Meat quality traits were unaffected by a quantitative trait locus affecting leg composition traits in Texel sheep[1]

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ABSTRACT: A QTL affecting leg muscle and fat traits has been identified within the New Zealand Texel population. The QTL maps to a region on OAR 2 with a two-marker haplotype test established at markers BULGE20 and BM81124. These markers encompass the likely position of Growth Differentiation Factor 8 (GDF8). The pleiotropic effects of this QTL on meat quality traits are tested. Objective measures of meat quality including pH, color (L*, a*, and b*), and tenderness (as assessed by Warner-Bratzler shear force measurements) were assessed on longissimus and semimembranosus muscles of 540 progeny from six Texel sires. Four of these sires were subsequently identified as segregating for leg muscle and fat traits. For these segregating sires, comparison of progeny that had inherited the favorable haplotype from their sire with those that had received the alternate haplotype revealed no significant differences in the meat quality traits assessed. This finding suggests that the muscling QTL does not have pleiotropic effects on meat quality. A general scan for meat quality QTL was carried out using genotype data for eight markers from FCB128 to RM356 flanking 122cM of OAR 2 using Haley-Knott regression. This analysis revealed two QTL for a single sire. A QTL detected in the region of Marker INRA40 for color L* mapped to a site close to the muscling QTL, but there was evidence to suggest it is at a distinct locus. The QTL in the region of Marker RM356 might map distal to Marker RM356, as no peak was observed. This QTL, which seems to affect pH, color a*, color b*, and Warner-Bratzler shear measurements, requires further characterization.

Key Words: Carcass Trait, Growth Differentiation Factor 8, Lamb, Meat Quality, Quantitative Trait Loci, Texel

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Introduction

The discovery of QTL affecting muscle and fat traits in sheep, together with the use of marker-assisted selection (**MAS**), offers the opportunity to improve rates of genetic gain for these economically important traits. Before widespread use of MAS, however, it must be ensured that a QTL does not have negative effects on other economically and biologically important traits, via direct pleiotropic effects, epistatic interactions, or linkage with other genes. Of particular importance for muscling QTL are possible associated effects on meat quality. Whether a QTL has pleiotropic or other effects on meat quality is influenced by the way in which the increased muscling is achieved.

A QTL affecting carcass muscle traits in Texel sheep on OAR 2 has now been reported independently by three groups (Laville et al., 2004; Walling et al., 2004; Johnson et al., 2005a). This QTL maps to a region that spans the location of Growth Differentiation Factor 8 (**GDF8** or myostatin); however, in preliminary work, Marcq et al. (2002) found no evidence for variation in the coding region of this gene. If GDF8 is the gene involved, extrapolation from cattle work suggests few negative changes in meat quality would be expected, as mutations in this gene predominately increase muscling through hyperplasia (Wheeler et al., 2001).

Marker-assisted selection for the muscling QTL may be implemented before the specific causative mutation is known and, therefore, the way in which increased

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muscling is achieved. Consequently, consideration of the effects of the QTL on meat quality must be considered independently. The objectives of this study were to use samples of LM and semimembranosus muscle obtained from the study described by Johnson et al. (2005a) to determine whether there was any association between the haplotype determined for the muscling QTL and meat quality traits and, additionally, to search for other QTL affecting meat quality traits in the OAR 2 region.

Materials and Methods

Animals

Six half-sib families were produced using four Texel (15, 429, 535, and 150) and two Texel × Coopworth sires (1170 and 1199). Five of the sires were either sons or grandsons of the foundation Texel sire (150), which also was used in the trial (see Johnson et al., 2005a for a more complete description of the relationships). Four of these sires (15, 429, 1170, and 1199) were informative for this study in that they were heterozygous for markers in the region of interest around GDF8. The sires were single-sire mated to ewes of unrelated breeds (Romney and Coopworth). Approximately 145 progeny were produced per sire, and power calculations indicated that only 90 progeny were required for measurements of the meat quality traits. Thus, meat quality data for the current analysis were collected from 540 lambs. For each sire, all progeny were born and raised together on pasture. Further details on the progeny and their management were given by Johnson et al. (2005b). The mean carcass weights over all sires were 16.7 and 17.6 kg for ewe and ram lambs, respectively.

Sample Collection

Lambs were slaughtered at two processing plants (see Johnson et al., 2005b for details). The lambs were held overnight at the processing plant before being slaughtered and were dressed under normal New Zealand commercial conditions. Postmortem treatment was such that tenderness standards were achieved according to New Zealand accelerated conditioning and aging standards (Chrystall et al., 1989). On the day following slaughter, the right leg and loin were packaged in plastic bags and stored for 1 to 5 mo at –18°C. Frozen legs were thawed at ambient temperature (10 to 20°C) for approximately 18 h before dissection and collection of samples for meat quality analysis.

