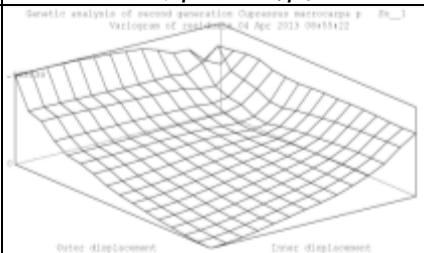
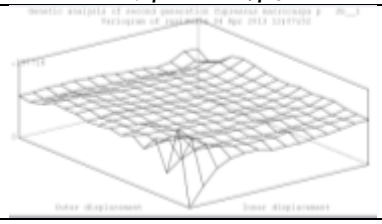
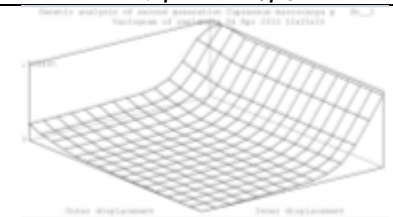
		
HTD06, ρ_r=0.97,ρ_c=0.91	BRA06, ρ_r=0.99,ρ_c=0.95	BRS13, ρ_r=0.90,ρ_c=0.87
		
ACC06, ρ_r=-0.46,ρ_c=0.34	ACC13, ρ_r=1.00,ρ_c=1.00	

Figure 3. Variograms of residuals for traits analysed in the spatial analysis (ρ_r and ρ_c are auto-correlations for row and column)

Genetic Correlations of Canker Severity with Other Traits

Genetic correlation between canker severity at ages of 6 and 13 years was high and significant (0.88), which means that families showing canker resistance at age 6 years also showed resistance at age 13 years. Canker disease significantly decreased growth rate at both ages of 6 and 13 years. Canker disease affected tree diameter more than tree height. Canker disease had high and significant genetic correlations with ACC13 (-0.69 - -0.89), which means that either canker disease at age 6 years or 13 years can reduce tree acceptability at later age by damaging the crown. No significant genetic correlation between canker disease and tree acceptability at age 6 years was found. This could imply that canker disease in trees was not fully expressed at age 6 years. No significant genetic correlations were found between canker disease and branch angle, branch size either.

High and significant positive genetic correlation between tree diameter and tree height was found. Low but highly significant genetic correlation between height and branch angle was found, implying that tall trees have big branch angle scores (flat branching). High and significant positive genetic correlation between growth traits and acceptability at age 13 (ACC13) was found. Acceptability at age 6 years had no significant genetic correlations with any other traits. This suggests that six years of age is too soon to assess tree acceptability.

Table 4. Genetic correlations among canker severity at ages of 6 and 13 years and other traits

Trait	CNK13	DBH13	HTD06	BRA06	BRS13	ACC13	ACC06
CNK06	0.88***	-0.33***	-0.31ns	-0.07 ns	0.21 ns	-0.69***	-0.04 ns
CNK13		-0.55***	-0.26*	-0.14 ns	0.09 ns	-0.89***	0.53 ns
DBH13			0.69***	0.19 ns	0.71***	0.81***	-0.20 ns
HTD06				0.13***	0.54 ns	0.56***	0.45 ns
BRA06					0.15 ns	0.05 ns	-0.50 ns
BRS13						0.22 ns	-0.47 ns
ACC13							-0.30 ns

Note: ns: not significant, * P<0.05, *** P<0.001

Index Selection

Table 5 shows the top twenty female families identified with the highest index for seed orchard purposes. Only one female parent family was selected from one grandparent. A reduction of 0.63 in canker severity, an increase of 39.09 mm in DBH, and an increase of 0.26 in stem acceptability can be expected in the progeny of these female parents. A full list of EBV of canker severity, DBH and stem acceptability for 135 female parents at age 13 years is attached in Appendix 1.Table 6 listed 40 progeny selected from the 20 top families, two from each family. A reduction of 0.59 in canker severity, an increase of 53.64 mm in DBH, and an increase of 0.25 in stem acceptability can be expected in the next generation if a forward selection scheme is applied.

Groups of selected progeny were compared with the phenotypic mean of all selected families and all progeny in the population (Table 7). In a backward selection scheme, canker severity reduced from 1.41 to 0.95, DBH increased from 148.17 mm to 169.42 mm and stem acceptability increased from 0.27 to 0.48 in the top 20 families selected. In a forward selection scheme using the top 40 selected progeny, canker severity reduced from 1.41 to 0, DBH increased from 148.17 mm to 214 mm, and stem acceptability increased from 0.27 to 1.

