

SEGREGATION ANALYSIS FOR MAJOR GENES AFFECTING ADAPTIVE TRAITS IN CATTLE GRAZED IN THE TROPICS

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SUMMARY

Segregation analysis was conducted to detect major genes affecting tick and worm counts for tropical beef cattle. For both traits, a model in which a significant proportion of the genetic variation was due to a major gene was found to be feasible. It was unlikely that the same gene had a significant effect on both traits.

Keywords: Worm resistance, tick resistance, major gene, tropical beef cattle.

INTRODUCTION

Genetic parameters for resistance to ticks and worms have previously been estimated for tropical beef cattle (Burrow 2001) under the assumption that log transformed parasite counts have a normal distribution, and that genetic variation in the traits is due to the effects of many genes. The heritabilities for both traits were moderate to high, with no significant maternal effects, and with a favourable genetic correlation ($r_g = 0.3$) between tick and worm counts. Genetic correlations between tick and worm counts and other traits of economic importance were estimated as either favourable or weak. This suggests that tick and/or worms counts may be suitable traits to include in a selection index (Burrow 2001).

Conventional selection methods require recording traits on large numbers of animals. For resistance to parasites, recording requires exposure to the parasite, which is undesirable from a management and animal welfare perspective. Consequently, quantitative trait loci (QTL) for parasite resistance, which offer the potential to select without trait recording, are of considerable interest.

This paper describes a preliminary screening of the data used by Burrow (2001) to determine whether a model in which a proportion of the genetic variation in tick or worm counts is due to a QTL is feasible, and if so, whether or not the same QTL affect both traits.

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MATERIALS AND METHODS

The animals used in the analysis were calves from stabilised composite herds at the National Cattle Breeding Station ‘Belmont’, Central Queensland reared between 1983 and 1991. The composites were either Hereford (25%), Shorthorn (25%) and Africander (50%) or Hereford (25%), Shorthorn (25%), Brahman (25%) and Africander (25%). For more details see Burrow (2001). The pedigree file consisted of 421 sires and 2109 dams and 2242 recorded progeny.

The raw data were repeated counts of ticks (T_1, T_2, \dots, T_m) and worm eggs per gram ($EPG_1, EPG_2, \dots, EPG_n$). The traits analysed were mean log tick count ($MLT = (\log_{10}(T_1 + 1) + \log_{10}(T_2 + 1))/2$) for animals with at least two tick counts and mean log worm count ($MLW = (\log_{10}(EPG_1 + 10) + \log_{10}(EPG_2 + 10) + \log_{10}(EPG_3 + 10))/3$) for animals with at least three worm EPG records. The data and the modelling of fixed and random effects are described in Burrow (2001). In all, there were 1629 MLT records and 2076 MLW records, 1473 animals had records for both traits.

Segregation analyses were carried out using the multiple trait version of ‘‘The Gene Detective’’ (Tier and Henshall 2001). Maternal effects were not significant in the analyses of Burrow (2001), so maternal effects were not fitted. Fixed effects of year, breed, sex, and treatment were fitted for both traits.

Where no QTL was included in the model, the final 1000 of 2000 samples were used to obtain the posterior distributions of the parameters of interest. Where a QTL was included in the model, the final 2000 of 4000 samples were used to obtain the posterior distributions of the parameters of interest. The mean of the posterior distribution was used to estimate each parameter. To ensure that the parameter space was adequately explored, starting values were chosen so that the polygenic variance was equal to the total additive variance, and the QTL variance equal to zero. Both univariate and bivariate analyses were performed. It was necessary to apply a moderate prior of 0.5 to the allele frequency to assist with stability.

Table 1. Estimated variance components from univariate analyses, additive (A) and QTL (Q) heritabilities (h^2) and error (E), additive (A), QTL (Q) and phenotypic (P) variances (Var)

| Trait | n | h^2_A | h^2_Q | Var(E) | Var(A) | Var(Q) | Var(P) |
|------------------|------|---------|---------|--------|--------|--------|--------|
| MLW ⁿ | 2076 | 0.57 | | 0.070 | 0.094 | | 0.164 |
| MLW ^m | 2076 | 0.25 | 0.43 | 0.052 | 0.041 | 0.069 | 0.162 |
| MLT ⁿ | 1639 | 0.42 | | 0.134 | 0.099 | | 0.233 |
| MLT ^m | 1639 | 0.24 | 0.32 | 0.106 | 0.060 | 0.079 | 0.245 |

ⁿ no QTL fitted

^m QTL fitted

RESULTS AND DISCUSSION

Estimated variance components from univariate analyses of MLT and MLW appear in Table 1. For both traits, a model with a QTL explaining a large proportion of the variance was consistent with the data. Fitting a QTL reduced both the error and additive variance, with the sum of the reductions similar to the QTL variance. That all of the QTL variance was not expressed in the additive variance when no QTL was fitted suggests that the effect of the QTL may not be additive. This was supported by the estimates of the QTL effects (Table 2). The desirable allele (a) was partially recessive for both traits. There was no evidence of any imprinting effect.