Traits Analyzed

Objective measures of meat quality were made on LM and semimembranosus muscles. Tenderness was assessed using Warner-Bratzler shear force measurements as described by Purchas and Aungsupakorn (1993) following cooking of 25-mm steaks in a 70°C water bath for 90 min. Twelve shears of 13-mm × 13-mm cores (two shears per core) were made using a square blade for each muscle following chilling overnight. Cooking loss was determined as the difference in weight between the precooked and cooked steaks, expressed as a percentage of the precooked weight. Sarcomere length was determined on uncooked samples collected at the time of dissection using the laser diffraction method described by Cross et al. (1981). Color and ultimate pH were measured on uncooked samples collected at the time of dissection and frozen until analysis was carried out. Color was determined using a Minolta ChromaMeter (Minolta Co., Ltd., Osaka, Japan) calibrated to a white standard using the L*, a*, b* scale. Ultimate pH was determined using a spear-tip pH probe calibrated to pH 4 and 7 using buffer standards. Further details of methods used were described in Johnson et al. (2005b).

Genotyping

The gene GDF8 has not yet been mapped in sheep, but based on conserved synteny, it most likely maps to the region of microsatellite Marker BM81124 on OAR 2 (Smith et al., 1997; Maddox et al., 2002). Eight microsatellite markers in this region were genotyped across all progeny (FCB128, BM81124, BULGE20, INRA40, TEXAN2, ILST30, FCB20, and RM356), and a further microsatellite was genotyped for Sire 15 (TGLA10). The amplification procedure was described by Crawford et al. (1995). All distances are reported relative to Marker FCB128, which maps to approximately 99.4 cM from the start of OAR 2 (Maddox et al., 2002).

Statistical Analyses

Based on the diagnostic two-marker haplotype for increased muscling reported by Johnson et al. (2005a), an analysis-fitting haplotype inherited (favorable or other) along with sex in a GLM for the progeny of those sires that were heterozygous for the desirable haplotype (the informative sires) was carried out. This procedure enabled the estimation of the size and significance of meat quality differences between lambs with the favorable QTL for leg muscle and fat weight vs. those without it.

A further analysis involved a search for any meat quality QTL within the region genotyped. Analysis of data took place at 2-cM intervals along the 122-cM region of interest. The basis of the analysis was the Haley-Knott regression method (Haley and Knott, 1992), further adapted for use with outbred populations (Knott et al., 1996). The analyses were carried out in SAS using the GLM procedure (SAS Inst., Inc., Cary, NC). The probability (within sire) of the sire passing his paternally inherited allele was fitted in all models; the only other fixed effect fitted was sex. Other fixed effects, including slaughter group and birth rank, were initially fitted, but were not significant and were subse-

Table 1. Least squares means ± SE for meat quality traits showing the effects of inheriting the favorable haplotype with respect to muscle and fat weight in the leg (Johnson et al., 2005a) vs. other haplotypes for progeny of those sires that were heterozygous in the region of interest

Item	Favorable haplotype[a]	Other haplotype[b]	*P*-value	Change, %[c]
No. of individuals	117	181		
Semimembranosus				
Ultimate pH	5.58 ± 0.07	5.60 ± 0.06	0.82	–0.32
Color L* (lightness)	31.28 ± 0.38	31.37 ± 0.34	0.83	–0.28
Color a* (redness)	14.46 ± 0.22	14.58 ± 0.19	0.63	–0.78
Color b* (yellowness)	6.18 ± 0.11	6.27 ± 0.10	0.45	–1.44
Sarcomere length, μm	1.39 ± 0.04	1.44 ± 0.04	0.33	–3.31
Cooking loss, %	37.07 ± 0.14	36.82 ± 0.12	0.10	0.68
Warner-Bratzler peak force, kg	10.41 ± 0.27	9.95 ± 0.24	0.12	4.66
LM				
Ultimate pH	5.64 ± 0.04	5.68 ± 0.04	0.30	–0.84
Color L*	33.03 ± 0.24	33.42 ± 0.21	0.14	–1.16
Color a*	14.20 ± 0.16	14.38 ± 0.14	0.31	–1.25
Color b*	6.19 ± 0.12	6.39 ± 0.10	0.11	–3.18
Sarcomere length, μm	1.76 ± 0.01	1.77 ± 0.01	0.68	–0.38
Cooking loss, %	30.23 ± 0.25	30.18 ± 0.22	0.86	0.16
Warner-Bratzler peak force, kg	6.32 ± 0.18	6.04 ± 0.16	0.16	4.54

[a]Alleles C and I at markers BM81124 and BULGE20.
[b]Alleles other than C and I at markers BM81124 and BULGE20.
[c]Change between favorable haplotype and other haplotype.

quently excluded from the final model. Interactions between the sire allele probability and sex were tested, but they were nonsignificant. Haley-Knott regression *F*-tests are reported as $-\log_{10}$ transformed probability values. Further details of the QTL analysis are described in Johnson et al. (2005a).

