



**BROAD AND NARROW SENSE HERITABILITIES IN A CLONED OPEN
POLLINATED *EUCALYPTUS GRANDIS* BREEDING POPULATION**

CYNTHIA LOUISE SNEDDEN

THESIS SUBMITTED IN PARTIAL FULFILMENT
OF THE REQUIREMENTS FOR
THE DEGREE OF MASTER OF SCIENCE IN
THE DEPARTMENT OF GENETICS IN
THE FACULTY OF NATURAL AND AGRICULTURAL SCIENCES
UNIVERSITY OF PRETORIA

SUPERVISOR: PROFESSOR C.Z. ROUX
CO-SUPERVISOR: DR. S.D. VERRYIN

2001

DECLARATION

I the undersigned hereby declare that the work contained in this thesis is my own original work and has not previously in its entirety or in part, been submitted to any other university.

Signature: 

Date: *02/08/2001*

SUMMARY

Genetic variances and heritabilities in a cloned *Eucalyptus grandis* breeding population of families derived from open pollinated selections were estimated and the results are presented. The genetic variance was partitioned into additive and non-additive genetic variance components that allowed the estimation of broad and narrow sense heritabilities. Predicted gains for breeding and production population options are discussed.

The magnitude of the coefficient of relationship between sibs was shown to have a considerable impact on the estimate of variance components and the importance of understanding the level of relatedness in the population is highlighted.

Growth traits (volume, diameter at breast height/DBH, height), stem form and disease tolerance were assessed at 38 and 66 months in each of the three separate trials established as sub-populations of the breeding population. The additive genetic variance was the largest proportion of genetic variance for the growth traits (84% for volume, 94% for height and 74% for DBH), whereas the proportion of non-additive genetic variance was notably higher for stem form and disease tolerance (37% and 46% respectively). The growth traits and stem form are, economically, the most important traits and a breeding strategy that exploits the additive genetic variation by selection to increase the frequency of the alleles causing the desirable genotypes is appropriate. The higher proportion of non-additive genetic variance for disease does, however, suggest higher gains (compared with the afore mentioned strategy of selection for general combining ability) will be achieved by exploiting the non-additive variance by for example, selection for specific combining ability, using inbred lines, clones.

The composition of the genetic variance was investigated separately in the F1 and F2 families to obtain an indication of whether or not there was a change in proportion of non-additive and additive genetic variance over the two generations. A notably larger proportion of non-additive variance was found for the growth traits and stem form among the F2 families. This is probably due to the reduction in additive variance through selection for these traits in the previous generations. No

selection for disease took place in earlier generations and the proportion of non-additive genetic variance for this trait remains approximately the same over both generations. These results may indicate that with advanced generations of breeding in this population, that gains achieved through selection for additive variance will decline compared with that achieved in previous generations. A strategy for future generations that exploits non-additive variance may be appropriate.

A high proportion of error variance was estimated and in situations such as these, cloning is particularly beneficial as is shown by the high clone mean heritabilities estimated in these trials. High mortality, resulting in fewer ramets per clone, erodes the benefit of cloning in these trials.

The predicted gains showed the benefit of the cloned breeding population both in terms of breeding population gains and production population gains. Reducing the breeding cycle by bulking up clones faster will also increase gains per year. High gains in the production population were predicted, particularly for the selection of tested clones for deployment, which can be done at the same time as selections are made for the next generation. The benefit of the cloned population was therefore shown to be twofold, namely increasing the accuracy of within family selection and increasing the gains in the rapid deployment of tested clones and therefore facilitating the faster realisation of predicted gain in the plantation.

Keywords: Broad sense heritability, narrow sense heritability, cloned breeding population, additive genetic variation, non-additive genetic variation, *Eucalyptus grandis*.

OPSOMMING

Genetiese variansies en oorerflikhede in 'n gekloonde *Eucalyptus grandis* teelpopulasie van families verkry van af oopbestuifde seleksies, is bereken en die resultate word aangebied. Die genetiese variansies is opgedeel in additiewe en nie-additiewe komponente wat die skatting van breë en eng sin oorerflikhede moontlik maak. Voorspelde vordering vir die teel- en produksiepopulasies word ook bespreek.

Die grootte van die koëffisiënt van die verwantskap tussen sibbe blyk redelike groot invloed op die skatting van die variansie komponente te hê en dit is dus belangrik om die mate van verwantskap in die populasie te verstaan.

