A Directed Search for QTL Affecting Carcass Composition Traits in Texel Sheep

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Abstract

In New Zealand moves towards lean-meat yield- and meat quality-based payment of lamb carcasses, which more accurately reflect consumer preferences, mean that breeders and producers will need to change their selection objectives and management practices to maximise returns. This thesis investigates approaches to achieving increased meat yields, while not detrimentally affecting meat quality.

The main objective was to search for Quantitative Trait Loci (QTL) affecting carcass composition traits in Texel sheep, in the region of Growth Differentiation Factor 8 (GDF8) on ovine chromosome 2. Dissection and meat quality data for legs and loins were collected for 90 Texel-cross progeny from each of six Texel or Texel-cross sires. All animals were genotyped for seven markers around GDF8. A QTL which increased leg muscle by 5-8% and decreased leg fat by 10-15% was identified for four of the six sires in the region of markers BM81124 and BULGE20. The two sires for which no QTL was detected, were homozygous and therefore uninformative at these markers. The QTL did not negatively affect meat quality. There was only limited evidence for a QTL affecting loin composition traits.

Candidate genes to explain the QTL effect, in addition to GDF8, were sought, based on the conserved synteny between the ovine and human genomes, but none were identified. A number of genes in the region are poorly documented, and new genes are still being mapped to the region so a candidate gene could yet emerge.

The progeny data set when analysed for sex differences, revealed that, relative to ewe lambs, meat from the ram lambs had 10% higher Warner-Bratzler shear values, was less red, and for *M. longissimus*, had 28% more samples that exceeded the "high pH" threshold of 5.8. These negative meat quality aspects for ram lambs need further investigation to determine their commercial importance.

Future research on the putative QTL for leg and loin composition should assess its effects on other parts of the carcass, and its effect in lambs homozygous for the QTL. Additionally its position needs to be more closely defined, and ideally the specific gene or genes involved identified, before industry implementation is carried out.

Dedicated to my father Tom Johnson,
who inspired me to become an animal scientist.
I know you would be so proud of this work.

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Chapter 1

Introduction

Lamb meat exported from New Zealand, which makes up over 95% of the total produced, accounts for approximately 9.7% of New Zealand's export earnings (Anon, 2002). In overseas markets there is consumer pressure to improve the attributes of lamb (and other meat products), with two particularly important attributes being:-

- Increased amounts of muscle in the product relative to fat because this:
  - Provides the consumer with improved value for money.
  - Heeds health warnings to lower the amount of fat in the diet.
- Consistent quality in terms of colour and tenderness because:
  - Colour provides a visual appeal in the important chilled supermarket trade.
  - Tenderness dictates whether a consumer will make repeat purchases.

Currently these market signals do not efficiently filter through to the on-farm producer, or to the stud breeder who makes breeding decisions. Meat processors have become increasingly aware of this consumer pressure, but the costs of developing and implementing suitable technology, have previously limited their ability to pass on market signals to lamb producers through accurate payment systems. New Zealand meat processors are currently addressing this issue with several implementing technology that will provide information on lean-meat yield and meat quality.

The resulting changes to payment systems, mean that the "ideal" lamb, which will return the highest price per kilogram when slaughtered, is likely to differ from that which achieves the highest returns under the current payment system. Therefore ram breeders will need to reassess their ram selection criteria, and commercial producers will also have to reconsider their management systems (e.g. the gender of lambs and how they are managed).

The main traits of interest in this thesis are those associated with proportions of muscle and fat and those associated with meat quality. There are a variety of ways in which muscle and fat traits can be expressed, independently and relative to one another, but those of most interest are the muscle-related traits. There are a number of measures relating to muscle that can be calculated, including the muscle to bone ratio; the percent muscle in a cut (lean-meat yield); muscle distribution; and muscularity. These traits tend to be highly correlated. In order to simplify this aspect of the overall thesis topic,

the term muscling is used throughout this thesis to generalise the description of the phenotype of interest. This phenotype is likely to involve an increased muscle to bone ratio, increased muscle percent and increased muscularity.