Table 2. Estimated QTL effects (standard deviations) and allele frequencies from univariate analyses. The first allele listed (a or A) is the one inherited from the sire, p is the frequency of the allele A in the base population. Standard deviations are estimated as the standard deviation of the samples used to estimate the means

| Trait | aa | AA | Aa | AA | p |
|-------|--------------|-------------|-------------|-------------|-------------|
| MLW | -0.49 (0.02) | 0.09 (0.02) | 0.08 (0.03) | 0.20 (0.02) | 0.53 (0.05) |
| MLT | -0.57 (0.07) | 0.02 (0.07) | 0.09 (0.05) | 0.17 (0.05) | 0.54 (0.06) |

Despite the QTL variance and effect estimates being so similar for the two traits, the multivariate analyses (Table 3) did not support the hypothesis that one QTL was affecting both traits. When no QTL was fitted, the estimated genetic correlation was positive and favourable but only moderate (0.26), and similar to the 0.30 reported by Burrow (2001). When a QTL was fitted, the estimated variance components for MLW were similar to those from the corresponding univariate analysis. However, the estimated variance components for MLT changed little from those from a model with no QTL, and the estimated QTL heritability for MLT was only 0.02. The QTL with an effect on MLW had negligible effect on MLT.

To confirm this result, the analysis was repeated with a QTL fitted, but with the likelihood used in QTL sampling estimated from MLT alone. As would be expected for this model, the estimated variance components for MLT were similar to those from a univariate analysis with a QTL fitted. However, this time the estimated variance components for MLW changed little from those from a model with no QTL, and the estimated QTL heritability for MLW was 0.00. The QTL with an effect on MLT had negligible effect on MLW.

As this segregation analysis did not include any information from molecular markers, the results do not necessarily mean that two different genes, each affecting one of MLT or MLW only, are segregating in this population. Without markers, segregation analysis can only show that a model including a QTL is plausible. It is possible that another effect, such as that from a loosely linked region of the chromosome, is responsible for the variance attributed to the QTL. With better data (more records, deeper pedigree) the probability of “false positive” results is reduced, but it can never be eliminated. However, if segregation analysis results suggest that a model including a QTL effect is plausible, and if the estimate of the effect of the postulated QTL suggests that the QTL may be of economic importance, then a marker study may be warranted. In this case, sires which have a higher probability of being heterozygous on the basis of the segregation analysis results may be chosen.

Table 3. Estimated heritabilities and phenotypic, genetic, polygenic and QTL correlations between MLW and MLT. Heritabilities appear on the diagonal, genetic, polygenic or QTL correlations appear above the diagonal and phenotypic correlations appear below the diagonal

| | | MLW | MLT |
|--|-----|------|------|
| No QTL in model | | | |
| Polygenic | | | |
| | MLW | 0.57 | 0.26 |
| | MLT | 0.15 | 0.44 |
| QTL fitted, with sampling on both traits | | | |
| Polygenic | | | |
| | MLW | 0.29 | 0.30 |
| | MLT | 0.13 | 0.39 |
| QTL | | | |
| | MLW | 0.38 | 0.35 |
| | MLT | | 0.02 |
| QTL fitted, with sampling on MLT only | | | |
| Polygenic | | | |
| | MLW | 0.55 | 0.29 |
| | MLT | 0.14 | 0.32 |
| QTL | | | |
| | MLW | 0.00 | 0.00 |
| | MLT | | 0.29 |

CONCLUSIONS

Segregation analysis suggests that QTL affecting tick and worm resistance may be segregating in the tropical beef cattle population analysed. Despite similarities in the modes of expression and allele frequencies estimated for QTL for tick and worm resistance, it does not appear that the same QTL is responsible for the effect on each trait. As the QTL are estimated to be recessive for the desirable alleles, gene tests would be required to fully exploit the variation due to the QTL.

REFERENCES

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