Significance thresholds were estimated at the genome-wide level using the formulas of Lander and Kruglyak (1995) and estimated at the experiment-wide level using the permutation test described by Doerge and Churchill (1996) using 10,000 replicates for reasons described by Johnson et al. (2005a). Confidence intervals for the position of the QTL peak were generated using the bootstrapping method of Visscher et al. (1996) with 500 replicates. Estimated effects are presented on both the measurement scale and in residual (adjusted for nongenetic effects) SD (σ_P) units.

Results

The region of interest for the leg muscle and fat trait QTL detected by Johnson et al. (2005a) and Laville et al. (2004) is between Markers BM81124 and BULGE20 (52 to 54 cM from Marker FCB128). Johnson et al. (2005a) showed that a consistent haplotype exists at these markers, which explains the results observed. As discussed by Johnson et al. (2005a), only four of the sires used in the study were heterozygous for markers in the region of GDF8; therefore, detection of QTL effects in this region was limited to these sires. For three of these sires (15, 1170, and 1199), the favorable haplotype (increased muscling and decreased fat content) was paternally inherited, whereas for the fourth (429), it was maternally inherited.

Comparison of lambs that inherited the favorable haplotype at Markers BM81124 and BULGE20 with those with other haplotypes inherited from their sire revealed no significant differences in meat quality characteristics (Table 1).

The general scan for meat quality QTL within the region revealed two potential QTL at 56 and 122 cM. A QTL affecting color L* of semimembranosus and LM was detected at 56 cM for sire 429 (Table 2). Although the peaks did not reach the Lander and Kruglyak genome-wide suggestive threshold, they reached the 95% threshold derived from the experiment-wide permutation tests. The size of the QTL effect was approximately 0.6 of 1 SD; the phase that results in the increased muscling (Johnson et al., 2005a) had the lower values (i.e., meat that reflects less light and is, therefore, darker). No other meat quality traits for this sire, or any other sire, reached any of the significance thresholds in this region.

There was evidence from the progeny of Sire 429 for a QTL affecting meat quality traits, including pH, color a* (redness), and color b* (yellowness), for both semimembranosus and LM. Progeny of Sire 429 also showed a QTL affecting Warner-Bratzler shear force measurements for the semimembranosus muscle in the region of Marker RM356, which is approximately 65 cM downstream from the position of the muscling QTL. The plots for the individual quality traits for this sire are shown in Figures 1 and 2, and further details of the significant peaks are provided in Table 2, including the size of the

Table 2. Details of maximum peaks for progeny of Sire 429 in a Texel-cross half-sib QTL analysis for a region of OAR 2—measurements of meat quality made on semimembranosus and longissimus muscles

Trait	Mean[a]	σ_P	Estimate ± SE[b]	Estimate in SD units[c]	Permutation[d] 95%	Permutation[d] 99%	Max-$\log_{10}$ probability[e]	Relative position, cM[f]	Confidence interval, cM[g]
Semimembranosus									
Ultimate pH	5.6	0.1	0.1 ± 0.02	0.6	2.1	2.8	2.3*	120	2 to 122
Color L* (lightness)	31.8	1.4	0.8 ± 0.3	0.6	2.1	2.8	2.2*	56	29 to 122
Color a* (redness)	14.5	1.1	–0.7 ± 0.3	0.7	2.0	2.7	2.3*	110	2 to 122
Color b* (yellowness)	6.2	0.6	–0.4 ± 0.2	0.6	2.1	2.8	2.1*	108	15 to 122
Sarcomere length, μm	1.5	0.6	0.2 ± 0.2	0.4	2.0	2.8	1.0	82	
Cooking loss, %	37.0	1.3	–0.7 ± 0.3	0.5	2.1	2.8	1.5	62	
Warner-Bratzler peak force, kg	12.0	2.6	1.6 ± 0.7	0.6	1.6	2.1	1.7*	122	28 to 122
LM									
Ultimate pH	5.6	0.1	0.1 ± 0.03	0.5	1.9	2.5	1.7	122	
Color L*	34.1	1.7	1.0 ± 0.4	0.6	1.8	2.4	2.0*	56	9 to 104
Color a*	14.5	1.6	–0.9 ± 0.3	0.6	2.3	3.2	2.6*	122	15 to 122
Color b*	6.5	0.9	–0.5 ± 0.2	0.6	2.0	2.8	2.0*	122	15 to 122
Sarcomere length, μm	1.8	0.1	–0.02 ± 0.02	0.4	1.9	2.5	0.7	122	
Cooking loss, %	30.6	1.5	–0.6 ± 0.4	0.4	2.1	2.9	0.9	104	
Warner-Bratzler peak force, kg	6.9	1.6	0.8 ± 0.4	0.5	1.7	2.3	1.5	96	

