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GENETICS AND TREE IMPROVEMENT
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Title: First assessment of field progeny trial of selections of *Pinus radiata* for resistance to *Diplodia* infection.

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SUMMARY

Wind-pollinated progenies of Pinus radiata trees which had been selected intensively for resistance to shoot dieback associated with Diplodia infection were assessed in Tarawera and Kaingaroa Forests 6½ years after planting. Separate records were made of dieback on leaders and laterals, while stem diameters were measured, and stem straightness and desirability of branching habit were scored.

Growth was faster, and dieback more prevalent at Tarawera, where there was quite good resolution of progeny differences (repeatabilities of progeny means 0.51-0.73, $P < 0.05$-< 0.001) in the amount of dieback, irrespective of the measure used. Resolution of progeny differences in the incidence of dieback at Kaingaroa was poor (repeatability of progeny means ≤ 0.35, $P > 0.05$), presumably because of a very low disease incidence. There was no convincing evidence that progeny rankings differed materially between the two sites.

The select material did not show significantly less shoot dieback in the field than two control lots (seed orchard and unselected bulk in this case), even though it had performed significantly ($P < 0.05$) better than controls in a glasshouse inoculation trial. More definitely, dieback incidence of individual progenies in the field was effectively uncorrelated with infection response in the glasshouse. This suggests that genetic resistance, if present, may be highly specific to the circumstances of infection.

Transformation of data, in an attempt to overcome strongly asymmetric distributions of dieback counts, had little effect on results of analysis of variance.

No single feature of monoterpene composition of the parent clones could be correlated convincingly with disease incidence among the progenies, either in the field or in the glasshouse.

Some P. muricata, which grew more slowly, showed more dieback, especially at Tarawera where animal damage was a complicating factor. However, it showed much less needle cast.

INTRODUCTION

Selection of *P. radiata* for resistance to shoot dieback associated with infection by *Diplodia pinea* has been done on a pilot scale. This was on sites where the incidence of dieback was very high and where, perforce, the occurrence of chance escapes from disease was least likely.

Seed from the selections has been used for establishing a field progeny trial, to test the effectiveness of the field selection and potentially for reselection of the parents.

Since the establishment of the progeny trial further seed has been collected from the parent ortets, and has been used in a glasshouse inoculation trial (Burdon *et al.*, 1976). Progeny of the select parents showed better resistance overall in the glasshouse than control lots, although the select families differed markedly among themselves. These results suggested (a) that the field selection had been reasonably effective, and (b) that glasshouse inoculation would be a valid and effective screening technique. Nevertheless, it was still clearly desirable to be able to confirm the glasshouse result in the progeny test in the field.

In a field assessment of dieback there are two major problems:

(i) Obtaining a satisfactory quantitative measure of the observable occurrences of dieback;

(ii) Expressing the incidence on a scale of variation that has satisfactory statistical properties for analysis of variance.

In obtaining a quantitative measure one must weigh up several considerations:

- That dieback on the leader is of greater practical importance than dieback on laterals.

- That laterals, because they represent many more potential infection sites than the leader, offer the prospect of a more precise expression of inherent susceptibility.

- That there might or might not be a good genetic correlation in susceptibility to dieback between the leader and the laterals.

- That whereas cumulative incidence of dieback would provide more information, past occurrences are often difficult to identify with certainty.

This report covers the first assessment of dieback in the field progeny trial, in which a number of alternative measures of dieback incidence were tried.

MATERIAL AND METHODS

The Select Parents and the Progeny Trial

Details of the selection of parents and the establishment of the progeny trial are given in GTI Work Plan 96, so only a brief account is given here.

Twenty-six trees were originally selected, at eight years after planting, in Fenton's Mill Flat, Tarawera Forest. They were selected for tree form and dominance as well as for virtual freedom from dieback. Most of the trees provided sufficient wind-pollinated seed for a progeny trial, although in some cases the number of available cones was very small.

Progenies were raised in the nursery, and planted out during the winter of 1971, on two sites:

(i) Tarawera Forest (Plot R904) on a flat river terrace.

(ii) Kaingaroa Forest (Cpt 1350, Plot R944/13) on undulating terrain typical of the Northern Boundary area of the forest.

The layout conformed basically to randomised complete blocks, with 12 replicates of 8-tree plots at each site. However, some progenies were not represented at Tarawera, while at Kaingaroa some were missing in certain block replicates and their place taken with additional plots of controls.

Two *P. radiata* controls were used:

(i) Seed collection from the RA 1 seed orchard (AL 1)

(ii) Kaingaroa unselcted bulk seed collection, Seedlot R69/854.

In addition, one lot of "blue" *P. muricata* was included.

On neither site was there any severe outbreak of dieback, but it was decided that it was necessary to assess for whatever dieback was present, in March 1978. The trees were 6½ years old from planting, when dieback incidence was expected to peak, and were approaching the stage when inspection of the crowns could become very difficult.

ASSESSMENT

Stem diameter and tree form characters were assessed in addition to dieback. Because of the relatively low incidence of dieback, individual occurrences were counted instead of each tree being rated for general prevalence of the disease.

The following data were recorded on each tree:

1. D.b.h.o.b. (mm)

2. Stem straightness (1-9 scale); 1 = v. crooked, 9 = v. straight

3. Branch habit quality (1-9 scale); 1 = heavy, rough, irregular; 9 = light, even, strongly multinodal type

4. Stem malformation score (1-6); 1 = multiple forks
   2 = two forks or one multifork
   3 = single fork
   4 = large ramicorn(s)
   5 = small ramicorn(s)
   6 = no forks or ramicorns

5. Number of definite occurrences of dieback on leader

6. Number of doubtful occurrences of dieback on leader

7. Number of definite occurrences of dieback on laterals

8. Number of doubtful occurrences of dieback on laterals.

Assessment was done by crews of two, with one person measuring diameter and booking, and one scoring tree form and counting occurrences of dieback. Each replicate within a site was scored and counted entirely by one individual.

Derivation of Variables for Analysis

Preliminary analysis (FRI Stats Pack Program F1Q4) was made of overall frequency distributions, site by site, for individual scores and combinations of dieback counts on each tree in order to decide what transformations of variables were worth using.

Measurements of d.b.h.o.b. and stem straightness and branch habit quality scores were used in the original form for analysis of variance.

Malformation scores were subjected to a normalising transformation as follows: The scores were transformed to give class intervals corresponding to the intervals (arbitrary units) in a normal distribution between the means of the percentile classes (over both sites pooled) represented by the respective scores. This was achieved by using tne formula

$$x' = (a + x)^b$$

where x' = the transformed variable,

x = the original score,

and a and b were constants chosen empirically to give roughly the desired intervals.

Dieback counts were used to derive alternative variables (measures of dieback) as shown in Table 1.

The general idea was to adopt the transformations where they materially reduced statistical interactions and improved the resolution of family differences. The use of a 0-1 scale was tried because, despite the sacrifice of information, there are corrections available with this scale to give heritability estimates that relate to an underlying continuous scale of variation (Dempster and Lerner, 1950; Van Vleck, 1972).

For the later stages of statistical analysis certain variables were dropped on the basis of early analyses.

Statistical Analysis

Analysis of variance was complicated by several types of imbalance in the classification:

(i) Some progenies (families) being represented only at Kaingaroa.

(ii) Unequal numbers of surviving trees per plot.

(iii) Not all progenies being represented in all block replicates.

(iv) Controls being represented by more than one plot in some replicates at Kaingaroa.

Accordingly, analyses of variance were carried out as follows:

1. Involving those *P. radiata* lots (14 progenies plus two controls that were represented in all reps on both sites (see Table 2A))

The Method of Unweighted Means was used, linking analyses of subclass means (FRI Stats Pack Programs F1P1 and F1Q1) with estimates of within-subclass variance (Program F1P1 on basic data).

2. Involving Tarawera data only (see Table 2B)

Again, the Method of Unweighted Means was used (FRI Stats Pack Programs F1P1 and F1P7 on subclass means).

3. Involving Kaingaroa data only

(i) Hendersons Method I (see Table 2C)

The unadjusted mean squares were obtained using FRI Stats Pack Programs F1P1 and F1Q6. Expectations of mean squares were calculated from subclass numbers using Program KMAT, and variance components estimated using Program F1FX. As can be seen from the Expectations of Mean Squares all F tests are only approximate.

(ii) Least Squares Analysis (FRI Stats Pack Program F1TG)

This gives an exact test for interaction, but biassed tests for main effects in the presence of interaction. It also gives estimates of lot means that are adjusted for rep effects, and rep means that are adjusted for lot effects, but without taking account of interaction.

P. muricata was omitted from these analyses.

Lots were provisionally treated as a random effect; and sites were considered both as a fixed effect and as a random effect, since the appropriate approach was debatable.