Table 5. Twenty female parent families with the highest index

Family	CNK13		DBH13		ACC13		Grand-	Index
	EBV	Accuracy	EBV	Accuracy	EBV	Accuracy	parent	
139	-1.27	0.84	146.00	0.94	0.56	0.81	297	12.88
44	-0.44	0.82	177.50	0.93	0.40	0.81	279	9.48
115	-1.03	0.82	38.75	0.93	0.42	0.81	325	7.88
42	-1.18	0.83	12.13	0.94	0.44	0.81	277	7.79
102	-0.90	0.83	37.07	0.93	0.32	0.81	273	6.71
72	-0.88	0.82	37.16	0.93	0.31	0.81	328	6.53
36	-0.79	0.84	22.74	0.94	0.41	0.81	267	6.31
30	-0.72	0.82	15.80	0.93	0.31	0.81	252	5.24
89	-0.65	0.82	23.31	0.93	0.27	0.81	348	4.95
69	-0.22	0.83	45.82	0.93	0.34	0.81	324	4.27
51	-0.42	0.83	41.27	0.93	0.20	0.8	299	4.12
75	-0.56	0.82	19.06	0.93	0.20	0.81	332	4.05
91	-0.61	0.83	6.71	0.93	0.20	0.81	253	3.89
4	-0.70	0.83	-11.83	0.94	0.21	0.81	19	3.73
90	-0.51	0.83	17.40	0.94	0.15	0.81	350	3.53
110	-0.44	0.83	16.97	0.93	0.16	0.81	300	3.28
124	-0.51	0.79	6.84	0.92	0.13	0.79	268	3.05
120	-0.01	0.83	82.48	0.94	0.08	0.81	317	2.96
1129	-0.49	0.82	16.63	0.93	0.04	0.81	340	2.75
1002	-0.26	0.82	29.90	0.93	0.13	0.81	13	2.72
Average	-0.63	0.83	39.09	0.93	0.26	0.81		

Table 6. Forty progeny selected from 20 female families, two from each family using index selection