Groei-eienskappe (volume, deursnit op borshoogte (DBH) en hoogte), stamvorm en weerstand teen siektes is op 38 en 66 maande in elkeen van die onderskeie proewe gemeet. Die additiewe genetiese variansie was die grootste proporsie van die genetiese variansie vir die groei-eienskappe (84% vir volume, 94% vir hoogte en 74% vir DBH). Die proporsie nie-additiewe genetiese variansie was merkbaar hoër vir stamvorm en siekteweerstand (37% en 46% onderskeidelik). Die groei-eienskappe en stamvorm is, ekonomies gesien, die belangrikste eienskappe en 'n teelstrategie wat die additiewe genetiese variansie ontgin deur seleksie om die frekwensie van "allele" wat die gunstige genotipes tot gevolg het te verhoog, is geskik. Die hoër proporsie van nie-additiewe genetiese variansie vir siekteweerstand wys dat 'n hoër wins gemaak sal word (in vergelyking met die bogenoemde strategie van seleksie vir algemene kombineringsvermoë) deur gebruik te maak van nie-additiewe variansie.

Die samestelling van genetiese variansie is afsonderlik in die F1 en F2 families ondersoek om 'n indikatie te kry of daar verskille tussen die proporsie nie-additiewe en additiewe variansies vir die verskillende generasies, bestaan. 'n Merkbare groter proporsie van nie-additiewe variansie is vir die groei-eienskappe en stamvorm in die F2 families gevind. Dit is moontlik te wyte aan die vermindering in additiewe variansie weens seleksie vir hierdie eienskappe in die vorige generasie. Geen seleksie vir siekteweerstand het in die vorige generasies plaasgevind nie en daarom kan dit wees dat die proporsie nie-additiewe genetiese variansie vir hierdie eienskap nie-merkbaar tussen



die twee generasies verskil nie. Hierdie resultate kan moontlik daarop dui dat, in gevorderde generasies van hierdie teelpopulasie die vordering deur seleksie vir additiewe variansie sal afneem in verhouding tot die vordering verkry deur seleksie in die vorige generasies. 'n Strategie vir die seleksie van toekomstige generasies wat die nie-additiewe variansie gebruik mag dan toepasliker wees.

'n Hoë proporsie vir die oorblywende foutvariensie was beraam en in sulke gevalle kan klonering hoogs voordelig wees, soos bewys deur die hoë erfbaarheidsyfers vir die klone verkry in hierdie proef. Die hoë mortaliteit wat tot gevolg gehad het dat minder ramette per kloon oorleef het bederf egter tot 'n mate die voordeel van klonering in hierdie proewe.

Die voorspelde vordering wys die voordeel van die gekloonde teelpopulasie in terme van beide die teel- en produksiepopulasie vordering. Verkorting van die teelsiklus deur klone vinniger te vermeerder sal ook bydra om vordering per jaar te verhoog. Hoë vordering in die produksiepopulasie is voorspel, veral vir die seleksie van getoetste klone vir aanwending wat plaas kan vind wanneer seleksie vir die volgende generasie gedoen word. Die voordeel van die gekloonde populasie is bewys tweeledig te wees, naamlik verhoging van die akkuraatheid van binne familie seleksie en vermeerdering van die vordering deur die vinniger aanwending van getoetste klone en daarom die vinniger verhoging van voorspelde vordering in die plantasie deur die ontplooiing van getoetste klone.

Sleutelwoorde: Breë sin oorerflikheid, eng sin oorerflikheid, gekloonde teel- populasie, additiewe genetiese variansie, nie-additiewe genetiese variansie, *Eucalyptus grandis*

ACKNOWLEDGEMENTS

I wish to express my gratitude to the following persons:

To my supervisor, Prof C.Z. Roux, and my co-supervisor Dr S.D. Verryn, for their guidance, time and patience.

To all those who were involved in the establishment, maintenance and assessment of these trials, particularly Brian Pierce (CSIR) and John Mather (Safcol) and their respective teams.

To Safcol who contributed land for the trials.

To the CSIR who provided the funding to complete this study.

To my colleagues at the CSIR for their support and encouragement.

To my husband Glen, for his unfailing support, continual encouragement, patience and understanding during the time I have been working on this study.