Where a lot was represented by more than one plot in a block replicate the plots were pooled. This approximation, which was made to simplify the analysis and to bring it within current computer capacity, presumably gives a slight underestimate of the statistical lots x replicates interaction.

Individual tree heritabilities (h^2) were estimated as follows:

$$\hat{h}^2 \text{ (within sites)} = \frac{4\,\hat{\sigma}^2_f}{\hat{\sigma}^2_f + \hat{\sigma}^2_{rf} + \hat{\sigma}^2_w}$$

$$\hat{h}^2 \text{ (over both sites)} = \frac{4\,\hat{\sigma}^2_f}{\hat{\sigma}^2_f + \hat{\sigma}^2_{fs} + \hat{\sigma}^2_{rf:s} + \hat{\sigma}^2_w}$$

σ^2_{fs} being included in the denominator only if sites are regarded as conforming to a random effect. The use of the coefficient of 4 in the numerator involves assuming that the families represent a random group of half-sib families.

Genetic correlations between two traits ($r_{A_{xy}}$) at a site were estimated as

$$\hat{r}_{A_{xy}} = \frac{\hat{Cov}_{f_{xy}}}{\sqrt{\hat{\sigma}^2_{fx} \cdot \hat{\sigma}^2_{fy}}}$$

where $Cov_{f_{xy}}$ is the between-families covariance between the two traits, estimated from mean cross-products in a manner analogous to the estimation of variance components,

and σ^2_{fx} and σ^2_{fy} are the between-families (lots) components of variance for the respective traits.

Genetic correlations between traits at different sites ($r_{G_{k\ell}}$) were calculated as

$$r_{G_{k\ell}} \frac{r_{k\ell}}{\sqrt{h^2_{\bar{f}k}\, h^2_{\bar{f}\ell}}} \qquad \text{(cf. Burdon, 1977a)}$$

where $r_{k\ell}$ is the phenotypic correlation between family means at sites k and ℓ

and $h^2_{\bar{f}k}$ and $h^2_{\bar{f}\ell}$ are heritabilities (repeatabilities) of family means at the respective sites.

In correlating family performances between the field and in the glasshouse, on one hand, and performances in the field and parent clone monoterpene composition, on the other hand, there was a complication. The clone labelled 870-387 in the archive had essentially the monoterpene of the ortet that was nominally clone 870-385 (Burdon *et al.*, 1977a). In this case, therefore, it was not quite certain which progenies in the inoculation trial corresponded to the progenies of clones 870-385 and 870-387 in the field trial. Accordingly, the correlations were calculated making the two alternative assumptions as to identity. Case A cross-referenced progeny 870-387 in the field with clone 870-387 in the archive and Lot 70 in the inoculation trial (Burdon *et al.*, 1976). Case B cross-referenced progeny 870-387 in the field with clone 870-385 in the archive (Lot 72 being absent from the inoculation trial).

RESULTS

General

At Tarawera the growth was appreciably faster and the incidence of dieback much higher than at Kaingaroa (Tables 3 and 4). Branch habit quality scores and malformation scores, however, were slightly poorer at Kaingaroa, but these latter comparisons are not rigorous.

The distributions of the dieback counts were strongly non-normal (Table 3), even after transformation. In fact the use of transformations did not materially affect the results of analyses of variance (Tables 5, 6, 7, 8, 10, 11). Hence some reservation must attach to most of the analyses of variance and resulting estimates of parameters. The significant site x lot interactions (Table 5) are particularly suspect.

Lot Differences

Clear differences between lots were evident for all variables at Tarawera (Table 7) with good repeatabilities of lot means (Table 8). At Kaingaroa there were clear differences between lots in respect of d.b.h. o.b. and the tree form traits, but not in respect of dieback variables (Tables 10 and 11).

Lot x site interactions were unimportant. Although analyses of variance suggested interactions for dieback variables (Table 5), the use of genetic correlation analysis (Table 13) makes it clear that such interactions were essentially an artifact of the non-normality of the data.

Comparing the controls with the progenies, neither control differed significantly from the progenies as a group in respect of dieback, either at Tarawera or Kaingaroa (Tables 9 and 12). At Tarawera AL 1 was slightly,

but not significantly (P > 0.05) better than R69/854 for all traits. At Kaingaroa AL 1 was significantly (P > 0.05) better than R69/854 in both d.b.h.o.b. and stem straightness. There it was significantly superior to the progenies as a group in d.b.h.o.b., while R69/854 was significantly (P < 0.01) worse than the progenies overall in stem straightness.

With the general lack of clear differences between the controls and the progenies it was deemed unnecessary to segregate the controls for obtaining heritability estimates. In fact, none of the estimated heritabilities (Tables 6, 8 and 11) were very high, the highest values (ca. 0.25) being for stem straightness and branch habit quality.

Inclusion of dieback counts on the laterals, in addition to leader dieback, gave a modest improvement in resolution of lot differences at Tarawera, but taking account of uncertain cases of dieback did not improve resolution. The more elaborate counts tended to show greater effects of replicates (which were confounded with observers) and more lot x replicate interaction.

Interrelationships between Traits in Field Trial

Estimates of intercorrelations between traits (Table 14) suggest that there were no material differences in lot rankings for dieback between the leaders and the laterals. (The Kaingaroa results are too imprecise to be very informative on this point). The expected pattern of strong phenotypic and genetic (between-lot) correlations was observed between malformation at Tarawera and the incidence of dieback at either site (Tables 13 and 14). The negative signs in the listed correlations reflect the fact that malformation was recorded on an inverse scale.

Variances and between-trait covariances for lots and lot means are shown in Table 14, in case it proves worthwhile to rank the families using a multi-trait selection index.

Relationship between Field Performance and Response in Glasshouse

Field performance of progenies and their inoculation responses did not correlate at all satisfactorily (Table 15, Figs 1 & 2), irrespective of assumptions concerning the identity of progenies (viz. Case A vs Case B). In fact, the correlations, which in general were non-significant (P > 0.05), tended to be of the opposite sign to what could be expected. The only significant correlations, between inoculation responses and malformation score, were in the 'wrong' direction and were presumably fortuitous.

In this situation no useful purpose was seen in pursuing estimates of genetic correlations.

Looking at Figs 1 and 2 (in which the expected association would be negative (owing to the nature of the scales used), it can be seen that the performance of the control R69/854, relative to the progenies as a group, was not actually inconsistent between the two studies. Considering the performance of individual progenies, however, even allowing for approximations and some uncertainties as to appropriate estimates of errors of progeny means, there are clearly some important discrepancies between the studies in progeny rankings. This is irrespective of assumptions as to identity of progeny [850]387.

Relationship between Field Performance and Parental Monoterpenes

The correlations in Tables 16 and 17 were calculated between progeny means observed in the field and mean levels of individual monoterpenes in parent clone material kept at FRI Headquarters (for details see Burdon *et al.*, 1977a). There was no convincing evidence of meaningful correlations, the occasional statistically significant correlations being readily attributable to chance in view of the large number of correlations being calculated.

Comparison between P. radiata and P. muricata

Predictably, the *P. radiata* grew considerably faster than the *P. muricata* (Table 18). Also it showed less dieback, although the differences were only significant ($P < 0.05$) at Tarawera. However, the dieback in the *P. muricata* could have been accentuated by deer damage (which was concentrated in this species) at Tarawera. In respect of stem diameter and the tree form traits the *P. muricata* performed much better relative to *P. radiata* at Kaingaroa than at Tarawera, presumably because it was not appreciably affected by dieback and not damaged by deer at Kaingaroa. In fact the *P. muricata* was significantly straighter at Kaingaroa.

On both sites the *P. muricata* showed dramatically less needle cast than the *P. radiata*. At Tarawera *Dothistroma pini* was strongly implicated in the needle cast, but at Kaingaroa *Naemacyclus niveus* appeared to be the prime culprit.

DISCUSSION

The scoring procedure for dieback represented the basis for the study, and the ideal approach was by no means clear. Although it was not explored exhaustively, several lessons seem clear enough. Unless the incidence of dieback is high, it seems inevitable that dieback records will have some undesirable statistical properties which demand caution in the use of analysis of variance. These statistical properties will not readily be overcome by transformation of data. Nevertheless, there appeared to be satisfactory resolution of family differences at Tarawera, although it must be remembered numbers of trees per lot were fairly large and the number of block replicates higher than in most GTI progeny trials.

Refinements of the scoring system appeared to add relatively little to the information obtained in this study. Recording dieback on branches as well as the leader slightly improved resolution of family differences, and could give more satisfactory estimates of between-trait covariances. Recording uncertain cases of dieback achieved virtually nothing, and seemed to introduce an important element of observer bias. With a large number of trees per family, adequate block replication, and the sort of dieback incidence that was observed at Tarawera, there would seem to be no great advantage in recording more than whether or not each tree had definite leader dieback; with fewer trees per family - say, in the region of 25 - it might be worth incorporating counts of dieback on laterals in the measure of dieback occurrence.

