

QTL FOR BIRTH WEIGHT IN *BOS TAURUS* CATTLE

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SUMMARY

Records of birth weight (BW) from a beef cattle experiment in New Zealand and Australia were analysed to test for linkage to DNA markers on all chromosomes except the sex chromosome. This was part of a search for quantitative trait loci (QTL) for production, carcass and meat quality traits. Two extreme *Bos taurus* breeds, Jersey (J) and Limousin (L), were used to generate over 400 back-cross progeny in each country. There were 5 significant BW QTL, with one on each of 5 different chromosomes, and two other locations where QTL effects approached significance. The effects of sire-derived J and L alleles on progeny BW for significant QTL ranged from -0.84 to 0.73 phenotypic standard deviations, or from -3.13 to 2.73 kg, indicating that QTL from both breeds were associated with increased BW.

INTRODUCTION

DNA-marker technology has the potential to assist seed-stock beef producers with genetic improvement of traits that are difficult or inconvenient to measure, and to assist research workers in identifying chromosomal regions containing quantitative trait loci (QTL), and eventually genes, which control animal performance traits. A collaborative study was established in 1995 between AgResearch in New Zealand (NZ) and Adelaide University in Australia to search for DNA markers significantly linked to production, carcass and meat quality traits in beef cattle. The present paper reports on a sub-set of those traits, namely evidence from microsatellite markers of significant linkage to birth weight (BW).

MATERIAL AND METHODS

Trial design. The trial design involved two of the more extreme *Bos taurus* dam breeds, Jersey (J) and Limousin (L), mated to JxL or LxJ first-cross sires to produce back-cross calves. A total of about 400 heifer and steer progeny were generated in each country, using three sires per country. Birth weights (BW) were recorded within 24 hours of birth. There were two calf crops in NZ (1996 and 1997 births), and three in Australia (1996-98 births). In NZ, the Jersey back-crosses were born in Jersey herds, whereas the Limousin back-crosses were born in 1996 as singles and twins to recipients in an embryo transfer programme on AgResearch's Whatawhata Station, and in 1997 they were born as singles in two Limousin herds. In Australia, back-crosses of both types were born to Jersey or Limousin dams at the University's Martindale property at Mintaro, SA. In total, 313 NZ and 365 Australian BW records were available for analysis.

Marker analyses and data analyses. Sire-derived alleles were determined for a total of 253 informative microsatellite loci (an average of 185 loci per sire group) spread across all chromosomes

(Chr), except for the X and Y Chr. Phenotypes were pre-adjusted to account for known fixed effects including country, year, herd, dam breed, birth type (for 1996 Limousin crosses in NZ), and age of dam where known. Residuals were stored after standardisation by dividing by the within-country phenotypic standard deviation (σ_p). Linkage with standardised BW was tested using Knott *et al.* (1996) interval-mapping regression procedures, with SAS (Version 8.02, Proc GLM). Positions of microsatellites were taken from the map of Kappes *et al.* (1997). When mapping QTL, a significantly linked marker ($P < 0.05$, genome-wide test) was required to have an F-test statistic > 10.0 (when the 6 individual sires were tested separately) or F-test statistic > 3.6 (all sires tested together), using the criteria of Lander and Kruglyak (1995).

RESULTS

Significant markers, their size and positions. Overall means from NZ and Australian BW records were 29.45 and 26.45 kg, respectively (combined $\sigma_p = 3.72$ kg). Five genome-wide significant chromosomal regions were identified for BW (Table 1). Two other regions, on Chr 3 and 10, approached significance. All seven regions were on different Chr. Four of the six sires (#394 from NZ, and all three from Australia) were represented. The effects ranged in size from -0.84 to 0.73 σ_p , or -3.13 to 2.73 kg, i.e. up to about 10 per cent of the mean. Two significant effects (Chr 5 and 21) resulted from a J allele increasing BW relative to an L allele, whilst the other five were L alleles increasing BW relative to J alleles. Positions are tabulated relative to the beginning of the Chr as defined by the map of Kappes *et al.* (1997). In addition to the statistical tests tabulated, combined results from all six sires were significant on Chr 14 and 21, each at about the same chromosomal position as when individual sires were tested separately. F-statistics for BW along Chr 5 and 14 are shown in Figure 1; these linkages represented the highest F-values obtained for BW across the genome in this study.