There are a number of existing options available to assist breeders, to aid in identification of superior meat animals and enhance genetic progress. These include: the use of BLUP generated breeding values in conjunction with sire-referencing schemes; progeny tests; effective use of ultrasound and CT technology; and efficient breeding scheme design. An emerging technology is Marker Assisted Selection (MAS). Marker Assisted Selection involves selecting animals based on a DNA haplotype (combination of alleles) associated with improved production. In the first instance it requires the detection of Quantitative Trait Loci (QTL), which are regions of DNA where different variants are associated with different levels of a trait. Marker Assisted Selection is likely to be of most benefit for traits that are difficult to measure, sex limited or require slaughter of animals in order to measure phenotypes (Meuwissen & Goddard, 1996). Once a QTL has been identified, animals carrying the favourable haplotype can be selected at birth. Identification of a QTL that increases muscling would offer an alternative method of genetic improvement for ram breeders. Two such QTL that have already been identified in sheep are the Callipyge (identified in the USA; Freking et al. 1998a) and the Rib-Eye-Muscling loci (identified in Australia/New Zealand; Jopson et al. 2001). However, the increased muscling phenotype associated with the Callipyge phenotype is negatively associated with meat quality (Koohmaraie et al. 1995), so selection pressure to increase its frequency has not been applied by ram breeders.

The New Zealand sheep population includes a number of pure breeds and composites. The Texel was introduced into New Zealand in the late 1980's to provide an alternative terminal sire breed, largely because of its reputed superior muscling. However, within the New Zealand Texel breed there is considerable variation (McEwan, Pers comm.). Based on previous work, both within purebred Texels in New Zealand (Broad et al. 2000) and with Texel x Romanov crosses in France (Marcq et al. 1998) there is preliminary evidence for a QTL within the Texel breed affecting muscling in the region of the Growth Differentiation Factor 8 (GDF8 also known as Myostatin) gene on ovine chromosome 2. Mutations within the GDF8 gene, cause the double-muscling

phenotype in cattle breeds such as the Belgian Blue. If such a QTL within the Texel breed can be accurately defined, it offers the opportunity for MAS aimed at increasing the muscling attributes of the breed and composites derived from them.

The objectives of the work in this thesis were to:-

1. Review breed comparisons involving the Texel to determine how well the breed will meet the requirements as a terminal sire under yield- and quality-based payment systems (Chapter 2).
2. Review gender comparisons for muscling and meat quality for sheep, and assess the impact gender may have under yield- and quality-based payment systems (Chapter 2).
3. Review QTL design and analysis methodology and examples of meat-related QTL discovered in sheep and cattle (Chapter 2).
4. Use the lamb population created to address Objective 5 (below) to analyse gender differences (ram vs ewe) in muscling and meat quality traits (Chapter 3).
5. Use rams, descended from the animals identified in the work of Broad et al. (2000), mated across commercial dams to create half-sib populations, with the resulting progeny slaughtered, with detailed dissection of the leg and loin carried out, with associated tests for selected meat quality characteristics (Chapter 4). To genotype these animals in the region of GDF8 (ovine chromosome 2) and to carry out QTL analyses.
6. Identify candidate genes in the region of interest on chromosome 2 (Chapter 5).
7. Discuss options available to New Zealand ram breeders and lamb producers to improve returns when payment systems based on improved estimates of lean-meat yield and/or meat quality come into use (Chapter 6). To discuss how Marker Assisted Selection may play a role in future breeding schemes (Chapter 6).

Chapter 2

Literature Review

2.1 Introduction

There are a number of ways in which muscling attributes and meat quality can be improved, some of which are explored in this thesis, specifically through exploitation of breed differences, gender differences and Quantitative Trait Loci (QTL). The purpose of this literature review is to:

- Provide an overview of the New Zealand lamb industry.
- Review breed comparisons involving the Texel to determine its attributes in light of the proposed new payment system.
- Review gender comparisons, to summarise knowledge of their differences.
- Review QTL literature, specifically to:
    - Provide a history of the development of this technique.
    - Provide a summary of methodologies relevant to carrying out a QTL search.
    - Provide a review of existing QTL relevant to this thesis.

Conclusions for each section are provided at the end of the chapter.

2.2 The New Zealand Sheep Industry

2.2.1 Numbers

The sheep industry is important to New Zealand's export economy, contributing via both wool and meat exports. However, the greater returns experienced in recent years in the dairy industry, in recent years have resulted in the large scale conversion of sheep to dairy farms on better land classes. Along with the removal of farm subsidies (in 1982) this has resulted in a decrease in the total number of sheep in New Zealand from 70.3 million in 1982, to 44 million in 2001 (Statistics New Zealand, 2003). Breeding ewes make up approximately 70% of sheep numbers (Anon, 2002). Although the total number of lambs has likewise decreased, having peaked at 50 million in 1982, the relative decline, in the number of lambs produced, has not been as large (36 million produced in 2001) due to lambing percentages increasing from 100% in the 1980's to 120% in 2001 (Statistics New Zealand, 2003).

2.2.2 Industry Structure

The New Zealand sheep industry can broadly be separated into three production classes: specialised wool production, specialised meat production and dual purpose (wool and

meat production).