However, the ideal situation for genetic studies of dieback resistance would probably be where most trees have multiple occurrences of dieback, so that one could visually rate individuals for the general amount of dieback.

Pattern of Dieback Incidence in Relation to Other Studies

The major and most disturbing result is the conflict between inoculation responses and the dieback figures for lots in the field. This conflict is sharp, since reasonably good resolution of lot differences was obtained in both studies. It is clear-cut in the rankings among the progenies themselves rather than in the comparisons between the controls and progenies.

Several possible explanations must be considered in some detail, although none appears altogether satisfactory:

(i) That inappropriate controls were used in the respective studies.

(ii) That the progeny samples from individual parents differed between the two studies.

(iii) That rankings of genotypes for dieback resistance differ according to environment.

(iv) That rankings for resistance change with age of trees.

(v) That different fungal strains were involved in the two studies, with tree genotypes having resistance that is specific to pathogen strains.

(vii) That certain lots were incorrectly identified at some stage.

The controls were not ideal in that they were of different origin from the stand in which the selection was done, and so neither was necessarily representative of the effective base population. Nevertheless, one control (R69/854) was common to the two studies, and its performance relative to that of the progenies as a group was not actually inconsistent.

The progeny samples used in the field had certain deficiencies, which would mean that they by no means conformed to half-sib progenies of the respective parents. In some cases very few cones were available on the parents, and these cones would not have included consecutive pollination years or consecutive clusters of cones within a pollination season, but this would seem unlikely to have caused radical discrepancies. The seed collections used for the inoculation trial, made five years later, would have come from a more select sample of pollen parents. This could account for a slightly better performance of select material relative to controls, but it cannot account for very different progeny rankings.

It would not be surprising if rankings of progenies for resistance did differ between the field progeny test and the inoculation trial, because it is well recognised that short-cut screening procedures can prove inapplicable to field conditions. What is noteworthy is that the inoculation trial results accord slightly better than the field results with the original circumstance of selection in the field, insofar as the select-parent progenies clearly excelled the controls only in the glasshouse. Logically, this suggests that the glasshouse inoculation conditions might have corresponded better to the conditions at Fentons Mill Flat prior to selection than did conditions in the field progeny trial. This, of course, would mean that any genetic gains in resistance would presumably be very specific to particular sites. But even though dieback was not very prevalent in the progeny trial the Tarawera trial site and Fentons Mill Flat seem very similar. Moreover, dieback incidence differed sharply between the two trial sites without material differences in lot rankings so, all told, the possible explanation seems implausible.

Diagnosis of *Diplodia*-associated dieback is always a problem, since it must be made inductively on the basis of gross visual symptoms combined with proper examination of a very small sample of cases. This problem would have applied alike both in the parent stand and in the progeny trial, while there is no reason to suspect that incorrect diagnosis was an important factor.

Variations in progeny rankings with age of trees, although likely enough in itself, would hardly account for the observed results. The seedlings in the glasshouse were of course much younger than the progeny trial material when it was assessed. Nevertheless, the parents, when selected, were older than the progeny trial at assessment, and the selection was endorsed rather more by the inoculation trial than by the field progeny trial.

Specificity of response of tree genotypes to strain of pathogen, with the presence of several different pathogen strains, is always a possibility, but again it does not provide a convincing explanation for the results. The inoculation trial used fungus spores from a single isolate. This isolate was from "Death Valley" in Tarawera Forest, where the dieback was similar to that at Fentons Mill Flat, although more extreme. The available evidence (Chou, 1977) does not suggest that isolates of *Diplodia* vary much in pathogenicity, although the comparisons were not precise and reflected only the average pathogenicity of an isolate to a sample population of seedling genotypes.

The possibility of identification errors always haunts the experimenter. In this study there was one case where identification was in serious doubt, but it clearly had no bearing on the general picture. The two control lots performed roughly as might be expected in relation to each other and to the progenies, which would argue against any general misidentification, but it is difficult to be entirely confident.

It is clear from Figs 1 and 2 that, even though reasonable repeatabilities of lot means were obtained, much better resolution of lot differences would have been desirable in order to give a precise picture of the extent of the discrepancies between the two studies.

Other Aspects of Results

The pattern of estimated heritabilities is consistent with other results obtained with *P. radiata*, in that stem straightness and quality of branch habit appeared to be more heritable than stem diameter or malformation rating. Dieback, as a trait which shows a threshold effect and has obvious elements of chance in its expression, could not be expected to show a high individual-tree heritability, unless the overall incidence was very high indeed. The initial field selection, since it could be expected to cause greater truncation of between-family variance in high-heritability traits, has probably damped down inherent differences between traits in apparent heritability.

The general lack of lot x site interaction was reassuring, even with two fairly similar trial sites. However, it should be noted that in the case of dieback, which showed undesirable data characteristics, it was necessary to use genetic correlation analysis in place of conventional analysis of variance in order to obtain the correct picture. The genetic correlation analysis could have been pursued in further detail, but this seemed unnecessary.

The lack of correlations between parental monoterpene levels and dieback variables is consistent with the finding of Burdon *et al.* (1977b), although the confused picture of progeny resistance means that the general situation is still not clear.

The seed orchard lot was generally superior to the bulk seed collection, except in the case of the obviously inconclusive figures for dieback at Kaingaroa. Although many of the differences were not statistically significant individually, this gives further confirmation of the efficacy of the main breeding programme.

Comparison of P. muricata and P. radiata

The general growth and form of *P. muricata* was as expected. The deer damage to *P. muricata* at Tarawera may not mean much, since the occasional plots of *P. muricata* would have had the novelty value that tends to attract animals.

It does seem that *P. muricata* is the more susceptible to *Diplodia*-associated shoot dieback, at least on these warm sites. However, it was much more resistant to needle casts.

INDICATIONS FOR FUTURE WORK

The sharp conflicts in results make it very difficult to decide what to do next, if anything.

The progeny trial plantings certainly need thinning within the next year, and despite likely problems of visibility it is recommended that another but less elaborate assessment of dieback be made during this autumn. Few decisions should probably be made until the results of such an assessment are known.

In any case field and glasshouse studies should probably be made of resistance in juvenile clones to infection and dieback, as already prescribed in Pathology Work Plan No. 117, Experiments 4 and 5. (See also addenda to GTI Work Plan No. 96). However, as an adjunct to any such work the possible specificity of clonal responses to different fungal isolates could be studied.

If conflicts in results cannot be resolved it might be appropriate to check the identity of progenies in the field using, say, monoterpene analysis. Even so this may not be very quick or easy, and further groundwork on the technique needs to be done.

Despite reservations concerning the nature of the progeny trial, it is not recommended that any immediate attempt be made to repeat the trial with more recent seed collections and additional control lots. A more promising approach might be control-crossing between the selections.

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TABLE 1: Derivation of dieback variables for analysis

Variable used in analysis	Weighting (W) given to each occurrence				Formula for variable
	Dieback:				
	Leader:		Lateral:		
	definite	doubful	definite	doubtful	
Dldrdef	1	0	0	0	If $\Sigma W = 0$, $x = 0$; $\Sigma W > 0$, $x = 1$
Dldrexp	1	0	0	0	$(\Sigma W)^{0.6}$
Dldrgen	2	1	0	0	ΣW
Edldrgen	2	1	0	0	$(\Sigma W)^{0.65}$
Dlatgen	0	0	2	1	ΣW
Edlatgen	0	0	2	1	$(\Sigma W)^{0.65}$
Alldbkge	10	5	2	1	ΣW
Elldbkge	10	5	2	1	$(\Sigma W)^{0.65}$
Defdbkge	5	0	1	0	$(\Sigma W)^{0.65}$

Dldrdef covers all definite occurrences of leader dieback

Dldrexp represents an empirical normalising transformation of Dldrdef

Dldrgen is a composite score covering both definite and doubtful occurrences of leader dieback

Edldrgen represents an empirical normalising transformation of Dldrgen

Dlatgen is a composite score covering both definite and doubtful occurrences of dieback on laterals

Edlatgen represents an empirical normalising transformation of Dlatgen

Alldbkge is a composite score covering definite and doubtful occurrences of dieback on both leader and laterals

Elldbkge represents an empirical normalising transformation of Alldbkge

Defdbkge (or Dfdbkge) is a composite score covering definite occurrences of dieback on both leader and laterals, subjected to an empirical normalising transformation