[a]Within-sire, raw, unadjusted phenotypic mean of the trait ± σ_P.
[b]Estimate of size of the substitution effect between the paternally inherited sire haplotype and alternative haplotypes. For this sire, the alternative (maternally inherited) haplotype was the haplotype associated with the increased muscling QTL (Johnson et al., 2005a).
[c]Magnitude of the QTL peak = estimate/σ_P.
[d]–$\log_{10}$ probability thresholds (derived by permutation tests with 10,000 replicates) determined for 95% and 99% confidence levels.
[e]The significance of the QTL peak in terms of –$\log_{10}$ of the nominal probability.
[f]Position of the QTL peak relative to Marker FCB128 in cM.
[g]95% confidence intervals were derived for the position in cM by bootstrapping with 500 replicates for traits for which significant QTL were found.
*Result reached the permutation test 95% threshold.

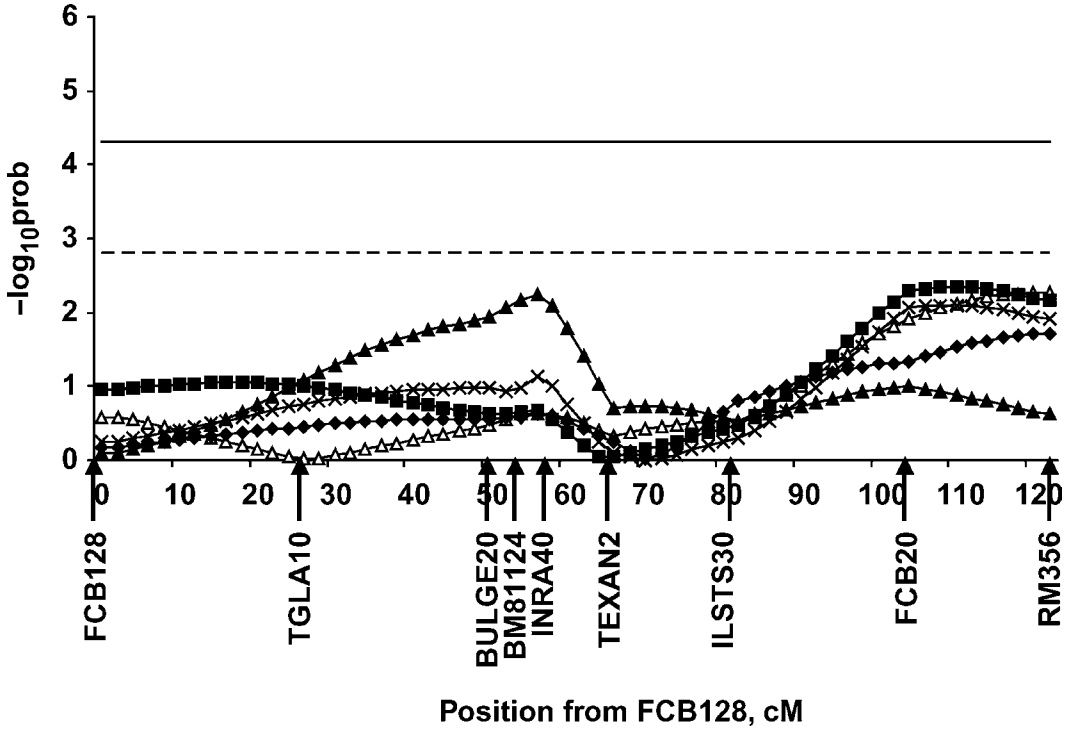

Figure 1. Probability (–$\log_{10}$prob: the negative logarithm of *P*) curves for Sire 429 half-sib Texel-cross population for a region of OAR 2 for semimembranosus meat quality measurements. Traits shown are ultimate pH (△-△-△), color L* (▲-▲-▲), color a* (■-■-■), color b* (×-×-×), and Warner Bratzler peak force (◆-◆-◆). Genome-wise thresholds derived from Lander and Kruglyak (1995) are suggestive (- - -) and genome-wise $P < 0.05$ (——).