In 1996 the predominant breed in New Zealand was Romney (58%) followed by Coopworth (10%), Merino (7.0%), Perendale (6.6%), Corriedale (5.5%) and Halfbred (3.8%). No other breed reached 3.8% of the total flock. Three of these breeds are specialist fine wool producing breeds (Merino, Corriedale and Halfbred) and will not be considered further. The remaining three (Romney, Coopworth and Perendale) although they fall predominately into the dual-purpose class, also have a role in specialised meat production.

The Romney, Coopworth and Perendale breeds form the basis of the commercial ewe population in New Zealand. The majority of commercial sheep properties are hill country properties, where, for the first four years of a ewe's breeding life she is used to generate flock replacements. Once they have bred the fourth time they are often no longer suitable for hill country environments and are sold to low land breeders who mate these ewes with a terminal sire such as the Suffolk, Texel or Poll Dorset. On hill country properties there has been an emphasis on wool production. The decline in returns for mid-micron wool has led to less emphasis being placed on wool and more on lamb/meat production. With this increased emphasis on lamb production, it is likely that there will be an increase in the number of composite sheep breeds derived from crosses with breeds such as Finnish Landrace and East Friesian. These breeds were introduced into New Zealand in the 1980's and are known for increased lambing rates.

2.2.3 Lamb Meat

A New Zealand view point

Approximately 95% of New Zealand lamb is exported, with the rest sold in the local trade (Anon, 2002). In terms of the New Zealand economy, 11% of New Zealand's export dollars comes from the sale of lamb and sheep meat to overseas customers ($2,284 million FOB), of which 88% is made up of the sale of lamb, with the balance mutton (Anon, 2002). This ranks it second, only to dairy returns, as the largest pastoral contributor to New Zealands export economy (Anon, 2002).

An international view point

The world sheep meat trade market has changed considerably in recent decades. In

1961, only 44 countries contributed to sheep meat exports and New Zealands share was 36% of that. In comparison in 2000 84 countries contributed and New Zealands share had declined to only 20% (http://apps.fao.org). Prior to 1973, New Zealands major export market was Great Britain, with 2/3 of exports destined for that country (Boutonnet, 1999). In 2001 although the majority of New Zealand meat is destined for the European Union and to Great Britain; the Middle East, North America, Northern Asia and the Pacific are also important markets (Anon, 2002).

The changing face of lamb in New Zealand

From the first refrigerated exports of New Zealand lamb in 1882, feed back from the consumers in Great Britain, was that the product was too fat (Barton, 1982). Although individual meat companies developed subjective criteria for grading carcasses, it was not until the late 1970's that an objective grading system, based on carcass weight and fat depth 110mm from the mid-line in the region of the 12^{th} rib (GR) was implemented (Kirton & Johnson, 1979).

Development of an objective grading system, combined with genetic improvements and farm management has lead to changes in the distribution of carcasses within the grades. In the 1981-1982 season 32% of carcasses met the fat criteria for the Y class (GR fat depth 6-9mm, depending on carcass weight) and 60% the criteria for the P class (GR fat depth 6-12mm depending on carcass weight) with 1.4% in the T and F classes (GR fat depth greater than 12mm). In comparison in the 2001-2002 season 55.6% of lambs met the fat grading criteria for the Y class and 25.6% the criteria for the P class, although there were 4.8% in the T and F classes (Anon, 2002). In terms of carcass weight the average increased from 13.3kg in the 1981-1982 season, to 16.9kg in the 2001-2002 season (Anon, 1982, 2002). There has been a shift towards heavier, leaner carcasses.

There have been significant changes in the level of processing of export carcasses, and the treatment and storage of the resulting cuts. In 1981, 84% of export weight was made up of whole carcasses, with only 15% as bone-in cuts and 1% as boneless cuts (Anon, 1982). In 2001, only 8% of weight exported was whole carcasses, with 64% of weight as bone-in cuts and 10% as boneless cuts (Anon, 2002). There has also been a move away from frozen shipment, to the export of chilled product which was made possible because of research into extending the storage time of chilled products.

Current and future market requirements and considerations

There are diverse requirements for the type of carcass or cut to be supplied for a particular overseas market. For example, light-weight carcasses (or cuts) are sought by the Middle East (<13kg carcass), whilst the USA seeks heavy lamb carcasses (or cuts) (>20kg carcass). Irrespective of the carcass weight ranges desired by the different markets, consumers are increasingly demanding improved product, both in terms of perceived value for money and eating quality (which includes food safety concerns).