TABLE 2: Analysis of Variance Models

M.S.	Source	d.f.	Expectation of mean squares
A. Both sites combined			
1.	†Sites (S)	1	$(^{1}/_{7.167}\ \sigma^2_w + \sigma^2_{fr:s}) + 16\ \sigma^2_{rs} + 12\ \sigma^2_{sf} + 192\ \sigma^2_s$
2.	Lots (F)	15	$(^{1}/_{7.167}\ \sigma^2_w + \sigma^2_{fr:s}) + [12\ \sigma^2_{sf}] + 24\ \sigma^2_f$
3.	S x F	15	$(^{1}/_{7.167}\ \sigma^2_w + \sigma^2_{fr:s}) + 12\ \sigma^2_{sf}$
4.	Reps:sites (R:S)	22	$(^{1}/_{7.167}\ \sigma^2_w + \sigma^2_{fr:s})\ 16\ \sigma^2_{rs}$
5.	F x R:S(Syn. plots)	330	$(^{1}/_{7.167}\ \sigma^2_w + \sigma^2_{fr:s})$
6.	Within plots	2582	$^{1}/_{7.167}\ \sigma^{2*}_w$
B. Tarawera			
	Lots (F)	16	$(^{1}/_{7.078}\ \sigma^2_w + \sigma^2_{fr}) + 12\ \sigma^2_f$
	Reps (R)	11	$(^{1}/_{7.078}\ \sigma^2_w + \sigma^2_{fr}) + 17\ \sigma^2_r$
	F x R (Syn. plots)	176	$(^{1}/_{7.078}\ \sigma^2_w + \sigma^2_{fr})$
	Within plots	1283	$^{1}/_{7.078}\ \sigma^2_w$
C. Kaingaroa			
	Lots (F)	21	$\sigma^2_w + 8.3609\ \sigma^2_{fr} + 1.0613\ \sigma^2_r + 87.9558\ \sigma^2_f$
	Reps (R)	11	$\sigma^2_w + 8.8038\ \sigma^2_{fr} + 161.7227\ \sigma^2_r + 1.0612\ \sigma^2_f$
	F x R (Syn. plots)	208	$\sigma^2_w + 7.9799\ \sigma^2_{fr} - 0.1072\ \sigma^2_r - 0.0561\ \sigma^2_f$
	Within plots	1700	σ^2_w

*Obtained by dividing within plots m.s. by harmonic mean of numbers of trees in plots

†$\hat{F} = 1. \div (3. + 4. - 5.)$

where

σ^2_w = within-plots variance

$\sigma^2_{fr:s}$ = lots x (reps within sites) (syn. plots) variance

$\sigma^2_{r:s}$ = reps within sites variance

σ^2_f = lots variance

σ^2_s = sites variance

σ^2_{fr} = lots x reps variance

Term in square brackets omitted if sites are treated as a fixed effect.

TABLE 3: Frequency distributions for the different variables, site by site

Variable	Bounds of distribution		Class interval									
	Lower	Upper	1	2	3	4	5	6	7	8	9	10
Kaingaroa												
Malftran	-5.03	2.83	21	0	124	0	0	372	277	399	0	748
Dldrdef	0	1	1749	0	0	0	0	0	0	0	0	192
Dldrexp	0	2.30	1749	0	0	0	175	0	15	0	1	1
Dldrgen	0	8	1690	57	171	5	13	0	3	1	0	1
Edldrgen	0	3.48	1690	0	57	0	171	5	13	3	1	1
Dlatgen	0	40	1824	83	16	8	4	4	2	0	0	0
Edlatgen	0	11.00	1548	194	125	41	15	9	3	6	0	0
Alldbkge	0	70	1684	200	35	17	4	0	1	0	0	0
Elldbkge	0	15.82	1528	120	187	65	27	12	1	1	0	0
Dfdbkge	0	10.08	1597	71	179	54	35	3	1	1	0	0
Tarawera												
Malftran	-5.03	2.83	55	0	209	0	0	363	184	266	0	410
Dldrdef	0	1	948	0	0	0	0	0	0	0	0	539
Dldrexp	0	2.30	948	0	0	0	436	0	85	0	17	1
Dldrgen	0	8	836	112	411	24	77	0	9	15	2	1
Edldrgen	0	3.48	836	0	112	0	411	24	77	9	15	3
Dlatgen	0	40	1054	249	93	47	26	10	6	0	0	2
Edlatgen	0	11.00	595	258	342	111	94	57	15	13	0	2
Alldbkge	0	70	809	362	154	91	43	20	3	4	0	1
Elldbkge	0	15.82	556	170	248	268	142	51	43	4	4	1
Dfdbkge	0	10.08	645	123	252	232	145	44	39	3	3	1
Overall												
Malftran	-5.03	2.83	76	0	333	0	0	735	461	665	0	1158
Dldrdef	0	1	2697	0	0	0	0	0	0	0	0	731
Dldrexp	0	2.30	2697	0	0	0	611	0	100	0	18	2
Dldrgen	0	8	2526	169	582	29	90	0	12	16	2	2
Edldrgen	0	3.48	2526	0	169	0	582	29	90	12	16	4
Dlatgen	0	40	2878	332	109	55	30	14	8	0	0	2
Edlatgen	0	11.00	2143	452	467	152	109	66	18	19	0	2
Alldbkge	0	70	2493	562	189	108	47	20	4	4	0	1
Elldbkge	0	15.82	2084	290	435	333	169	63	44	5	4	1
Dfdbkge	0	10.08	2242	194	431	286	180	47	40	4	3	1

TABLE 4: Overall means for *P. radiata* at Kaingaroa and Tarawera (Based on lots that were fully represented at both sites)

Variable	Kaingaroa	Tarawera	Tarawera - Kaingaroa
D.b.h.o.b.	151.02	162.02	11.0 ***
Straightness	5.57	5.77	[0.20] N.S.
Branching	4.69	5.07	[0.38] **
Malf(tran)	0.88	0.17	-0.71 **
Dldrdef ++	0.095	0.365	-0.250 ***
Dldrexp ++	0.099	0.406	-0.307 ***
Dldrgen ++	0.242	0.995	-0.753 ***
Edldrgen ++	0.184	0.702	-0.518 ***
Dlatgen ++	1.000	3.831	-2.831 ***
Edlatgen ++	0.570	1.906	-1.336 ***
Alldbkge ++	2.211	8.798	-6.587 ***
Elldbkge ++	1.052	3.462	-2.410 ***
Dfdbkge ++	0.579	2.038	-1.459 ***

++ denotes high score undesirable
N.S. denotes not significant (P >0.05)
** denotes highly significant (P <0.01)
*** denotes very highly significant (P <0.001)

TABLE 5: F ratio and significance levels in Anova involving both sites

Variable	Sites 1,K d.f.†	Blocks:Sites 22,330 d.f.	Lots		Lots x Sites 15,330 d.f.	Lots x Reps:Sites 330,2582 d.f.
			15,330 d.f.	15,15 d.f.		
D.b.h.o.b.	23.16 ***	4.55 ***	6.27 ***	4.29 **	1.34 N.S.	1.11 N.S.
Straightness	[1.77 N.S.]	8.40 ***	10.87 ***	10.87 ***	<1 N.S.	1.25 **
Branching	[10.69 **]	3.59 ***	10.97 ***	10.97 ***	<1 N.S.	≦1 N.S.
Malftran	15.32 **	3.27 ***	4.76 ***	3.98 **	1.20 N.S.	1.21 *
Dldrdef	82.39 ***	3.03 ***	3.21 ***	1.62 N.S.	1.98 *	1.01 N.S.
Dldrexp	92.39 ***	2.81 ***	2.92 ***	1.59 N.S.	1.83 *	1.70 ***
Dldrgen	116.10 ***	2.41 ***	2.62 ***	1.70 N.S.	1.54 N.S.	1.23 **
Edldrgen	110.73 ***	2.82 ***	3.21 ***	1.71 N.S.	1.88 *	1.01 N.S.
Dlatgen	27.66 ***	8.42 ***	3.91 ***	2.05 N.S.	1.91 *	1.70 ***
Edlatgen	32.26 ***	10.61 ***	3.69 ***	2.03 N.S.	1.82 *	1.68 ***
Alldbkge	76.71 ***	5.30 ***	3.75 ***	1.72 N.S.	2.18 **	1.23 **
Elldbkge	77.80 ***	6.74 ***	3.89 ***	1.74 N.S.	2.29 **	1.25 **
Defdbkge	73.01 ***	6.17 ***	3.95 ***	1.61 N.S.	2.46 **	1.25 **

N.S. denotes Not significant (P >0.05)
* denotes Significant (P <0.05)
** denotes Highly significant (P <0.01)
*** denotes Very highly significant (P <0.001)

†K is variable, but exact values are clearly immaterial

TABLE 6: Estimates of variance components and heritabilities from Anova involving both sites