TABLE OF CONTENTS

DECLARATION.....	i
SUMMARY	ii
OPSOMMING.....	iv
ACKNOWLEDGEMENTS	vi
TABLE OF CONTENTS	vii
LIST OF TABLES	ix
LIST OF FIGURES.....	xi
NOMENCLATURE AND DEFINITIONS	xiii
CHAPTER 1: INTRODUCTION AND OBJECTIVES	1
1.1 Background.....	1
1.2 The use of clones in forestry to estimate genetic variance components.....	2
1.3 Objectives of the Study.....	6
CHAPTER 2: MATERIALS.....	8
2.1 Genetic Material	8
2.2 Trial Design	9
2.3 Trial Establishment.....	11
2.4 Trial Assessment.....	14
CHAPTER 3: METHODS	15
3.1 Variation in a Trait	15
3.2 Analysis of Variance	17
3.3 Estimation of Variance Components.....	20
3.3.1 Scenarios for estimating genetic variation	25
Scenario 1	25
Scenario 2	25
Scenario 3	25
3.3.2 Editing genetic variance component estimates.....	26
3.4 Correlations	26
3.5 Genetic Gains	29
3.5.1 Breeding Population Gains.....	30
3.5.2 Production Population Gains.....	31
3.6 Effects of selection on additive genetic variance	34
CHAPTER 4: DATA EDITING	36
CHAPTER 5 : SEPARATE TRIAL RESULTS	41

CHAPTER 6 : COMBINED TRIAL RESULTS	50
CHAPTER 7 : RESULTS FOR GENERATIONS F1 AND F2.....	60
CHAPTER 8 : PREDICTED GAINS.....	70
8.1 Predicted Breeding Population Gains.....	71
8.2 Predicted Production Population Gains.....	75
CHAPTER 9 : CORRELATIONS	78
CHAPTER 10 : DISCUSSION	80
CHAPTER 11 : CONCLUSION	94
LIST OF REFERENCES	97
APPENDIX A : ASSESSMENT TECHNIQUES.....	105
APPENDIX B : DATA EDITING	109
APPENDIX C : ANALYSIS OF VARIANCE IN THE SEPARATE TRIALS (B1, B2, B3)	114
APPENDIX D : ANALYSIS OF VARIANCE IN POOLED DATA WITH TRIAL EFFECT	124
APPENDIX E : T-TEST FOR GENERATIONS F1 AND F2.....	132

LIST OF TABLES

Table 1.	Summary of trial design for trials B1, B2 and B3.	11
Table 2.	Details of the site location and conditions of trials B1, B2 and B3 at Port Durnford (Schulzere, 1997).....	12
Table 3.	The analysis of variance and variance component estimation for volume, height, DBH, stem form and disease tolerance in trials B1, B2 and B3.	18
Table 4.	The analysis of variance and variance component estimation for volume, height, DBH, stem form and disease tolerance in the pooled data for trials B1, B2 and B3.	19
Table 5.	Percentage dead trees, runts and broken tops at 38 and 66 months in trials B1, B2 and B3.....	37
Table 6.	Percentage survival at 38 and 66 months in trials B1, B2 and B3.	37
Table 7.	Percentage survival at 38 and 66 months in the blocks removed from the data sets of trials B1, B2 and B3.	38
Table 8.	Trial means and descriptive statistics for the 38 and 66 months assessment of trials B1, B2 and B3.	42
Table 9.	Family and clone frequencies, both established and realised in the data for the two ages of assessment, in trials B1, B2 and B3.	44
Table 10.	Estimates of variance components and heritabilities for trials B1, B2 and B3.	45
Table 11.	Estimates of heritabilities for trials B1, B2 and B3.....	49
Table 12.	Means and descriptive statistics for the pooled data (data set B123) of the 38 and 66 months assessment of trials B1, B2 and B3.....	50
Table 13.	Estimates of variance components and heritabilities for all three trials (B1, B2 and B3) combined.	52
Table 14.	Heritability estimates and composition of genetic variance for tolerance to <i>Coniothyrium</i> , <i>Cryphonectria</i> , <i>Endothia</i> and <i>Botryosphaeria</i> at 38 and 66 months....	57
Table 15.	Frequencies of first generation (F1) and second generation (F2) families and clones in the pooled data for trials B1, B2 and B3.	60
Table 16.	Means and descriptive statistics for F1 and F2 families in the pooled data of the 38 and 66 months assessment of trials B1, B2 and B3.....	61