Like in many of New Zealand's European markets, there has been a downwards trend in per capita consumption of red meat in the United Kingdom (DEFRA, 2000) as is shown in Figure 2.1. Consumers purchasing red meat, are becoming increasingly particular about what they will purchase, with consumers preferring lean, quality product, particularly from the high price cuts (e.g. French Rack which is an 8 rib loin cut). This is in part due to health warnings about the amount of fat consumed in diets, but is also driven by the improved value for money achieved by buying lean product. Consumers are also demanding a consistently tender and attractive product.

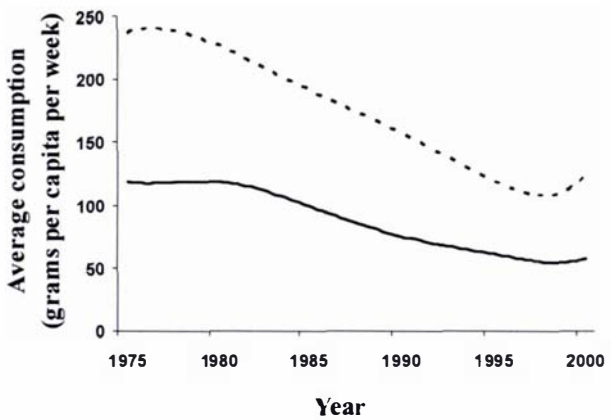


Figure 2.1 Average consumption per capita of red meat in the United Kingdom. ---- Beef; —— Lamb (taken from DEFRA (2000))

The current grading systems in New Zealand do not accurately reflect consumer demands. In an attempt to send accurate market signals to producers, New Zealand meat companies have been investigating saleable yield-based payment (Chalmers, 2003). Although not implemented at the time of this review meat companies will progressively introduce such systems in the 2003-2005 lamb seasons, with payment on such systems commencing in the 2005-2006 season. Although the exact means of

assessing yield are commercially sensitive, it is likely to involve image-based scanning techniques (Clarke et al., 1999).

A consistent, quality product is currently somewhat harder to reward, however, New Zealand meat companies appear committed to addressing the issue. In 1997 the New Zealand sheep and beef industry introduced the "New Zealand Beef and Lamb Quality Mark Standards", to set standards for animal welfare, meat eating quality, microbiological quality and storage life (Frazer, 1997). To qualify for the "Beef and Lamb Quality Mark" the product must be from animals of New Zealand origin, be within certain age categories and not treated with growth promotants. For lamb tenderness, the mean shear force of the product should average 8kgF or less with 95% of samples less than 11kgF at the point of retail sale as measured with the MIRINZ Tenderometer (Frazer, 1997). It seems likely, however, that many key markets will also require the implementation of a traceability system (Shackell et al., 2001). DNA-based traceability systems will allow consumer to farm-of-origin tracing of product, allowing feedback from consumer to producer of the quality of the product (Shackell et al., 2001).

For producers to make better financial returns, there will be increased emphasis on the type of lamb produced. This will include, whether it meets strict quality criteria and whether its composition will be highly valued under a yield-based payment system. Most improvements in meat quality are likely to arise from improved management of animals prior to slaughter, however, genetic improvement will also play a role.

2.3 The Texel breed as an Option for Prime Lamb Production

Throughout the world, there are over 250 different breeds of sheep (Ponting, 1980). Few can be considered true terminal sire breeds that have above average growth and muscling characteristics. In New Zealand there are eight main terminal sire breeds:- Suffolk, Texel, Poll Dorset, South Suffolk, Dorset Down, Southdown, Hampshire and Oxford (Meadows, 1997). The Texel is reputed as having the highest level of muscling. The purpose here is to provide a background to this breed, and review the published trials that have compared the Texel with other breeds, and to consider how well the Texel may perform under yield- and quality-based payment systems.

2.3.1 History

A comprehensive review of the development of the breed, based on local documents and knowledge was made by Visscher and Bekedam (1984). Some of the key points from that document are summarised in this section.

The Texel breed originated on the island of Texel, one of the North-Western Islands off the Netherlands. The Texel has been documented since Roman times, and belonged to the group of white-faced, short-tailed marsh sheep kept along the coast from Denmark to Northern France. This "old" Texel was described as late maturing fine boned, short and round ribbed, with well developed hindquarters, it also had good milk production with ewes sometimes even milked after the weaning of lambs at 6-8 weeks of age. Selection against twinning was practised in these animals and they were not prolific.