Variables	$\hat{\sigma}^2_s$	$\hat{\sigma}^2_{r:s}$	$\hat{\sigma}^2_{f(a)}$	$\hat{\sigma}^2_{f(b)}$	$\hat{\sigma}^2_{fs}$	$\hat{\sigma}^2_{fr:s}$	$\hat{\sigma}^2_w$	$\hat{h}^2_{\bar{f}a}$	$\hat{h}^2_{\bar{f}b}$	$\hat{h}^2_{(a)}$	$\hat{h}^2_{(b)}$
D.b.h.o.b.	57.80	22.73	22.51	21.06	2.89	10.19	661.06	0.84	0.79	0.13	0.17
Straightness	0.0151	0.2067	0.1840	0.1840	0	0.0887	2.57124	0.91	0.91	0.26	0.26
Branching	0.0655	0.0586	0.1501	0.1501	0	0	2.7175	0.91	0.91	0.21	0.21
Malftran	0.2380	0.0893	0.0988	0.0937	0.1028	0.1097	3.7291	0.79	0.75	0.10	0.09
Dldrdef	0.03583	0.00268	0.00194	0.00108	0.00172	0.00025	0.14924	0.69	0.38	0.051	0.028
Dlatgen	3.8617	1.3826	0.3614	0.2840	0.2261	1.2294	12.5524	0.74	0.42	0.10	0.08
Edlatgen	0.8647	0.2792	0.0520	0.0362	0.0362	0.1881	1.9827	0.73	0.51	0.09	0.06
Alldbkge	21.4109	2.2519	0.9596	0.5505	0.8238	1.5481	48.9916	0.73	0.42	0.07	0.04
Elldbkge	2.8659	0.3225	0.1083	0.0622	0.09211	0.1821	5.1421	0.74	0.43	0.08	0.05
Defdbkge	1.0489	0.1185	0.0450	0.0228	0.04557	0.07321	2.1092	0.75	0.38	0.08	0.04

$\hat{\sigma}^2_{f(a)}$ assumes sites are a fixed effect

$\hat{\sigma}^2_{f(b)}$ assumes sites are a random effect

$$\hat{h}^2_{\bar{f}(a)} = \frac{\hat{\sigma}^2_f}{\hat{\sigma}^2_f + \hat{\sigma}^2_{fr:s}/_{24} + \hat{\sigma}^2_w/_{7.167}} = \frac{1 - F}{F}$$

$$\hat{h}^2_{\bar{f}(b)} = \frac{\hat{\sigma}^2_f}{\hat{\sigma}^2_f + \hat{\sigma}^2_{fs}/_{2} + \hat{\sigma}^2_{fr:s}/_{24} + \hat{\sigma}^2_w/_{7.167}} = \frac{1 - F}{F}$$

$$\hat{h}^2_{(a)} = \frac{4\,\hat{\sigma}^2_f}{\hat{\sigma}^2_f + \hat{\sigma}^2_{fr:s} + \hat{\sigma}^2_w}$$

$$\hat{h}^2_{(b)} = \frac{4\,\hat{\sigma}^2_f}{\hat{\sigma}^2_f + \hat{\sigma}^2_{fs} + \hat{\sigma}^2_{fr:s} + \hat{\sigma}^2_w}$$

TABLE 7: F ratios and significance levels at Tarawera

Variable	Reps 11,176 d.f.	Lots 15,176 d.f.	Reps x Lots 176,1283 d.f.
D.b.h.o.b.	3.81 ***	4.40 ***	<1 N.S.
Straightness	4.88 ***	4.64 ***	1.37 **
Branching	1.77 *	4.46 ***	<1 N.S.
Malftran	1.81 *	3.06 ***	1.17 N.S.
Dldrdef	3.69 ***	2.79 **	<1 N.S.
Dldrexp	3.42 ***	2.48 **	<1 N.S.
Dldrgen	2.56 **	2.56 **	<1 N.S.
Edldrgen	2.87 ***	2.87 **	<1 N.S.
Dlatgen	8.25 ***	3.00 **	2.56 ***
Edlatgen	9.74 ***	2.93 ***	1.61 **
Alldbkge	4.66 ***	3.18 ***	1.13 N.S.
Elldbkge	5.14 ***	3.51 ***	1.15 N.S.
Dfdbkge	5.78 ***	3.68 ***	1.11 N.S.

TABLE 8: Estimates of variance components and heritabilities at Tarawera

Variable	Statistic					
	$\hat{\sigma}^2_f$	$\hat{\sigma}^2_r$	$\hat{\sigma}^2_{fr}$	$\hat{\sigma}^2_w$	$\hat{h}^2_{\bar{f}}$	$\hat{h}^2$
D.b.h.o.b.	30.3	17.7	0	782.0	0.77	0.15
Straightness	0.149	0.118	0.141	2.665	0.78	0.20
Branching	0.116	0.018	0	2.986	0.78	0.15
Malftran	0.124	0.035	0.107	4.351	0.67	0.11
Dldrdef	0.00459	0.00487	0	0.22167	0.64	0.08(0.30)†
Dlatgen	0.785	2.009	1.814	20.506	0.67	0.14
Alldbkge	2.412	2.412	1.485	83.643	0.69	0.13
Elldbkge	0.263	0.306	0.1609	7.770	0.72	0.13
Dfdbkge	0.115	0.145	0.0531	3.257	0.73	0.13

$$\hat{h}^2_{\bar{f}} = \frac{\hat{\sigma}^2_f}{\hat{\sigma}^2_f + \hat{\sigma}^2_{fr/12} + \hat{\sigma}^2_{w/12x7.078}} = \frac{\hat{\sigma}^2_f}{m.s._{\bar{f}} \div 12}$$

$$\hat{h}^2 = \frac{4\,\hat{\sigma}^2_f}{\hat{\sigma}^2_f + \hat{\sigma}^2_{fr} + \hat{\sigma}^2_w}$$

†Adjusted to continuous underlying scale of variation

TABLE 9: Lot means at Tarawera

Lot	Variable												
	D.b.h. o.b.	Straight -ness	Branch -ing	Malftran	Dldrdef	Dldrexp	Dldrgen	Edldrgen	Dlatgen	Edlatgen	Alldbkge	Elldbkge	Dfdbkge
378	151	6.14	5.00	-0.46	0.49	0.54	1.25	0.90	4.69	2.20	10.98	4.20	2.52
380	160	5.99	5.22	0.06	0.32	0.36	0.92	0.64	3.69	1.92	8.30	3.39	1.94
381	154	5.24	4.50	0.59	0.42	0.46	1.08	0.75	3.94	2.04	9.35	3.72	2.25
383	166	6.42	5.54	-0.00	0.27	0.30	0.80	0.58	2.47	1.36	6.51	2.69	1.44
386	159	5.43	5.46	0.63	0.34	0.38	0.89	0.63	3.68	1.85	8.16	3.20	1.92
387	156	6.41	5.67	0.80	0.28	0.31	0.71	0.50	5.10	2.37	8.67	3.37	2.05
388	156	5.80	4.93	0.11	0.39	0.43	1.06	0.72	4.07	1.92	9.40	3.56	2.13
391	161	5.87	5.03	0.77	0.28	0.30	0.73	0.53	2.78	1.57	6.44	2.71	1.58
392	160	5.10	4.58	0.06	0.32	0.39	0.99	0.65	3.25	1.68	8.23	3.23	1.91
393	163	5.83	5.13	-0.00	0.44	0.48	1.19	0.87	3.98	1.95	9.95	3.91	2.28
394	161	5.63	4.88	0.02	0.28	0.33	0.86	0.58	3.23	1.72	7.56	3.12	1.82
395	172	6.26	5.73	0.82	0.19	0.22	0.61	0.43	2.45	1.33	5.51	2.39	1.32
399	164	5.91	4.37	-0.15	0.40	0.44	1.08	0.77	2.98	1.68	8.39	3.48	2.03
400	171	6.28	5.28	-0.25	0.38	0.44	1.07	0.75	4.34	2.11	9.71	3.69	2.21
401	173	5.14	4.90	-0.50	0.50	0.55	1.32	0.92	6.76	3.02	13.40	4.92	2.96
AL 1	166	5.58	5.09	0.38	0.34	0.39	1.00	0.70	2.96	1.51	7.99	3.19	1.80
R69/ 854	160	5.28	4.95	-0.07	0.43	0.47	1.17	0.87	4.53	2.10	10.42	3.94	2.29
Site means	161.9	5.79	5.08	0.166	0.363	0.404	0.989	0.698	3.82	1.91	8.77	3.46	2.03
LSD	8.29	0.577	0.510	0.682	0.141	0.159	0.338	0.247	1.74	0.664	2.93	0.90	0.58

There are no significant differences

- Between the two controls
- Between either control and the selections as a whole

TABLE 10: F ratios and approximate significance levels at Kaingaroa

Variable	Effect			
	Reps (11,208 d.f.)	Lots (21,208 d.f.)	Reps x Lots (a) (208,1700 d.f.)	Reps x Lots (b) (208,1700 d.f.)
D.b.h.o.b.	6.81 ***	2.88 ***	1.48	1.56 ***
Straightness	17.61 ***	6.38 ***	1.15	1.15 N.S.
Branching	7.10 ***	6.08 ***	1.08	1.09 N.S.
Malftran	3.47 **	2.68 ***	1.21	1.24 *
Dldrdef	1.87 *	1.54 ($P \geq 0.05$)	1.02	1.03 N.S.
Dldrexp	1.86 ($P \cong 0.05$)	1.57 ($P \cong 0.05$)	1.03	1.04 N.S.
Dldrgen	3.24 ***	1.57 ($P \cong 0.05$)	1.09	1.09 N.S.
Edldrgen	3.80 ***	1.47 N.S.	1.07	1.07 N.S.
Dlatgen	13.84 ***	1.40 N.S.	2.12	2.12 ***
Edlatgen	17.40 ***	1.34 N.S.	2.11	2.11 ***
Alldbkge	9.20 ***	1.33 N.S.	1.70	1.71 ***
Elldbkge	11.78 ***	1.32 N.S.	1.64	1.64 ***
Dfdbkge	8.18 ***	1.42 N.S.	1.63	1.62

(a) Denotes F ratio obtained from unadjusted mean squares

(b) Exact test for interaction in least squares ANOVA

NOTE: Where interaction was negligible the tests for main effects in least squares ANOVA gave essentially the same results as presented here.