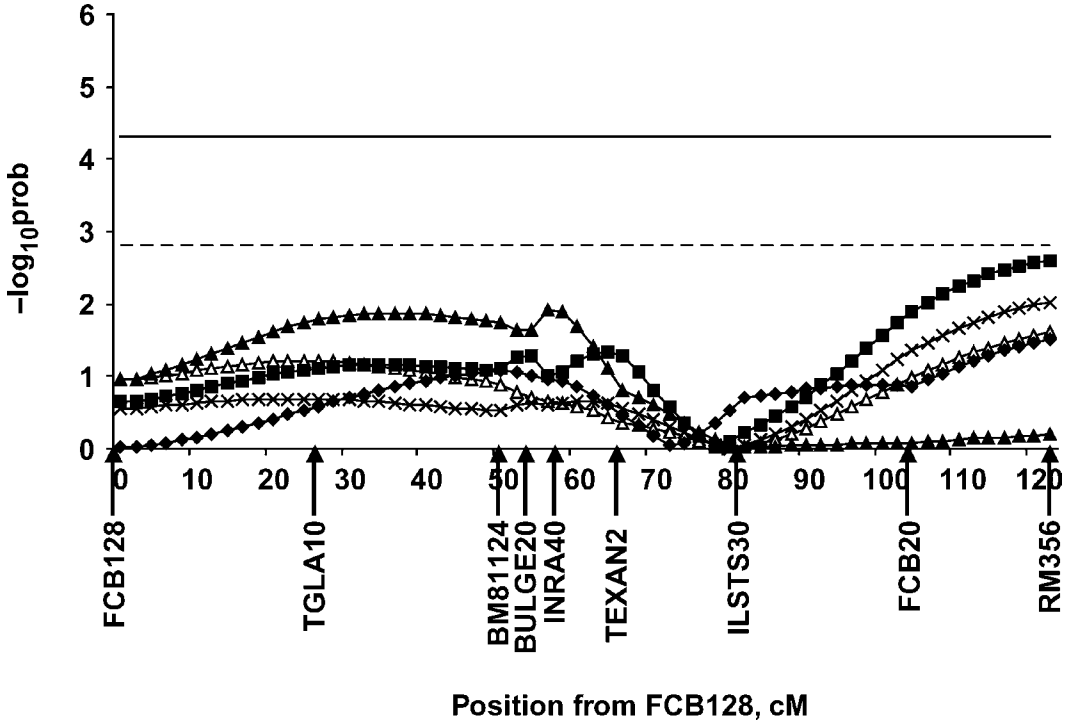

Figure 2. Probability ($-\log_{10}$prob: the negative logarithm of *P*) curves for Sire 429 half-sib Texel-cross population for a region of OAR 2 for LM meat quality measurements. Traits shown are ultimate pH (△-△-△), color L* (▲-▲-▲), color a* (■-■-■), color b* (×-×-×), and Warner Bratzler peak force (◆-◆-◆). Genome-wise thresholds derived from Lander and Kruglyak (1995) are suggestive (- - -) and genome-wise $P < 0.05$ (——).

effect and significance of the peaks. Although the peaks did not reach the Lander and Kruglyak genome-wide suggestive threshold, they reached the 95% threshold derived by the more accurate permutation tests. As indicated in the figures, Marker RM356 was the rightmost marker genotyped, and the significance level of this peak was still increasing for several traits at this marker. The size of the QTL effect was approximately 0.6 to 0.7 of 1 SD. The combined effects of the phase that resulted in the increased muscling (which for Sire 429 was the maternally inherited haplotype; Johnson et al., 2005a) were to lower ultimate pH, increase the redness and yellowness of the meat, and decrease the Warner-Bratzler shear force (thereby making the meat more tender).

Discussion

Evidence for QTL in the GDF8 Region

Detection of a QTL affecting meat quality traits in the region of the muscling QTL on OAR 2 indicates that either the underlying muscling gene has pleiotropic effects on meat quality or that there is a gene within a tight linkage group with the muscling gene, which has effects on meat quality. It is important to resolve this issue before widespread commercial use of MAS for this muscling QTL.

Increased muscling occurs either because of hyperplasia (increase in muscle fiber number) or hypertrophy (increase in muscle fiber size). With hypertrophy and increased protein accretion, the increase can be due to an increase in protein synthesis with no change in protein degradation, a decrease in protein degradation with no change in protein synthesis, or a combination of both (Koohmaraie et al., 2002). It is only the decrease in protein degradation that will likely have an effect on meat quality because the same factors that regulate protein degradation also are involved with the regulation of postmortem meat tenderness (Koohmaraie et al., 2002).