Table 1. Significant QTL for birth weight, showing their chromosomal position, and sizes of effects in units of phenotypic standard deviations (σ_p) and in kg; signs represent effects of Limousin-derived minus Jersey-derived alleles

Chr	Sire ^A	Position (cM)	Effect (σ_p)	Effect (kg)	F value	$-\log P$
1	368	93	0.73±0.21	2.73±0.78	12.29	3.31
3	394	22	0.64±0.21	2.37±0.79	8.95 ^B	2.54
5	398	46	-0.84±0.22	-3.13±0.81	14.89	3.90
10	394	86	0.71±0.23	2.64±0.85	9.62 ^B	2.70
14	361	35	0.69±0.19	2.58±0.69	13.81	3.66
20	394	9	0.71±0.22	2.65±0.82	10.58	2.92
21	368	5	-0.62±0.19	-2.31±0.72	10.32	2.86

^A Sire 394 was used in New Zealand; 361, 368 and 398 in Australia.

^B Results approaching significance.

DISCUSSION

Size of effects. The sizes of significant effects generally ranged from 0.6 to 0.8 σ_p , which reflected the power of the experimental design. Thus we were able to identify significant allelic effects of

about 2.5 to 3 kg, a size that would be agriculturally useful for making changes. Intermediate optima for BW are well known in beef calves (e.g. Morris *et al.* 1986), and marker-assisted selection could be used to select for change in either direction.

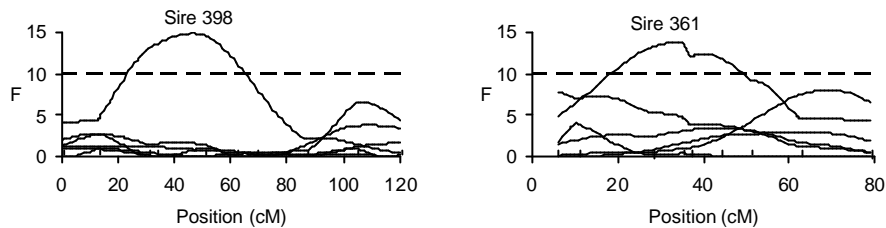


Figure 1. F-statistics for birth weight in the progeny of 6 sires, at 1 cM positions on chromosomes 5 (left figure) and 14 (right); arrows indicate the positions of the microsatellites.

Other reported BW QTL. There have been at least nine BW QTL in other cattle studies where the QTL are on the same Chr as in this study: Chr 1, Stone *et al.* (1999); Chr 5, Davis *et al.* (1998), Imumorin *et al.* (2001) and Li *et al.* (2002); Chr 14, Davis *et al.* (1998), Buchanan *et al.* (2000); Chr 20, Casas *et al.* (2002); and Chr 21, Davis *et al.* (1998) and Casas *et al.* (2002). In addition, cervine linkage group 21 (which is homologous to bovine Chr 14) has been reported to carry a QTL for BW (Slate *et al.* 2002). Some of the QTL in these other studies also provide confirmation of the approximate positions of all five QTL on Chr 1, 5, 14, 20, 21. In contrast, the BW QTL which we found on Chr 3 and 10 appear not to have been reported before. Interestingly, these two were the QTL which just failed to exceed the threshold, so perhaps they are genuinely smaller effects not identified elsewhere, or they may turn out on further investigation not to be real. Slate *et al.* (2002) reported another BW QTL on cervine linkage group 12 which is homologous to cattle Chr 10, and possibly in a similar position. Other BW QTL on bovine Chr absent from Table 1 have been reported by others: Chr 2, Grosz and MacNeil (2001); Chr 6, Davis *et al.* (1998) and Casas *et al.* (2000); and Chr 18, Davis *et al.* (1998). Imumorin *et al.* (2001) also reported maternally expressed QTL for BW on Chr 3 and 19.

CONCLUSIONS

Our results showed that five QTL were significantly linked to BW in our experiment. These were consistent with BW QTL identified by other research groups. Considering the genetic correlations of BW with dystocia, gestation length, and also with weight-gains or later weights, marker-assisted selection may be a cost-effective option, once an objective has been defined and appropriate genetic relationships determined. In addition, fine mapping around the marker sites may eventually lead to cloning of some of the underlying genes associated with BW, and this may provide important insights into the biology of BW variation and its control.

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