Table 17.	Estimates of variance components and heritabilities for the first (F1) and second (F2) generation families from data of all three trials (B1, B2 and B3) combined.	63
Table 18.	Predicted and actual additive genetic variance for volume and stem form at 66 months in the F1 and F2.	69
Table 19.	Estimate of predicted gain in the breeding population for selection at 66 months for volume in the cloned open pollinated breeding population B1, B2 and B3.	71
Table 20.	Estimate of predicted genetic gain in the breeding population for selection for volume at 66 months in an open pollinated breeding population with the same number of families and total number of trees as the cloned population.	72
Table 21.	Estimate of predicted genetic gain in the breeding population for selection for volume at 66 months in an open pollinated breeding population with the same number of families and individuals per family as the cloned population.	73
Table 22.	Estimate of predicted gain in the breeding population with 5 ramets per clone, for selection at 66 months for volume in the cloned open pollinated breeding population B1, B2 and B3.	74
Table 23.	Estimate of predicted genetic gain in the breeding population for selection for volume at 66 months in an open pollinated breeding population with the same number of families and total number of trees as the cloned population with 5 ramets per clone.	74
Table 24.	Predicted genetic gains for the production population scenarios.	76
Table 25.	Phenotypic age-age correlations estimated in the pooled data of trials B1 and B2 between 38 and 66 months, on an individual tree, family mean and clone mean basis.	78
Table 26.	Phenotypic age-age (38-66 months) correlations estimated on an individual tree basis in trials B1 and B2.	79

LIST OF FIGURES

Figure 1.	Map of Kwa-Zulu Natal with the location of trials B1, B2 and B3 indicated at Port Durnford.	13
Figure 2.	<i>E.grandis</i> progeny trial B1 at Port Durnford, age 66 months (November 1999).....	14
Figure 3.	Estimated additive (A) and non-additive (NA) variances as a percentage of total genetic variance in volume at age 38 and 66 months for each of the three scenarios considered.	54
Figure 4.	Estimated additive (A) and non-additive (NA) variances as a percentage of total genetic variance in height at age 38 and 66 months for each of the three scenarios considered.	55
Figure 5.	Estimated additive (A) and non-additive (NA) variances as a percentage of total genetic variance in DBH at age 38 and 66 months for each of the three scenarios considered.	55
Figure 6.	Estimated additive (A) and non-additive (NA) variances as a percentage of total genetic variance in stem form at age 38 and 66 months for each of the three scenarios considered.	56
Figure 7.	Estimated additive (A) and non-additive (NA) variances as a percentage of total genetic variance in disease tolerance at age 38 and 66 months for each of the three scenarios considered.	56
Figure 8.	Estimated additive (A) and non-additive (NA) variances as a percentage of total genetic variance in tolerance to <i>Coniothyrium</i> at age 38 and 66 months for each of the three scenarios considered.	58
Figure 9.	Estimated additive (A) and non-additive (NA) variances as a percentage of total genetic variance in tolerance to <i>Endothia</i> at age 66 months for each of the three scenarios considered.	59
Figure 10.	Estimated additive (A) and non-additive (NA) variances as a percentage of total genetic variance in tolerance to <i>Botryosphaeria</i> at age 38 months for each of the three scenarios considered.	59

Figure 11. Estimated additive (A) and non-additive (NA) variances as a percentage of total genetic variance for volume at 66 months over generations (F1 and F2) for the three scenarios considered.	66
Figure 12. Estimated additive (A) and non-additive (NA) variances as a percentage of total genetic variance for height at 66 months over generations (F1 and F2) for the three scenarios considered.	66
Figure 13. Estimated additive (A) and non-additive (NA) variances as a percentage of total genetic variance for DBH at 66 months over generations (F1 and F2) for the three scenarios considered.	67
Figure 14. Estimated additive (A) and non-additive (NA) variances as a percentage of total genetic variance for stem form at 66 months over generations (F1 and F2) for the three scenarios considered.	67
Figure 15. Estimated additive (A) and non-additive (NA) variances as a percentage of total genetic variance for disease tolerance at 66 months over generations (F1 and F2) for the three scenarios considered.	68

NOMENCLATURE AND DEFINITIONS

A list of abbreviations, contractions and definitions frequently used in the text is included for ease of reference; others are expanded in the text:

A	Additive genetic variance component
DBH	Diameter at breast height (1.3metres) in millimetres
Disease	Disease tolerance (Mean tolerance to <i>Coniothyrium</i> , <i>Crypohonectria</i> , <i>Botryosphaeria</i> and <i>Endothia</i> scored on a 5 point scale.)
P0	Parental generation
F1	First generation
F2	Second generation
Fam	Family
G	Genetic variance component
Ht	Height in metres
NA	Non-additive genetic variance component
SE	Standard error
Stem	Stem form (8 point scale)
VAR	Variance
h^2	Narrow sense heritability
H^2	Broad sense heritability
σ	Standard deviation
σ^2	Variance component

Subscripts:

A	Additive genetic variance component
c(f)	Clone within family
F	Family
G	Genotypic or genetic variance component
NA	Non-additive genetic variance component
P	Phenotypic

Definitions:

- Clone: a group of genetically identical individuals
- Family: Genotypes raised from the seed of a single tree
- Provenance: the original native origin (geographic) of a population
- Ramet: an individual member of a clone