TABLE 11: Estimates of variance components and heritabilities at Kaingaroa

Variable	Statistic					
	$\hat{\sigma}^2_f$	$\hat{\sigma}^2_r$	$\hat{\sigma}^2_{fr}$	$\hat{\sigma}^2_w$	$\hat{h}^2_{\bar{f}}$	h^2
D.b.h.o.b.	17.81	30.54	35.71	579.34	0.63	0.11
Straightness	0.1695	0.2903	0.0526	2.4629	0.82	0.25
Branching	0.1520	0.0990	0.0277	2.4554	0.83	0.23
Malftran	0.0703	0.1715	0.0872	3.1470	0.60	0.09
Dldrdef	0.00055	0.00048	0.00027	0.08795	0.35	0.025 (0.09) †
Dlatgen	0.0363	0.8293	0.7612	5.3180	0.22	0.024
Elldbkge	0.0119	0.3174	0.2379	2.9213	0.17	0.015
Dfdbkge	0.00740	0.08152	0.0903	1.1367	0.25	0.024

$$\hat{h}^2 = \frac{4\,\hat{\sigma}^2_f}{\hat{\sigma}^2_f + \hat{\sigma}^2_{fr} + \hat{\sigma}^2_w}$$

$$h^2_{\bar{f}} = \frac{\hat{\sigma}^2_f}{(M.S._f) \div 87.96}$$ (applicable to unadjusted lot means)

†Adjusted to continuous underlying scale of variation

TABLE 12: Lot means at Kaingaroa, adjusted for Rep effects

Lot (Progeny/ seedlot)	Variable													Missing subclasses
	D.b.h.o.b. (mm)	Straightness	Branching	Malftran	Dldrdef	Dldrexp	Dldrgen	Edldrgen	Dlatgen	Edlatgen	Alldbkge	Elldbkge	Dfdbkge	
378	141	5.73	4.27	0.34	0.175	0.186	0.43	0.32	1.35	0.76	3.49	1.60	0.95	0
379	146	5.27	5.54	0.93	0.118	0.142	0.40	0.26	1.13	0.63	3.11	1.28	0.69	6
380	146	5.91	4.73	1.02	0.070	0.070	0.16	0.13	0.75	0.44	1.56	0.76	0.45	2
381	144	5.38	4.27	1.06	0.094	0.099	0.24	0.18	0.55	0.34	1.75	0.85	0.43	0
383	150	5.84	4.89	0.75	0.151	0.167	0.40	0.27	1.05	0.62	3.04	1.36	0.81	0
384	158	5.10	4.27	0.64	0.082	0.132	0.40	0.22	0.52	0.35	2.51	0.99	0.59	7
385	155	5.30	4.92	0.79	0.172	0.178	0.41	0.30	1.45	0.72	3.49	1.48	0.85	0
386	152	5.22	5.14	1.30	0.062	0.062	0.17	0.14	0.83	0.53	1.67	0.90	0.47	0
387	154	6.29	5.48	1.37	0.065	0.065	0.14	0.11	0.72	0.45	1.42	0.72	0.40	0
388	150	5.41	4.79	0.70	0.128	0.128	0.32	0.25	0.57	0.36	2.18	1.02	0.52	0
390	148	5.46	4.69	0.76	0.056	0.057	0.15	0.13	0.16	0.17	0.93	0.56	0.20	4
391	148	5.53	4.07	0.81	0.071	0.071	0.18	0.14	1.47	0.81	2.35	1.15	0.59	0
392	149	4.51	4.33	1.12	0.121	0.121	0.33	0.27	0.64	0.44	2.30	1.13	0.54	0
393	153	5.85	4.88	0.97	0.119	0.125	0.27	0.20	1.03	0.58	2.39	1.10	0.67	0
394	152	5.68	4.26	0.60	0.022	0.022	0.08	0.07	0.79	0.46	1.17	0.64	0.32	0
395	159	6.20	5.18	1.21	0.085	0.090	0.22	0.17	0.93	0.56	2.03	1.00	0.55	0
396	145	5.72	4.33	0.40	0.111	0.121	0.26	0.19	1.11	0.56	2.43	1.04	0.62	4
397	148	5.39	3.90	0.70	0.065	0.071	0.17	0.13	1.11	0.58	1.97	0.89	0.51	0
400	157	5.75	5.15	0.16	0.066	0.072	0.20	0.15	1.08	0.60	2.08	1.01	0.54	0
401	152	4.75	4.81	0.16	0.085	0.085	0.20	0.16	2.26	1.06	3.27	1.45	0.83	0
AL 1	158	5.62	4.88	1.19	0.127	0.127	0.28	0.22	0.79	0.47	2.21	1.07	0.62	0
R69/854	148	4.84	4.69	0.60	0.090	0.098	0.26	0.19	0.77	0.45	2.06	0.93	0.51	0
Effects														
AL 1	7.73	0.13	0.17	0.30	0.030	0.023	0.02	0.03	-0.16	-0.08	-0.04	0.03	0.04	
R69/854	-2.56	-0.64	-0.02	-0.29	-0.007	-0.006	0.00	0.00	-0.19	-0.09	-0.17	-0.11	-0.07	
LSD (approx)	8.69	0.500	0.483	0.089	-	-	-	-	-	-	-	-	-	

Significance of comparisons involving controls

*	*	N.S.	N.S.	Between the controls
*	N.S.	N.S.	N.S.	Between AL 1 and progenies
N.S.	**	N.S.	N.S.	Between R69/854 and progenies

$$t_{p=0.05} \simeq \sqrt{\frac{M.S._{rf}}{88} + \frac{\sigma_f^2}{20}} \times 1.96$$

TABLE 13: Estimated correlations between mean levels of dieback in lots at Tarawera and Kaingaroa respectively

A. Phenotypic correlations between lot means

Tarawera variables	Kaingaroa variables						$h^2_{\bar{f}}$
	D.b.h.o.b.	Malftran	Dldrdef	Dlatgen	Elldbkge	Dfdbkge	
D.b.h.o.b.	0.68 **	0.05	-0.22	0.47	0.15	0.18	0.77
Malftran	0.30	0.61 **	-0.27	-0.47	-0.51 *	-0.53 *	0.61
Dldrdef	-0.46	-0.29	0.31	0.37	0.42	0.43	0.64
Dlatgen	-0.11	0.01	-0.01	0.51 *	0.27	0.28	0.67
Elldbkge	-0.30	-0.19	0.16	0.49	0.38	0.40	0.72
Dfdbkge	-0.30	-0.17	0.12	0.47	0.34	0.35	0.73
$\hat{h}^2_{\bar{f}}$	0.63	0.60	0.35	0.36	0.17	0.25	-
B. Genetic correlations							
D.b.h.o.b.	0.98	0.07	-0.42	0.87	0.41	0.41	
Malftran	0.46	0.96	-0.56	-0.96	-1.51	-1.29	
Dldrdef	-0.72	-0.47	0.65	0.77	1.27	1.08	
Dlatgen	-0.17	0.02	-0.02	1.04	0.80	0.68	
Elldbkge	-0.45	-0.29	0.32	0.96	1.09	0.94	
Dfdbkge	-0.45	-0.26	0.24	0.92	0.97	0.82	

TABLE 14: Estimates of genetic and phenotypic variances and correlations among lots at Tarawera. Variances are shown on the diagonals of matrices, covariances above diagonals and correlations below

	D.b.h.o.b.	Straight.	Br qual	Malftran	Dldrdef	Dlatgen	Alldbkge
Genetic							
D.b.h.o.b.	30.3	0.109	0.386	-0.423	-0.086	-	-
Straight.	0.05	0.149	0.094	0.0112	-0.0142	-	-
Br qual	0.21	0.71	0.116	0.0497	-0.0143	-	-
Malftran	-0.22	0.08	0.41	0.124	-0.0179	-	-
Dldrdef	-0.23	-0.54	-0.62	-0.75	0.0046	0.054	0.106
Dlatgen	-	-	-	-	0.90	0.785	1.341
Alldbkge	-	-	-	-	1.01	0.97	2.412
Phenotypic							
D.b.h.o.b.	39.2	0.244	0.587	-0.401	-0.103	-	-
Straight.	0.09	0.192	0.106	0.0219	-0.015	-	-
Br qual	0.24	0.63**	0.150	0.065	-0.0171	-	-
Malftran	-0.15	0.12	0.41	0.184	0.0245	-	-
Dldrdef	-0.19	-0.39	-0.52*	-0.68**	0.00715	0.063	0.145
Dlatgen	-	-	-	-	0.69	1.177	1.848
Alldbkge	-	-	-	-	0.91	0.91	3.520

* denotes significant ($P < 0.05$)
** denotes highly significant ($P < 0.01$)
) for phenotypic correlations

Phenotypic variances (σ^2_p) are estimated as

$$\hat{\sigma}^2_p = m.s._{\bar{f}} \div 12$$

where $m.s._{\bar{f}}$ = families mean square calculated from subclass means.