From 1846 there were numerous attempts at breed improvement, however, the process which led to the "modern" Texel involved various crosses to long woolled breeds (Lincoln, Leicester, Wensleydale, Cotswold and Hampshire Down) in the early 1900's. In 1909 the Union for improvement of sheep breeding in North-Holland was founded. Their breeding goals were the breeding of "lambs which were most suitable to convert grass to carcass and ewes which would produce strong lambs and raise them well".

The resulting "new Texel" was considered to be predominately a meat sheep, and so the breed standard was for an early-maturing sheep having excellent slaughter characteristics. The breed definitions were as follows:

- Top of the head should be flat and unwooled and the nose dark, preferably black. The head must be covered with fine white hair, when there are spots on the ear or eyelids they should be black.
- Neck and body should be thick set with broad loins, rump and hindquarters. Muscling should be excellent.
- Udder should be covered with white hair and be lined with wool. The scrotum should be covered with fine hair or short wool.
- Legs should be sturdy and strong, relating to the thick set build of the body.
- Tail should be covered with wool and not reach lower than the heel.
- Wool should be dense and of 'crossbred' fineness.

2.3.2 Establishment of the Texel outside the Netherlands

The first Texels were imported into France in 1933 (Visscher and Bekedam, 1984). The French have the oldest foreign Texel Flock book, started in 1935. In the early years the French placed breeding emphasis on producing a small, well muscled animal with a low carcass fat covering. Their modern day breeding objective, however, places more emphasis on prolificacy, growth rate until 4 months of age and carcass quality. As a result the French Texel is quite different from the Dutch Texel, which, having been selected for conformation, has a lower weight for age (Visscher and Bekedam, 1984).

There were large importations of Texels into Germany in the 1960's (Visscher and Bekedam, 1984). The Germans placed similar selection pressure on Texels as the Dutch, although with more emphasis on growth and development and less on physical appearance. Belgium also commenced imports of Texels at a similar time.

The first Texels imported into England were French in origin with importations in 1970, 1971 and 1973 (www.texel.co.uk). The first direct import from the Netherlands occurred in the late 1970's. Today the English Texel population is made up of a mixture of Dutch and French strains, although the exact proportions of each are not known. The Texel sheep in Ireland are derived from imports from the Netherlands in 1964 and 1974 (McEwan et al., 1988). By the early 1980's Texels had also been imported by Italy, Brazil, Peru, Spain, Denmark and Switzerland.

The first Texels imported into the USA were from Finland and Denmark in 1985 (Leymaster & Jenkins, 1993), when 20 pregnant Texel ewes mated to five Texel rams were sampled from one flock in Finland. Four of the five rams were also imported. Five Texel rams were also imported from Denmark, with each ram from a different flock. The importation into the USA was a sample of the same Finnish and Danish Texels that produced embryos for importation into New Zealand in 1985.

The Texel was introduced into New Zealand on the basis of recommendations of the maximum security quarantine advisory committee appointed by the Ministry of Agriculture to review the need for new sheep breeds (Clarke et al., 1988). This review found that New Zealand needed a breed with increased lean-meat production, improved distribution of fat and higher muscle to bone ratios, all traits which the Texel possessed.

Frozen embryos were imported and implanted in Coopworth recipient ewes from 1985-1987, with 40 progeny of Dannish descent and 28 of Finnish descent born (Tervit et al., 1986). These first Texel progeny were mated as hoggets in 1986 and subjected to embryo transfer programmes in subsequent years to increase numbers. A second importation of 125 Texel ewes and ewe lambs was made by New Zealand Dairy Board interests (LambXL) in February 1986 (Allison et al., 1989). Hormonal induction of breeding resulted in more than 80% of the LambXL ewes lambing again in November 1986. During 1987 and 1988 comprehensive embryo transfer programmes for both importations increased animal numbers during a MAF-specified quarantine period of 5 years. The breed was commercially released from quarantine in 1990.

An Australian flock began quarantine in New Zealand in 1988 (www.texel.org.au). In 1993 selectors appointed by the Australian Texel Stud Breeders Association Inc. chose a total of 790 Texel ewes and 50 Texel rams from a base flock of 2220 Texels.

2.3.3 Comparison with Other Breeds

Since their introduction into other countries, a number of breed comparison studies involving the Texel have been carried out. Comparisons between studies can not be made, because the conditions of each study vary, not only in location but also in management practices and traits measured. Not all studies included statistical evaluations of differences. Where this did not occur, alternatives such as standard errors are presented. The country in which the study was carried out is included, as different countries have different strains of the Texel. The following summaries will be limited to traits of direct importance to meat production namely: average daily gain, dressing-out percentage, linear carcass dimensions, *M. longissimus* dimensions, muscularity/carcass shape, muscle distribution, lean-meat- yield, muscle to bone ratio and meat quality. A number of trials also included comparisons of dam breeds, however, these were not considered. Not all breeds were represented in all trials (with the exception of the Texel). The most common comparisons were with Suffolks, but other breeds included Poll Dorset, South Down, Oxford, Dorset Down, Dorset Horn, Charmoise and Charollais.

