Proc. Assoc. Advmt. Anim. Breed. Genet. Vol 14

MAJOR GENE EFFECTS ON RESISTANCE TO BODY STRIKE AND FLEECE ROT IN MERINO SHEEP REVISITED

S.I. Mortimer[1], J.M. Henshall[2] and B. Tier[2]

[1]NSW Agriculture, Agricultural Research Centre, Trangie NSW 2823
[2]Animal Genetics and Breeding Unit[*], University of New England, Armidale NSW 2351

SUMMARY
Segregation analyses of fleece rot and body strike records from divergent lines of Merino sheep selected for increased (Susceptible line) and reduced (Resistant line) expression of fleece rot and body strike were used to examine the possibility of major gene effects influencing the expression of resistance to these disease traits. Analyses were based on a Markov Chain Monte Carlo algorithm that accounted for the categorical nature of the traits by sampling variables from an unobserved underlying continuous distribution. The analyses suggest that the data were consistent with a model that includes a major gene influencing both resistance to fleece rot and body strike. Under this model, the proportion of the phenotypic variance due to a major gene was 20% for fleece rot and 15% for body strike incidences. The desirable alleles for both traits were dominant and at high gene frequencies.
Keywords: Major genes, body strike, fleece rot, resistance, Merino.

INTRODUCTION

In an effort to use management practices to minimise costs due to treatment and lost production (McLeod 1995) and produce wool with minimum levels of chemical resisdues in a safe and environmentally friendly way (James 1990), non-chemical methods for the control of blowfly strike in the Australian wool industry are receiving increased attention from wool growers. Breeding for increased resistance to body strike and its precursor, fleece rot, provides one long-term option for blowfly strike control in Australian Merino sheep. Resistance to both body strike and fleece rot, under natural conditions, are unique traits in that selection can exploit genetic variation available between (Atkins and McGuirk 1979; Raadsma 1991; Mortimer and Atkins 2001) and within Merino bloodlines (Mortimer *et al.* 1998). Whether or not part of this genetic variation is due to genes of major effect is unknown. If resistance to body strike and fleece rot is influenced by major genes, methods can be developed to select for these genes using DNA markers.

The availability of pedigreed data from divergent lines of Merino sheep selected for and against resistance to these disease conditions (Mortimer *et al.* 1998) allows testing for the influence of possible major genes through segregation analysis. Earlier segregation analyses of these data, using a regression procedure implemented by the FINDGENE software, assumed a normal distribution for the traits and provided inconclusive evidence on the possibility of major gene effects influencing fleece rot and body strike resistance (Mortimer *et al.* 2000). New methods for segregation analysis now make it possible to analyse traits with a categorical distribution. These methods are used here to

[*] AGBU is a joint institute of NSW Agriculture and the University of New England.

re-examine the possibility of major gene effects influencing resistance to fleece rot and body strike in Merino sheep selected for and against these conditions.

MATERIALS AND METHODS

Pedigreed data from divergent lines of Merino sheep selected for increased (Susceptible line) and reduced (Resistant line) expression of body strike and fleece rot were used. A description of the experiment and selection responses is provided by Mortimer *et al.* (1998). For this study, observations on the natural occurrences of hogget fleece rot (scored at 13 months of age) and body strike (recorded between birth and 13 months of age) were analysed. Records were available from animals born between 1977 and 1997. Fleece rot and body strike incidences were recorded as categorical traits, where unaffected animals had scores of 0.

'The Gene Detective' was used to analyse the data. This software uses a Markov Chain Monte Carlo (MCMC) algorithm (Tier and Henshall 2001) to detect possible quantitative trait loci for one or more traits from pedigreed data while estimating fixed effects and polygenic variance components simultaneously. The data augmentation method of Albert and Chib (1993) was used to analyse fleece rot and body strike incidences as categorical traits by sampling variables from an underlying continuous distribution. The model fitted for each trait included fixed effects and effects for an animal's estimated breeding value, its dam's breeding value and permanent environmental influence, error and a quantitative trait loci with two alleles. Fixed effects for both traits were contemporary group (based on sex-year of birth classification) and birth-rearing type (single-born and -reared versus multiple-born and single-reared versus multiple-born and -reared). Analysed as single traits, variances due to polygenic, quantitative trait loci and error were estimated for each trait. There were 3927 records for fleece rot incidence and 2859 records for body strike incidence.

RESULTS

Mean fleece rot and body strike incidences in the unadjusted data were 21% and 5% respectively. The data for fleece rot and body strike incidences were consistent with a model that includes major gene effects as influences on these traits. The proportion of the phenotypic variance due to a major gene was estimated as 20% for fleece rot incidence and 15% for body strike incidence. Estimates of direct genetic heritability were 0.20 for fleece rot incidence and 0.08 for body strike incidence. Maternal genetic heritability was estimated at 0.08 and 0.04 for the respective traits, with maternal permanent environmental effect estimated at 0 and 0.02 respectively.

