# QTL FOR COOKING LOSS IN *BOS TAURUS* CATTLE

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## SUMMARY

Records of cooking loss for *M. longissimus dorsi* and *M. semitendinosus* 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 chromosomes. 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 around 400 back-cross progeny in each country. For *M. longissimus dorsi*, there were four significant QTL for cooking loss (BTA 6, 7, 10, and 14) and three other locations where QTL effects approached significance (BTA 16, 24, and 25). There was also a QTL approaching significance on BTA 21 for cooking loss from the *M. semitendinosus*. The effects of L alleles relative to J alleles ranged from -0.66 to 1.42 phenotypic standard deviations (SD=1.8%) which would be sufficient to account for breed differences observed in other trials. None of the QTL were in regions homeologous to the RN or RYR1 genes in pigs that result in increased cooking loss.

## 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 in identifying chromosomal regions containing quantitative trait loci (QTL) and eventually the 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 with significant linkage to cooking loss. Sensory perception of tenderness and juiciness of steak are difficult to predict with objective measures. However, the loss of moisture from a steak when cooked is related to both traits (Perry *et al.* 2001b).

## MATERIAL AND METHODS

**Trial design.** The trial design involved two very different *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 in Australia and a half-sib brother of each sire in New Zealand. 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, Mintaro SA. In total, 410 NZ and 353 Australian records were available for analysis.

**Definition of trait.** Cattle were stunned with a captive bolt before carotid arteries were severed. In Australia, carcasses were electrically stimulated using extra low voltage (<45V) within five minutes of sticking. Carcasses were trimmed to minimum standards, then standard traits (e.g. carcass weight) were measured before entering the chiller (0-4°C). After being chilled overnight to a deep butt temperature <20°C, the carcasses were split at various rib sites according to market specifications. Full striploin (*M. longissimus dorsi*) and eye round (*M. semitendinosus*, Australia only) primals were cut into individual steaks and aging treatments were randomly assigned along the muscle. In New Zealand, steaks were aged at 15°C and measurements taken at days 1-4 post-slaughter. In Australia, steaks were aged in a commercial chiller and then frozen at days 1, 5, 12 or 26 post-slaughter to achieve equivalent aging to the New Zealand protocol. Once frozen, the Australian samples were transported to the laboratory, thawed and then cooking loss was measured. The cooking procedure was in plastic bags in a water bath to an internal temperature of 70°C (75°C in NZ) to achieve a "medium" degree of doneness as outlined by Perry *et al.* (2001a). Cooking loss was measured five times in New Zealand and four times in Australia. There was no systematic trend over time so the values were averaged and mean cooking loss was analysed as a single trait. Tenderness and pH were measured at the same time as cooking loss, but are not reported herein.

**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 bovine chromosomes (BTA), except for the X and Y. 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 ($\sigma_p$). Linkage with standardised cooking loss 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) and are tabulated relative to the beginning of the chromosome. 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 (when all sires were tested together), using the criteria of Lander and Kruglyak (1995).

**RESULTS AND DISCUSSION**

On average, moisture lost during cooking was 21.8% for *M. longissimus dorsi* and 26.3% for *M. semitendinosus* and the residual standard deviations were 1.8% and 1.3%, respectively. Three significant QTL were identified on chromosomes 6, 7, and 14 (Table 1). A QTL on chromosome 10 (located 80cM from the centromere) was significant when analysed across all six sire families (F>3.6). In addition, there were four QTL that approached significance based on a single sire analysis (F<10.0). The QTL on chromosome 25 approached significance for two sires but was still not significant across the six sires. None of the QTL were significant for both muscles.

**Table 1. Significant QTL for cooking loss, showing their chromosomal (Chr.) position, and sizes of effects in units of phenotypic standard deviations ($\sigma_P$) and in percent (%); signs represent effects of Limousin-derived minus Jersey-derived alleles**

| Chr. | Sire [A] | Position (cM) | Effect ($\sigma_P$) | Effect (%) | F value | -log P |
|---|---|---|---|---|---|---|
| *M. longissimus dorsi* | | | | | | |
| 6 | 417 | 8 | 0.92±0.24 | 1.65±0.43 | 14.5 | 3.81 |
| 14 | 368 | 7 | 0.78±0.23 | 1.40±0.41 | 11.6 | 3.17 |
| 7 | 394 | 0 | 1.42±0.43 | 2.57±0.77 | 11.1 | 3.04 |
| 10 | All six | 80 | | | 3.6 | 2.84 |
| 10 | 402 | 79 | -0.65±0.21 | -1.17±0.38 | 9.5 [B] | 2.67 |
| 16 | 394 | 30 | -0.59±0.19 | -1.07±0.35 | 9.5 [B] | 2.66 |
| 24 | 368 | 43 | 0.61±0.21 | 1.10±0.37 | 8.7 [B] | 2.47 |
| 25 | 402 | 21 | 0.56±0.19 | 1.01±0.34 | 8.6 [B] | 2.46 |
| 25 | 417 | 34 | -0.66±0.24 | -1.19±0.43 | 7.8 [B] | 2.27 |
| *M. semitendinosus* | | | | | | |
| 21 | 361 | 39 | -0.60±0.20 | -0.76±0.25 | 9.2 [B] | 2.59 |

[A] Sires 394, 402 and 417 were used in New Zealand; 361, 368 and 398 in Australia.
[B] Results approaching significance.