A list of the abbreviations used throughout this section and the following section, comparing gender groups, are provided in Table 2.1.

Table 2.1 Abbreviations used throughout the review of breed comparisons including the Texel and gender group comparisons

Abbreviation	Definition
Breed	
BL	Border Leicester
C	Coopworth
C1	Composite 1
Cha	Charolais
Che	Cheviot
Chm	Charmoise
Com	Composite
Cp	Coopworth
CT	Conformation Texel (selection line for conformation)
D	Dorset
DD	Dorset Down
DH	Dorset Horn
DL	Damline
EF	East Friesian
FL	Finnish Landrace
G	Galway
GF	Greyface
H	Hampshire
HC	High Conformation Texel (selection line for conformation)
HD	Horned Dorset
LT	Lean Texel (selection line for lean)
M	Mule
Mer	Merino
Nor	Normal Texel (no selection)
O	Oxford
OD	Oxford Down
PD	Poll Dorset
R	Romney
S	Suffolk
SBF	Scottish Blackface
SD	Southdown
T	Texel
(a)	Ewes used by More O'Ferrall and Timon (1977, 1974) - G, Che, SBF, BL GxChe, BLxChe, FLxChe, GxSBF, BLxSBF, FLxSBF
(b)	Ewes used by Kempster et al. (1987) and Croston et al. (1987) - SBF, M
(c)	Ewes used by Wolf et al. (1980) - DL, BLxSBF
Significance Results	
^{abc}	Breeds with different superscripts within a column differ (P<0.05)
^{ns} ,+, *, **, ***	Non significant, differ at the P<0.1, P<0.05, P<0.01 and P<0.001 level respectively
approx se, av sem, LSD	Approximate standard error of the difference, average standard error of the meant, least squares difference
Other Abbreviations	
Crypt	Cryptorchids
CWT	Carcass weight
No specified adj.	No covariates specified in materials and methods
SLGAGE	Age at slaughter
LWT	Live weight
SLGWT	Slaughter weight
IMF	Intermuscular fat
SCF	Subcutaneous fat

Average daily gain (ADG)

A range of ADGs covering differing periods between either birth to weaning or birth to slaughter have been reported (Table 2.2). The time period used, however, does not appear to have had an effect on the results which varied between studies. In some studies Texel sired lambs have growth rates not significantly different from other breeds, however, in the majority of studies the Texel-sired lambs had growth rates significantly lower than most other terminal sire breeds, particularly Dorset Down, Oxford Down and Suffolk rams. The only study in which the Texel-sired lambs grew faster than any of these breeds was that of Flanagan and Hanrahan (1992), where between the age of five weeks and sale at approximately 37kg liveweight, the Texel-sired lambs grew faster than the Suffolk-sired lambs. A likely cause of the between trial differences is the source of the Texel. As discussed by Kempster et al. (1987) the Texels used in their trial had been sourced from French Texel whereas other United Kingdom trials up until then had used Texels of Dutch origin, which had experienced less selection for growth rate than other strains of Texels.

Table 2.2 A summary of selected results from breed comparisons for average daily gain (ADG) (g/d) including the Texel as a sire breed[1]

Authors; Country; Comments	Breeds	ADG (1)	ADG(2)
More O'Farrell and Timon (1977a)	S x (a)	251[a]	195[a]
Ireland	T x (a)	240[bc]	181[b]
ADG(1) birth to weaning (~14 weeks)	DH x (a)	251[a]	192[a]
ADG(2) birth to slaughter (~36-45kg LWT)	H x (a)	243[b]	180[b]
	OD x (a)	249[a]	192[a]
	DD x (a)	238[c]	158[c]
Latif and Owen (1979)	T x (FL x DH)	200 (67)	232 (65)
England	S x (FL x DH)	203 (54)	231(48)
ADG(1) 6 weeks to slaughter(30-36kg) (+/-sd)			
ADG(2) birth to slaughter (30-36kg) (+/-sd)			
Hanrahan (1979a)	S		320[ns]
Ireland	T		322
ADG from 56-112 days			
Hanrahan (1979b)	T x		294[ns]
Ireland	S x		323
ADG from 18-19kg to slaughter (~40kg LWT)			
Latif and Owen (1980)	S x (FLxDH)		283[ns]
England	T x (FLxDH)		268
ADG from weaning to slaughter (~30-36kg LWT)			
Wolf et al. (1980)	DD x (c)		278[a]
Scotland	OD x (c)		302[d]
Adjusted for birthday deviations	S x (c)		296[cd]
ADG from 8-12 weeks (~ 30kg LWT)	T x (c)		262[c]
Cameron and Drury (1985)	T x (h)		255[b]
Scotland	O x (h)		267[d]
Adjusted for birth date	Cha x (h)		258[bc]
ADG from 0-16 weeks	Chm x (h)		244[a]