Table 1. Estimated quantitative trait loci effects (with standard errors in brackets) on the underlying scale for body strike and fleece rot incidences (A is the preferred allele)

	Genotype			
	AA	Aa	aA	aa
Fleece rot incidence	-0.2086(0.023)	-0.0349(0.068)	0.0203(0.061)	1.3098(0.083)
Body strike incidence	-0.0111(0.003)	-0.0002(0.015)	-0.0023(0.014)	1.5585(0.124)

Estimated quantitative trait loci effects on the underlying scale for each trait are presented in Table 1. Alleles reducing fleece rot and body strike incidences (A) are considered favourable. For both traits,

the desirable allele was dominant, with a gene frequency of 0.75 for fleece rot incidence and a gene frequency of 0.90 for body strike incidence. Individuals carrying at least one copy of the desirable allele were expected to have scores of 0 for the occurrences of fleece rot and body strike. The desirable allele for both traits being dominant and of large effect was consistent with the large proportions of phenotypic variation found to be due to major gene effects when fitted in the models for each trait.

DISCUSSION

The method applied enabled the traits to be analysed categorically, each with an unobserved underlying distribution, rather than as continually distibuted traits. The method also allowed fixed and genetic effects to be estimated simultaneously using an MCMC algorithm. The data for both fleece rot and body strike incidences were observed to be consistent with models that included effects of major genes and polygenes on resistance to these disease conditions. The possibilities of major gene effects are consistent with the results from the earlier segregation analyses (Mortimer *et al.* 2000) obtained with FINDGENE. For both traits, a high proportion of the genetic variance was explained by the major gene effects. This may be due to the magnitude of the allele effects. At least one copy of the desirable allele led to animals being unaffected by fleece rot and body strike i.e. scores of 0 for these traits.

Other analyses of data from the Resistant and Susceptible selection lines have not yet examined the importance of maternal effects on these disease traits, but they have only small effects in this study. Maternal effects have not been reported as influencing these traits in the few studies where animal models have been fitted to data on fleece rot incidence (Li *et al.* 1999) or body strike incidence (H.H. Montaldo and S.I. Mortimer unpub. data). Further analyses will examine the need for and impact of including maternal effects in models fitted to this data.

Within the data for both traits, the desirable alleles favouring resistance were at high frequencies (at least 0.75). These frequencies may be a consequence of conventional selection on phenotype practiced within the selection lines, which has yielded significant responses in these lines (Mortimer *et al.* 1998). This suggests there is little additional value in being able to select for these major genes for resistance to fleece rot and body strike. However, expression of these traits can be limited by environmental conditions (for example insufficient rainfall to allow prolonged wetting of a sheep's skin and/or conditions preventing blowfly activity) such that selection on phenotype may not always be possible. The availability of markers for any major gene effects influencing fleece rot and body strike incidences would overcome this difficulty of assessing resistance in the paddock, particularly for those environments where incidences vary across years. Resistance could also be assessed at any age. Furthermore, markers for major gene effects on these traits could be utilised by ram breeders who breed rams, which are rarely or not challenged by the diseases, to be sold and used in commercial flocks run in environments of greater challenge from fleece rot and body strike.

If a marker for major gene effects influencing resistance to fleece rot and body strike were identified in the selection lines, the gene(s) for resistance could then be introgressed into ram breeding flocks using a DNA test as suggested by Goddard and Kinghorn (1998). Introgression of resistance genes

using a marker may prevent unfavourable changes in clean fleece weight occurring as would be expected following introgression based on phenotype for fleece rot (Mortimer and Atkins 2001).

Unfortunately segregation analysis can only indicate whether or not the data and the pedigree support the possibility of a major gene being present in the population. Without markers, it is imposssible to conclude that such a gene is present. Henshall *et al.* (2001) have demonstrated the efficacy of combining large amounts of phenotypic data with genotypes on key parents only to map quantitative trait loci. A possible next step for this project, if funding is available, is to screen the population for markers.

ACKNOWLEDGMENTS

The efforts of Dr J.E. Watts, Dr B.J. McGuirk, Mr K.J. Thornberry and Dr H.W. Raadsma during the establishment of the selection lines are acknowledged. We thank the many technical staff members for their inputs to the conduct of the experiment and data collection.

REFERENCES

Albert , J.H. and Chib, S. (1993) *J. Am. Stat. Assoc.* **88**: 669.

Atkins, K.D. and McGuirk, B.J. (1979) *Wool Tech. Sheep Breed.* **27**: 15.

Goddard, M.E. and Kinghorn, B.P. (1998) "The Use of Genetic Markers in Sheep Breeding", International Wool Secretariat, Melbourne.

Henshall, J.M., Tier, B. and Kerr, R.J. (2001) *Proc. Assoc. Adv. Anim. Breed. Genet.* **14**: 301.

James, P.J. (1990) *Proc. Aust. Soc. Anim. Prod.* **18**: 37.

Li, Y., Swan, A.A. and Purvis, I. (1999) *Proc. Assoc. Adv. Anim. Breed. Genet.* **13**: 524.

McLeod, R.S. (1995) *Inter. J. Parasit.* **25**: 1363.

Mortimer, S.I. and Atkins, K.D. (2001) *Proc. Assoc. Adv. Anim. Breed. Genet.* **14**: 183.

Mortimer, S.I., Atkins, K.D. and Raadsma, H.W. (1998) *Proc. 6th Wld Congr. Gen. Appl. Livest. Prod.* **27**: 283.

Mortimer, S.I., Montaldo, H.H., Kinghorn, B.P. and Atkins, K.D. (2000) *Asian-Aus. J. Anim. Sci.***13** Supplement C: 152.

Raadsma, H.W. (1991) *Aust. J. Agric. Res.* **42**: 279.

Tier, B. and Henshall, J.M. (2001) *Gen. Sel. Evol.* **33**: (In press).