The Australian values for cooking loss were slightly higher than those reported for the "Southern Crossbreeding Project" (Pitchford *et al.* 2002). The difference may have been an age effect as heifers measured for cooking loss were slaughtered at 15 months and steers at 26 months in the Crossbreeding Project, both much younger than around 72 months for the Mapping Project. Similar variation was also found within another large Australian trial (Perry *et al.* 2001b).

The Crossbreeding project found large differences in cooking loss of the *M. longissimus dorsi* between Jersey x Hereford (15.3%) and Limousin x Hereford (18.2%). This could have been associated with intramuscular fat content since fat cells have a lower moisture content than muscle cells. However, there was clearly breed variation in cooking loss independent of intramuscular fat content. Thus, it was concluded that cooking loss was more likely a function of maintaining cell integrity during storage and cooking or with connective tissue content. The cell integrity hypothesis is supported by Mitsumoto *et al.* (1995) who showed cell integrity could be improved by vitamin E supplementation.

Low cooking loss is associated with improved juiciness as judged by a sensory panel (Perry *et al.* 2001b). Limousin have a high cooking loss (Pitchford *et al.* 2002) and the current study found five Limousin-derived QTL with increased cooking loss (BTA6, 7, 14, 24, and 25, Table 1). However, there were also three Limousin derived QTL with decreased cooking loss (BTA10, 16, and 25). Of the three that were significant, all were in favour of the Jersey allele and could have accounted for the breed difference found in the Crossbreeding Project.

The size of the QTL effects identified would easily account for the difference of 2.9% between $F_1$ crosses reported by Pitchford *et al.* (2002). The size of QTL effects generally ranged from 0.6 to 1.4 $\sigma_P$, which is as small as the Mapping Project experimental design had power to detect. Thus, significant allelic effects of about 1.0 to 2.6% were identified, a size that would allow genetic progress under marker or genotype assisted selection.

Lastly, while cooking loss is an important meat quality trait, the authors are aware of only one other cooking loss QTL mapping report. Malek *et al.* (2001) mapped a QTL for cooking loss in pork on SSC14. Comparative mapping with this chromosome is difficult because of homology with a number of human chromosomes. However, it is most likely homeologous to BTA5, which was not identified herein. The halothane gene (RYR1), which causes malignant hypothermia (in humans) and pale, soft and exudative (PSE) meat (in pigs) which has been reported to be associated with increased cooking loss (e.g. Lundstrom *et al.* 1998). RYR1 maps to SSC6q which is homeologous to BTA18 and did not correspond to any of the QTL identified herein. The Rendement Napole (RN) gene also affects pork quality and has been mapped to approximately 74cM from the centromere on SSC15 (Milan *et al.* 1995). This location is homeologous to human HSA2 and bovine BTA2. Again, the study herein found no QTL for cooking loss on BTA2. On the other hand, Malek *et al.* (2001) reported a QTL for a water holding capacity of raw pork on SSC2 which is homeologous to the QTL on BTA7 identified herein (Table 1), although there are no obvious candidate genes.

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**REFERENCES**

Kappes, S.M., Keele, J.W., Stone, R.T., McGraw, R.A., Sonstegard, T.S., Smith, T.P., Lopez-Corrales, N.L. and Beattie, C.W. (1997) *Genome Res.* **7**:235.

Knott, S.A., Elsen, J.M. and Haley, C.S. (1996) *Theoret. Appl. Genet.* **93**:71.

Lander, E. and Kruglyak, L. (1995) *Nature Genet.***11(3)**:241.

Lundstrom, K., Enfalt, A.C., Tornberg, E., Agerhem, H. (1998) *Meat Sci.* **48**:115.

Malek, M., Dekkers, J.C.M., Lee, H.K., Baas, T.J., Prusa, K., Huff-Lonergan, E., and Rothschild, M.F. (2001) *Mammalian Genome* 12:637.

Milan, D., Le Roy, P., Woloszyn, N., Caritez, J.C., Elsen, J.M., Sellier, P., Gellin, J. (1995) *Genet. Sel. Evol.* **27**:195.

Mitsumoto, M., Arnold, R.N., Schaefer, D.M., and Cassens, R.G. (1995) *J. Anim. Sci.* **73**:2289.

Perry, D., Shorthose, W.R., Ferguson, D.M. and Thompson, J.M. (2001a) *Aust. J. Exper. Agric.* **41**:953.

Perry, D., Thompson, J.M., Hwang, I.H., Butchers, A and Egan, A.F. (2001b) *Aust. J. Exper. Agric.* **41**:981.

Pitchford, W.S., Kruk, Z.A., Stephens, E.M., Speck, P.A. and Deland, M.P.B. (2002)*Anim. Prod. Aust.* **24**:177