Authors; Country; Comments	Breeds	ADG (1)	ADG(2)
Kempster et al. (1987)	DH x (b)		82
England	OD x (b)		79
nb late flock used	SD x (b)		80
ADG in CWT from birth to ~180 days	S x (b)		84
	T x (b)		83
Sinnett-Smith and Woolliams (1988)	O		150
Scotland	T		182
ADG from 7-11 weeks	(approx se)		(24)
McEwan et al. (1988)	T	240	323
Ireland	S	240	359
ADG(1) birth to slaughter (~38kg LWT)	(approx se)	(9)	(23)
ADG(2) birth to slaughter (~55kg LWT)			
Flanagan and Hanrahan (1992)	S	264[ns]	261*
Ireland	T	272	277
ADG(1) birth to 5 weeks			
ADG(2) 5 weeks to sale (~37kg LWT)			
Jorgensen et al. (1993)	T		140 (0.008)
Denmark	OD		167 (0.010)
ADG from birth to 4 months			
Leymaster and Jenkins, (1993)	T x C1		219
USA	S x C1		224
ADG from birth to weaning (~51 days)	(av sem)		(8.0)
Cruickshank et al. (1996)	T x	126 (8.1)	113 (8.5)
New Zealand	O x	155(5.7)	126 (7.5)
(se in brackets)	S x	157 (5.7)	142 (6.3)
ADG(1) Rams 23-26kg for 3 months	T x		
ADG(2) Ewes 23-26kg for 3 months	O x		
Hopkins et al. (1996)	PD x Mer		278[b]
Australia	T x Mer		256[b]
*Adjusted for starting live*weight			
ADG from 6 months for 42 days			
Ellis et al. (1997)	Cha x M		249[ns]
England	Cha x S		262
ADG from birth to slaughter (~40kg)	Cha x T		250
Wylie et al. (1997)	T x GF (Ram)		297
Ireland	T x GF (Ewe)		222
ADG from birth to slaughter (~44kg LWT)	S x GF (Ram)		289
	S x GF (Ewe)		226
	(se)		(11)
Hanrahan (1999)	T	310	292
Ireland	S	312	303
ADG(1) birth to 5 weeks	(sed)	4.4	3.2
ADG(2) birth to 14 weeks			
Johnston et al. (1999)	S x GF	286***	177[ns]
Scotland	T x GF	254	170
ADG(1) from birth to weaning			
ADG(2) from weaning to slaughter			
Fogarty et al. (2000)	PD x (BL x Mer)		234[a]
Australia	PD x Mer		203[c]
ADG from birth to weaning (~85 days)	T x (BL x Mer)		224[b]
	T x Mer		199[c]
Scales et al. (2000)	PD x Mer		102
New Zealand	T x Mer		91
ADG over first year	OD x Mer		94
	S x Mer		100
	(LSD)		4

[1] Abbreviations are defined in Table 2.1

Dressing-out percentage (DO%)

There was considerable variation in the DO% values between studies (Table 2.3). This is due to the weight/age at which the lambs were slaughtered varying between the studies, however, it does not appear to have affected the overall rankings of the sire breeds. In general the Texel-sired lambs tended to have DO% not significantly different from, or significantly better than, most other breeds. The only study in which the Texel had a significantly lower DO% was More O'Ferrall and Timon (1977a) where the Dorset Down-sired lambs had a significantly better DO% than the Texel-sired lambs.