The most likely candidate gene for the lamb phenotypes observed by Johnson et al. (2005a) and Laville et al. (2004) is GDF8. The normal role of GDF8 is that of a negative regulator of skeletal muscle growth; however, mutations to the protein coding region of the gene have been shown to either turn down or turn off this regulatory role, resulting in an increased muscling phenotype in cattle (Charlier et al., 1995; Varga et al., 2003), which primarily is due to increased protein synthesis. Therefore, based on the hypothesis of Koohmaraie et al. (2002), if GDF8 is the gene involved, it would not be expected that the QTL would have negative effects on meat quality. Given that the evidence for GDF8 being the gene involved is inconclusive (Marcq et al., 2002) and that it could be another gene, testing for an association is important. This is especially true given that the only two increased muscling phenotypes in sheep identified to date, Callipyge and the rib-eye muscling

locus (previously known as Carwell), are both associated with decreased meat tenderness (Koohmaraie et al. 1995; Jopson et al. 2001). For the Callipyge phenotype, Koohmaraie et al. (1995) reported that, 21 d after slaughter, Warner Bratzler shear-force measures on the LM for nonCallipyge and Callipyge were 3.3 and 8.2 kg, respectively. Albeit not as severe, the rib-eye muscling QTL, also identified in Dorsets, is associated with decreased tenderness relative to normal lambs, although aging of the meat overcomes the problem (Jopson et al., 2001). For Callipyge, it has been shown that this decrease in tenderness occurs because the underlying mutation results in increased calpastatin levels, which cause a decrease in protein degradation in living muscle (Lorenzen et al., 2000) and decrease the extent of postmortem proteolysis and, thereby, tenderization (Koohmaraie et al., 1995).

The present results failed to provide any clear evidence that the OAR 2 QTL associated with more muscle and less fat in the leg reported by Johnson et al. (2005a) has any negative effect on meat quality. In particular, there were no significant differences between the favorable vs. nonfavorable haplotype on meat quality. A QTL of small effect for color L* was detected in a QTL search at 56 cM for Sire 429; this is outside the region of the conserved favorable haplotype, although the confidence interval does overlap this region. The color QTL was only detected, however, in one of the four sires, which further suggests that the color QTL is at a locus distinct from the muscling QTL.

Evidence for QTL in the Region of Marker RM356

Evidence was found for a QTL affecting ultimate pH, color, and tenderness for Sire 429 in the region of, or distal to, Marker RM356, which is 65 cM from the region predicted to contain GDF8 in sheep. This meat quality QTL does not seem to be associated with any muscling phenotype in Sire 429 in the region of Marker RM356. The maternally inherited haplotype of Sire 429 at Markers OARFCB20 and RM356 is unique compared with the haplotypes of other sires, explaining why the QTL was only detected for this sire. The improved phenotype (lower Warner Bratzler values) is in the same phase as the favorable muscling haplotype in the region of GDF8. The current work is insufficient to identify the frequency and association of these two QTL, but the limited evidence suggests the second QTL is present at a different frequency. The current lack of knowledge and the magnitude of the effect on meat tenderness suggests that simultaneous monitoring of the RM356 region also should be undertaken during commercial selection.

Nebulin and titin are intramyofibril proteins whose degradation is involved in postmortem tenderization of meat (Koohmaraie, 1996). Both proteins map to the region of interest; nebulin maps in cattle close to Marker OARFCB20 (Snelling et al., 2005; a marker used in the current study) and based on conserved synteny with HSA2, Titin maps close to GDF8. How mutations in these genes might directly affect pH and color is unclear; therefore, the genes encoding these proteins are less likely to be involved directly.

To date, the only genes identified that affect pH and color along with tenderness have been in pigs. Mutations in the Ryanodine Receptor (**RYR1**; previously referred to as the halothane [Hal] gene) result in inferior meat quality, including lower pH and poorer color (Pommier and Houde, 1993). However, RYR1 maps to SSC6 and HSA19 (Yerle et al., 1990) and is, therefore, based on conserved synteny between the species, unlikely to map to OAR 2. Mutations in the protein kinase, AMP-activated, gamma 3 noncatalytic subunit (**PRKAG3**) gene in pigs result in variation in pH, meat color, water-holding capacity, cooking loss, and tenderness. The RN^- mutation at this gene results in extremely inferior meat quality; other mutations to the PRKAG3 gene also affect meat quality but to a lesser degree (Milan et al., 2000; Ciobanu et al., 2001; Andersson, 2003). The PRKAG3 gene maps to 219 mega base pairs on HSA2, telomeric to GDF8, and it is flanked by SCLA11A1 and INHA (219 and 220 Mbp; http://genome.ucsc.edu; Human May 2004 Assembly). The SCLA11A1 and INHA have been mapped to 256.6 and 258.1 cM, respectively, on OAR 2 (http://rubens.its.unimelb.edu.au/~jillm/jill.htm; Maddox v4.3 sheep map). Marker RM356 maps to 211 cM on OAR 2 on the Maddox v4.3 sheep map, indicating that the RN locus maps 45 cM telomeric to RM356. Whether RN is the gene underlying the meat quality QTL locus is unclear based on the current work, and further investigation is warranted.