TABLE 15: Field vs glasshouse correlations, involving progeny means

Glasshouse response variable	Dieback variable						$h^2_{\bar{f}}(a)$ †	$h^2_{\bar{f}}(b)$ ††
	D.b.h.o.b.	Malftran	Dldrdef	Dlatgen	Elldbkge	Dfdbkge		
CASE B				Kaingaroa				
Infection	-0.24	0.50	-0.40	-0.03	-0.23	-0.22	0.66	0.58
Dbk	-0.32	0.55	-0.30	0.18	-0.10	-0.10	0.76	0.60
Dbk ratio	-0.38	0.50	-0.15	0.27	-0.01	-0.01	-	-
Score d ‡‡	0.26	-0.50	0.33	-0.12	0.13	0.13	0.80	0.60
CASE A								
Infection	-0.13	0.32	-0.28	-0.24	-0.21	-0.25	0.66	0.58
Dbk	-0.21	0.32	-0.12	-0.03	-0.08	-0.10	0.76	0.60
Dbk ratio	-0.30	0.28	0.05	0.11	0.04	0.01	-	-
Score d ‡‡	-0.15	-0.28	0.19	0.08	0.10	0.13	0.80	0.67
$h^2_{\bar{f}}$	0.77	0.67	0.64	0.67	0.72	0.73	-	-
CASE B				Tarawera				
Infection	0.00	0.45	-0.41	-0.16	-0.41	-0.33	0.66	0.58
Dbk	-0.09	0.53*	-0.21	-0.08	-0.26	-0.19	0.76	0.60
Dbk ratio	-0.24	0.53*	0.05	0.02	-0.03	0.02	-	-
Score d ‡‡	0.07	-0.49	0.31	0.07	0.30	0.23	0.80	0.67
CASE A								
Infection	0.00	0.25	-0.41	-0.16	-0.41	-0.33	0.66	0.58
Dbk	-0.09	0.27	-0.21	-0.08	-0.26	-0.19	0.76	0.60
Dbk ratio	-0.25	0.31	0.06	0.03	0.02	0.03	-	-
Score d ‡‡	0.07	-0.26	0.31	0.07	0.30	0.23	0.80	0.67
$h^2_{\bar{f}}$	0.63	0.60	0.35	0.36	0.17	0.25	-	-

† $h^2_{\bar{f}}(a)$ assumes fixed effects in inoculation trial

†† $h^2_{\bar{f}}$ assumes random effects in inoculation trial

‡‡ Reverse scale to infection/dieback incidence.

TABLE 16: Correlations between parental monoterpene levels (% total monoterpenes in cortical oleoresin) and mean incidence of dieback in progenies in field planting at Tarawera (13 d.f.)

Variable	Monoterpene (see Burdon *et al.*, 1977a)										h^2_f
	α-pinene	Camphene	β-pinene	Sabinene	Δ^3-carene	Myrcene	Limonene	β-phellendrene	γ-terpinene	Terpinolene	
CASE A											
D.b.h.o.b.	-0.11	0.02	-0.02	0.21	0.36	0.12	-0.36	-0.32	0.67***	0.30	0.77
Malftran	0.31	0.25	-0.11	-0.17	0.02	-0.07	-0.16	0.27	-0.25	-0.17	0.67
Dldrdef	-0.07	0.02	0.06	0.35	-0.26	-0.47	0.02	-0.15	0.17	0.31	0.64
Dlatgen	0.10	0.54*	0.09	0.11	-0.34	-0.44	-0.00	0.21	-0.19	0.03	0.68
Elldbkge	-0.02	0.25	0.09	0.25	-0.31	-0.41	0.06	-0.05	0.00	0.18	0.72
Dfdbkge	0.00	0.28	0.10	0.22	-0.34	-0.42	0.05	-0.01	-0.03	0.16	0.73
CASE B											
D.b.h.o.b.	0.02	0.29	-0.04	0.20	0.14	0.22	-0.37	-0.18	0.63*	0.27	0.77
Malftran	0.10	-0.13	-0.08	-0.15	0.31	-0.26	-0.14	-0.02	0.02	-0.10	0.67
Dldrdef	0.05	0.27	0.04	0.34	-0.40	-0.25	0.02	-0.02	-0.05	0.28	0.64
Dlatgen	-0.06	0.29	0.12	0.13	-0.08	-0.51	0.01	-0.03	0.03	0.09	0.68
Elldbkge	-0.01	0.32	0.09	0.25	-0.30	-0.30	0.06	-0.03	-0.02	0.19	0.72
Dfdbkge	-0.01	0.30	0.10	0.23	-0.28	-0.33	0.05	-0.03	-0.03	0.17	0.73
h^2_c	0.98	0.98	0.96	0.98	0.95	0.91	0.98	0.99	0.89	0.98	-

h^2_c = repeatability of clonal means

TABLE 17: Correlations between parental monoterpene levels (% total monoterpenes in cortical oleoresin) and mean incidence of dieback in progenies in field planting at Kaingaroa (19 d.f.)

Variable	Monoterpene										$h^2_{\bar{f}}$
	α-pinene	Camphene	β-pinene	Sabinene	Δ^3-carene	Myrcene	Limonene	β-phellendrene	γ-terpinene	Terpinolene	
CASE A											
D.b.h.o.b.	-0.15	-0.17	-0.28	-0.17	0.29	0.19	-0.03	0.21	0.23	-0.11	0.63
Malftran	-0.17	-0.08	-0.06	0.24	0.06	-0.34	0.07	-0.24	0.08	0.27	0.60
Dldrdef	0.07	0.25	-0.10	-0.07	-0.15	0.15	-0.12	0.59*	-0.32	-0.11	0.35
Dlatgen	0.05	0.36	-0.10	0.15	-0.01	-0.04	-0.18	0.09	0.20	0.15	0.22
Elldbkge	0.11	0.39	-0.10	0.05	-0.15	0.25	-0.21	0.45*	-0.07	0.01	0.17
Dfdbkge	0.06	0.30	-0.16	0.01	-0.07	0.21	-0.18	0.46*	-0.04	-0.01	0.25
CASE B											
D.b.h.o.b.	-0.06	-0.03	-0.29	-0.18	0.18	0.28	-0.04	0.31	0.08	-0.14	0.63
Malftran	-0.02	0.15	-0.09	0.22	-0.16	-0.22	0.05	0.00	-0.14	0.19	0.60
Dldrdef	-0.01	0.12	-0.09	-0.06	-0.05	0.09	-0.11	0.41	-0.18	-0.08	0.35
Dlatgen	-0.02	0.24	-0.09	0.16	0.09	-0.11	-0.17	-0.02	0.28	0.18	0.22
Elldbkge	-0.01	0.19	-0.08	0.06	0.01	0.16	-0.20	0.23	0.10	0.06	0.17
Dfdbkge	-0.04	0.14	-0.15	0.02	0.06	0.14	-0.17	0.26	0.10	0.04	0.25
$h^2_{\bar{c}}$	0.98	0.98	0.96	0.98	0.95	0.91	0.98	0.99	0.89	0.98	-