Progeny	CNK13		DBH13		ACC13		Family	Index	Mean phenotypic values		
	EBV	Acc	EBV	Acc	EBV	Acc			CNK13	DBH13	ACC13
1074139	-0.92	0.62	126.70	0.91	0.39	0.59	139	12.90	0	249	1
1274139	-0.77	0.62	144.30	0.91	0.35	0.59	139	12.19	0	332	1
1252044	-0.45	0.61	144.40	0.91	0.29	0.58	44	9.75	0	336	1
1132044	-0.46	0.61	130	0.91	0.31	0.58	44	9.53	0	286	1
1054115	-0.84	0.61	51.86	0.91	0.34	0.59	115	9.57	0	183	1
1064115	-0.81	0.61	51.19	0.91	0.34	0.59	115	9.39	0	173	1
1021042	-0.78	0.61	16.86	0.91	0.33	0.59	42	18.77	0	154	1
1211042	-0.81	0.61	8.886	0.91	0.32	0.59	42	18.12	0	177	1
1115102	-0.68	0.61	125.60	0.91	0.28	0.59	102	10.46	0	290	1
1055102	-0.80	0.61	39.32	0.91	0.31	0.59	102	8.61	0	165	1
1104072	-0.70	0.61	50.83	0.91	0.28	0.58	72	8.21	0	185	1
1114072	-0.66	0.61	28.67	0.91	0.26	0.58	72	7.09	0	156	1
1073036	-0.71	0.61	38.41	0.91	0.33	0.59	36	8.29	0	155	1
1233036	-0.58	0.62	61.28	0.91	0.29	0.59	36	7.92	0	272	1
1043030	-0.68	0.61	36.40	0.91	0.28	0.59	30	7.65	0	188	1
1163030	-0.58	0.61	44.99	0.91	0.26	0.59	30	7.08	0	208	1
1242089	-0.53	0.61	34.78	0.91	0.24	0.58	89	6.29	0	266	1
1232089	-0.54	0.61	26.97	0.91	0.24	0.58	89	6.10	0	243	1
1283069	-0.32	0.61	35.17	0.91	0.26	0.59	69	5.18	0	192	1
1203069	-0.46	0.61	32.32	0.91	0.28	0.59	69	6.16	0	192	1
1245051	-0.45	0.61	70.59	0.91	0.21	0.58	51	6.69	0	270	1
1035051	-0.57	0.61	34.32	0.91	0.24	0.58	51	6.56	0	180	1
1205075	-0.63	0.61	39.15	0.91	0.24	0.58	75	7.06	0	175	1
1155075	-0.45	0.61	55.88	0.91	0.20	0.58	75	6.23	0	218	1
1032091	-0.67	0.61	21.49	0.91	0.24	0.59	91	6.73	0	176	1
1212091	-0.60	0.61	4.802	0.91	0.22	0.59	91	5.64	0	168	1
1124004	-0.58	0.61	40.34	0.91	0.23	0.59	4	6.73	0	180	1
1224004	-0.59	0.61	33.15	0.91	0.22	0.59	4	6.49	0	229	1
1104090	-0.54	0.61	31.71	0.91	0.21	0.59	90	6.05	0	162	1
1254090	-0.47	0.61	7.827	0.91	0.18	0.59	90	4.61	0	198	1
1201110	-0.55	0.61	111.50	0.91	0.22	0.59	110	8.72	0	273	1
1231110	-0.49	0.61	31.71	0.91	0.20	0.59	110	5.64	0	212	1
1213124	-0.51	0.61	45.76	0.91	0.19	0.58	124	6.12	0	261	1
1043124	-0.60	0.61	14.28	0.91	0.21	0.58	124	5.87	0	150	1
1185120	-0.36	0.61	94.90	0.91	0.19	0.59	120	6.76	0	235	1
1175120	-0.32	0.61	90.72	0.91	0.18	0.59	120	6.35	0	241	1
1264129	-0.49	0.61	51.57	0.91	0.14	0.59	129	5.84	0	225	1
1254129	-0.48	0.61	50.01	0.91	0.14	0.59	129	5.69	0	246	1
1162002	-0.40	0.61	52.34	0.91	0.19	0.59	2	5.70	0	198	1
1172002	-0.38	0.61	31.59	0.91	0.18	0.59	2	4.79	0	182	1
Average	-0.58	0.61	53.64	0.91	0.25	0.58			0	214	1

Table 7.Comparison of mean phenotypic values for all progeny, selected families and selected progeny

Trait	Mean of progeny	Mean of top 20 selected families	Mean of top 40 selected progeny
CNK13	1.41	0.95	0
DBH13 (mm)	148.17	169.42	214
ACC13	0.27	0.48	1

CONCLUSION

This study revealed a significant genetic variation in most traits except stem acceptability at age 6 years in *Cupressus macrocarpa*. The narrow sense heritability for canker severity was low (0.07) at age 6 years, but it became moderate (0.26) at age 13 years. Gea and Low[8] reported a similar trend in narrow-sense canker severity heritability in the first generation progeny trial of *Cupressus macrocarpa*. In another study of clonal *Cupressus macrocarpa* for canker resistance, narrow-sense heritability for canker severity was 0.37 at age 9 years (FFR technical note on the assessment of the 2003 C. macrocarpa clonal trial (in preparation)). This suggests that canker resistance may not be fully expressed at age 6 years. The narrow-sense heritabilities for DBH (0.65), height (0.35), branch angle (0.43), branch size (0.17) and stem acceptability at year 13 (0.22) were high to moderate.

Canker disease significantly reduced growth rate and stem acceptability at later age. The genetic correlations of canker severity with DBH (-0.55), tree height (-0.26) and (-0.89), were negatively strong to moderate, but it had no effect on branch angle and branch size.

A selection index was developed using canker severity, DBH and stem acceptability with a heavy weight on canker severity. Twenty open pollinated families were identified, with a maximum of one family selected per grandparental family. Forty progeny were selected from the twenty top families, with two top progeny from each family. A reduction of 0.59 in canker severity, an increase of 58.33 mm in DBH, and an increase of 0.25 in stem acceptability can be expected in the next generation, based on data at age 13 years, if a forward selection scheme is applied.

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APPENDICES

EBVs of Parents

A full list of EBVs of canker severity, DBH, stem acceptability for 135 female parents is available in Excel Spreadsheet.

EBVs of Progeny

A full list of EBVs of canker severity, DBH, stem acceptability for 4497 progeny is available in Excel Spreadsheet.