Conclusion

The evidence presented from this study strongly suggests that the muscling QTL identified in the region of GDF8 by Johnson et al. (2005a) has no detectable negative associations with objectively assessed meat quality traits. This finding means that the meat industry will support the use of such a QTL, as the product will require less trimming and cuts will have a greater muscle depth at the same carcass weight. The knowledge to date means that industry implementation of the muscling QTL can commence in terminal sires; however, before widespread MAS occurs in dam lines, further consideration of the mode of inheritance (dominant vs. additive) and the effects of the QTL on nonmeat-related traits, such as reproduction, wool growth, disease resistance, and lamb survival, also should be considered. The results of the current study have also revealed possible QTL affecting objectively assessed meat quality characteristics in a region of OAR 2 distinct from the predicted position of GDF8. A potential candidate gene for this QTL is PRKAG3, as mutations in this gene have been shown to affect meat quality traits in pigs; however, further localization of this QTL is required, along with mapping of the PRKAG3 gene in sheep. Its magnitude is sufficient that commercial MAS

for other QTL on OAR 2 also should monitor linkage with this region.

Literature Cited

Andersson, L. 2003. Identification and characterization of the AMPK $\gamma 3$ mutation in the pig. Biochem. Soc. Trans. 31:232–235.

Charlier, C., W. Coppieters, F. Farnir, L. Grobet, P. L. Le Roy, C. Michaux, M. Mni, A. Schwers, P. Vanmanshoven, R. Hanset, and M. Georges. 1995. The mh gene causing double-muscling in cattle maps to bovine chromosome 2. Mamm. Genome 6:788–792.

Chrystall, B. B., C. E. Devine, G. R. Longdill, C. O. Gill, J. E. Swan, and G. V. Petersen. 1989. Trends and developments in meat processing. Pages 185–207 in Meat Production and Processing. R. W. Purchas, B. W. Butler-Hogg, and A. S. Davies, eds. Simon Print Ltd., Palmerston North, New Zealand.

Ciobanu, D., J. Bastiaansen, M. Malek, J. Helm, J. Wollard, G. Plastow, and M. Rothschild. 2001. Evidence for new alleles in the protein kinase adenosine monophosphate activated γ_3-subunit gene associated with low glycogen content in pig skeletal muscle and improved meat quality. Genetics 159:1151–1162.

Crawford, A. M., K. G. Dodds, A. J. Ede, C. A. Pierson, G. W. Montgomery, H. G. Garmomway, A. E. Beattie, K. Davies, J. F. Maddox, S. W. Kappes, R. T. Stone, T. C. Nguyen, J. M. Penty, E. A. Lord, J. E. Broom, J. Buitkamp, W. Schwaiger, J. T. Epplen, P. Matthew, M. E. Matthew, D. J. Hulme, K. J. Beh, R. A. McGraw, and C. W. Beattie. 1995. An autosomal genetic linkage map of the sheep genome. Genetics 140:703–724.

Cross, H. R., R. L. West, and T. R. Dutson. 1981. Comparison of methods for measuring sarcomere length in beef semitendinosus muscle. Meat Sci. 5:261–266.

Doerge, R. W., and G. A. Churchill. 1996. Permutation tests for multiple loci affecting a quantitative character. Genetics 142:285–294.

Haley, C. S., and S. A. Knott. 1992. A simple regression method for mapping quantitative trait loci in line crosses using flanking markers. Heredity 69:315–324.

Johnson, P. L., J. C. McEwan, K. G. Dodds, R. W. Purchas, and H. T. Blair. 2005a. A directed search in the region of GDF8 for quantitative trait loci affecting carcass traits in Texel sheep. J. Anim. Sci. 83:1988–2000.

Johnson, P. L., R. W. Purchas, J. C. McEwan, and H. T. Blair. 2005b. Carcass composition and meat quality differences between pasture-reared ewe and ram lambs. Meat Sci. 71:383–391.

Jopson, N. B., G. B. Nicoll, J. M. Stevenson-Barry, S. Duncan, G. J. Greer, W. E. Bain, E. M. Gerrard, B. C. Glass, T. E. Broad, and J. C. McEwan. 2001. Mode of inheritance and effects on meat quality of the rib-eye muscling (REM) QTL in sheep. Proc. Assoc. Advancement Anim. Breeding Genet., Queenstown, New Zealand 14:111–114.

Knott, S. A., J. M. Elsen, and C. S. Haley. 1996. Methods for multiple-marker mapping of quantitative trait loci in half-sib populations. Theor. Appl. Genet. 93:71–80.

Koohmaraie, M. 1996. Biochemical factors regulating the toughening and tenderization process of meat. Meat Sci. 43:S193–S201.