$h^2_{\bar{c}}$ = repeatability of clonal means

TABLE 18: Comparisons between *P. radiata* and *P. muricata*

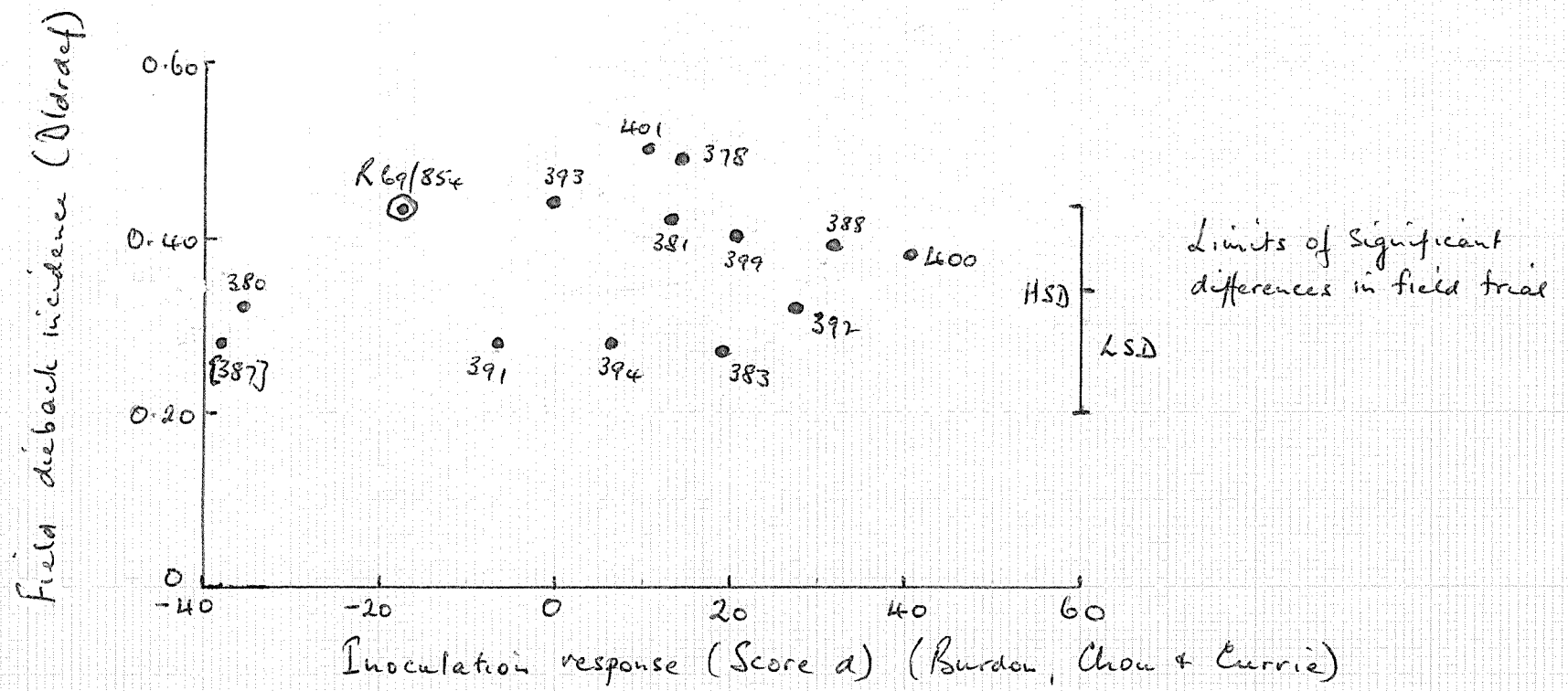
Variable	Site							
	Tarawera†				Kaingaroa			
	P.rad.	*P.mur.*	Diff.	P	*P.rad.*‡	*P.mur.*	Diff.	P
D.b.h.o.b.	162.0	97.8	64.2	***	153.7	117.6	36.1	***
Straightness	5.77	5.85	-0.08	N.S.	5.61	6.56	-1.05	*
Branching	5.07	4.67	0.40	N.S.	4.80	4.55	0.25	N.S.
Malftran	0.17	-0.89	1.06	**	0.84	1.05	-0.21	N.S.
Dldrdef‡‡	0.36	0.71	0.35	***	0.08	0.12	0.04	N.S.
Dlatgen‡‡	3.83	7.41	3.58	*	0.66	0.92	0.26	N.S.
Elldbkge‡‡	8.80	18.90	9.10	***	1.76	2.56	0.80	N.S.
Dfdbkge‡‡	2.04	3.85	1.81	***	0.46	0.66	0.20	N.S.

†Some *P. radiata* familes missing

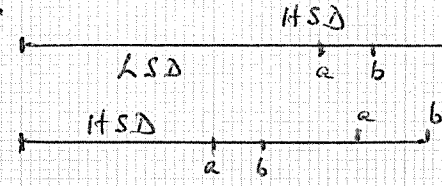
‡Based on means of block means (adjusted for missing subclasses) for those blocks in which *P. muricata* was represented

‡‡ High score undesirable

FIG 1 Plot of field performance vs performance in glasshouse inoculation trial — Dieback on leaders only, at Tarawera

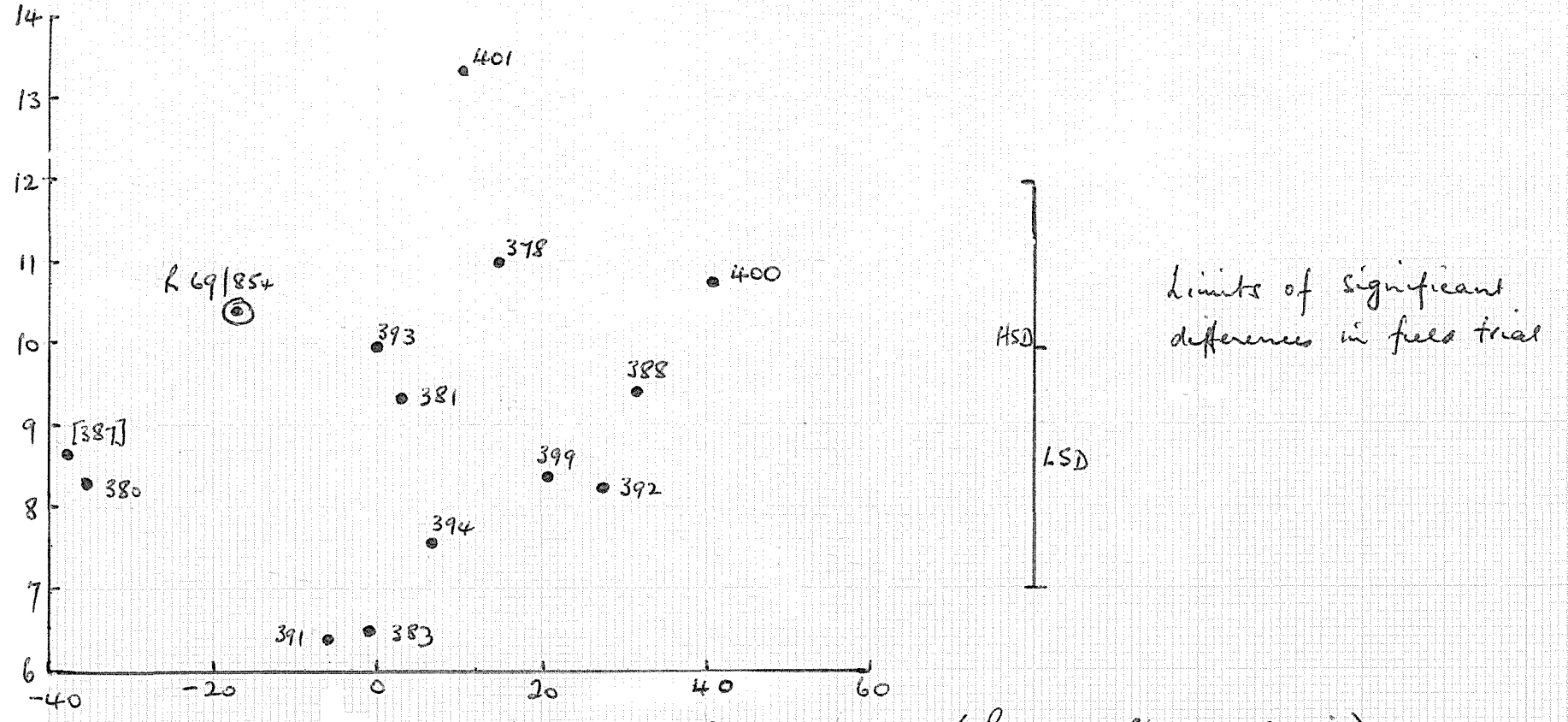


Approximate limits of significant difference in inoculation response

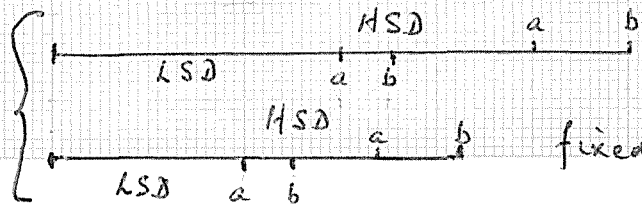


a - between control

FIG 2. Plot of field performance vs performance in glasshouse inoculation trial — Diebak overall at Tarawera



Approximate limits of significant differences in inoculation response



random effects model

fixed effect model.

a - between control and a progeny

b - between two progenies