Koohmaraie, M., M. P. Kent, S. D. Shackelford, E. Veiseth, and T. L. Wheeler. 2002. Meat tenderness and muscle growth: Is there any relationship? Meat Sci. 62:345–352.

Koohmaraie, M., S. D. Shackelford, T. L. Wheeler, S. M. Lonergan, and M. E. Doumit. 1995. A muscle hypertrophy condition in lamb (Callipyge): Characterization of effects on muscle growth and meat quality traits. J. Anim. Sci. 73:3596–3607.

Lander, E., and L. Kruglyak. 1995. Genetic dissection of complex traits: Guidelines for interpreting and reporting linkage results. Nat. Genet. 11:241–247.

Laville, E., J. Bouix, T. Sayd, B. Bibé, J. M. Elsen, C. Larzul, F. Eychenne, F. Marcq, and M. Georges. 2004. Effects of a quantitative trait locus for muscle hypertrophy from Belgian Texel sheep on carcass conformation and muscularity. J. Anim. Sci. 82:3128–3137.

Lorenzen, C. L., M. Koohmaraie, S. D. Shackelford, F. Jahoor, H. C. Freetly, T. L. Wheeler, J. W. Savell, and M. L. Fiorotto. 2000. Protein kinetics in Callipyge lambs. J. Anim. Sci. 78:78–87.

Maddox, J. F., I. Franklin, C. D. Bottema, U. DeSilva, D. Adelson, C. Diez-Tason, G. Nattrass, C. Gill, G. Webb, K. G. Dodds, and D. Vaiman. 2002. An enhanced sheep linkage map comprising 240 gene and EST associated markers. Proc. 28th Int. Conf. Anim. Genet., Gottingen, Germany 28:108.

Marcq, F., C. Larzul, V. Marot, J. Bouix, F. Eychenne, E. Laville, B. Bibé, P. L. Leroy, M. Georges, and J. M. Elsen. 2002. Preliminary results of a whole-genome scan targeting QTL for carcass traits in a Texel × Romanov intercross. Proc. 7th World Congr. Genet. Appl. Livest. Prod., Montpellier, France. CD-ROM Communication No. 02–14.

Milan, D., J. T. Jeon, C. Looft, V. Amarger, A. Robic, M. Thelander, C. Rogel-Gaillard, S. Paul, N. Iannuccelli, L. Rask, H. Ronne, K. Lundstrom, N. Reinsch, J. Gellin, E. Kalm, P. L. Roy, P. Chardon, and L. Andersson. 2000. A mutation in PRKAG3 associated with excess glycogen content in pig skeletal muscle. Science 288:1248–1251.

Pommier, S. A., and A. Houde. 1993. Effect of the genotype for malignant hyperthermia as determined by a restriction endonuclease assay on the quality characteristics of commercial pork loins. J. Anim. Sci. 71:420–425.

Purchas, R. W., and R. Aungsupakorn. 1993. Further investigations into the relationship between ultimate pH and tenderness for beef samples from bulls and steers. Meat Sci. 34:163–178.

Smith, T. P., N. L. Lopez-Corrales, S. M. Kappes, and T. S. Sonstegard. 1997. Myostatin maps to the interval containing the bovine mh locus. Mamm. Genome 8:742–744.

Snelling, W. M., E. Casas, R. T. Stone, J. W. Keele, G. P. Harhay, G. L. Bennett, and T. P. L. Smith. 2005. Linkage mapping bovine EST-based SNP. BioMed Central Genomics 6:74.

Varga, L., G. Muller, G. Szabo, O. Pinke, E. Korom, B. Kovacs, L. Patthy, and M. Soller. 2003. Mapping modifiers affecting muscularity of the myostatin mutant ($Mstn^{Cmpt\text{-}dl1Abc}$) compact mouse. Genetics 165:257–267.

Visscher, P. M., R. Thompson, and C. S. Haley. 1996. Confidence intervals in QTL mapping by bootstrapping. Genetics 143:1013–1020.

Walling, G. A., P. M. Visscher, A. D. Wilson, B. L. McTeir, G. Simm, and S. C. Bishop. 2004. Mapping of quantitative trait loci for growth and carcass traits in commercial sheep populations. J. Anim. Sci. 82:2234–2245.

Wheeler, T. L., S. D. Shackelford, E. Casas, L. V. Cundiff, and M. Koohmaraie. 2001. The effects of Piedmontese inheritance and myostatin genotype on the palatability of longissimus thoracis, gluteus medius, semimembranosus, and biceps femoris. J. Anim. Sci. 79:3069–3074.

Yerle, M., J. Gellin, M. Dalens, and O. Galman. 1990. Localization on pig chromosome 6 of markers GPI, APOE, and ENO1, carried by human chromosomes 1 and 19, using in situ hybridization. Cytogenet. Cell Genet. 54:86–91.