Table 2.3 A summary of selected results from breed comparisons for dressing-out percentage (DO%) including the Texel as a sire breed[1]

Author/s; Country; Comments	Breeds	DO%
More O'Ferrall and Timon, (1977a)	S x (a)	44.6[b]
Ireland	T x (a)	44.6[b]
No specified adjustments	DH x (a)	45.1[b]
At ~16.9 kg CWT	H x (a)	44.8[b]
	OD x (a)	43.5[c]
	DD x (a)	45.9[a]
Latif and Owen (1979)	T x (FLx DH)	47.4[ns]
England	S x (FLx DH)	47.5
No specified adjustments		
~16kg CWT		
Latif and Owen, (1980)	Tx (FLxDH) (Ram)	48.8
England	Tx (FLxDH) (Ewe)	49.6
No specified adjustments	Sx (FLxDH) (Ram)	50.1
~17kg CWT	Sx (FLxDH) (Ewe)	51.2
	(sem)	(0.66)
Wolf et al. (1980)	DD x (c)	44.4[a]
Scotland	O x (c)	43.6[c]
Adjusted for slaughter group and age	S x (c)	43.6[c]
~35/40 kg LWT	T x (c)	44.5[a]
Cameron and Drury (1985)	SBF x Chm	42.5[a]
Scotland	SBF x T	41.1[ab]
Adjusted for DOB	SBF x Cha	41.9[b]
~16.8 kg CWT	SBF x O	40.2[ab]
Hanrahan et al. (1986)	T (Ram)	47.4[ns]
Ireland	S (Ram)	47.4
Estimated from LWT and CWT	T (Ewe)	50.6*
	S (Ewe)	46.1
Hanrahan (1988)	T	45.0[ns]
Ireland	S	44.8
No specified adjustments		
~18.2 kg CWT		
McEwan et al. (1988)	T (Ram)	51.5[ns]
Ireland	S (Ram)	51.3
Adjusted for CWT		
~27.8 kg CWT		

Author/s; Country; Comments	Breeds	DO%
Flanagan and Hanrahan (1992)	T	46.5 *
Ireland	S	45.7
No specified adjustments		
~17.4 kg CWT		
Young et al. (1993)	OD x Mer	43.6
New Zealand	S x Mer	44.5
Calc. from mean LWT at slaughter and CWT	PD x Mer	44.5
~19.2 kg CWT (except M ~13.2 kg CWT)	T x Mer	44.0
Hopkins et al. (1996)	T x Mer	50.5^{ns}
Australia	PD x Mer	50.6
No specified adjustments	T x (BL x Mer)	50.1
~28.5 kg CWT	PD x (BL x Mer)	50.1
Ellis et al. (1997)	Cha x M	45.0^{ns}
England	S x M	44.4
Adjusted to estimated SCF of 100g/kg	T x M	45.2
~18.6 kg CWT		
Wylie et al. (1997)	T x GF (Ram)	47.3
Ireland	T x GF (Wether)	48.6
Adjusted for SLGWT	T x GF (Ewe)	48.3
~19.5 kg CWT	S x GF (Ram)	47.0
	S x GF (Wether)	47.6
	S x GF (Ewe)	48.1
	(se)	(2.9)
Hanrahan (1999)	T (High LMI)	46.2
Ireland	T (Low LMI)	46.5
No specified adjustments	S (High LMI)	45.0
~ 19 kg CWT	S (Low LMI)	45.0
Fogarty et al. (2000)	D x (BL x Mer) (Ram)	50.8^{b}
Australia	T x (BL x Mer) (Ram)	51.3^{a}
Adjusted to 24 kg CWT for Ram lambs	D x Mer (Ram)	50.4^{b}
Adjusted to 19 kg CWT for Ewe lambs	T x Mer (Ram)	50.7^{b}
	D x (BL x Mer) (Ewe)	49.9^{a}
	T x (BL x Mer) (Ewe)	49.7^{a}
	D x Mer (Ewe)	49.7^{a}
	T x Mer (Ewe)	49.7^{a}
Hall and Henderson (2000)	T x (CW)	46.9^{a}
England	DD x (CW)	45.8^{a}
No specified adjustments	S x (CW)	45.4^{a}
~16 kg CWT		
Dawson and Carson (2001)	S	43.3***
Northern Ireland	T	44.8
Adjusted to 19 kg CWT	(sem)	(0.16)
~19 kg CWT		

[T] Abbreviations are defined in Table 2.1

M. longissimus dimensions

A number of studies have considered *M. longissimus* dimensions (Table 2.4). Texel sires always rank well for *M. longissimus* area and dimensions, although differences have not always been significant. In some studies, however, the progeny of Texel sires have had smaller dimensions than other terminal-sired lambs. For example in the work

of Kirton et al. (1997) the Texel-sired lambs ranked below the Poll Dorset, and Southdown Fat-sired lambs. In the work of Clarke et al. (1988) the Oxford had a larger EMA, although the difference was not significant. In all comparisons involving Suffolk sires, Texel-sired lambs had larger EMA at the same carcass weight, although in the study of Kempster et al. (1987) the Texel-sired lambs had *M. longissimus* that were not as wide, but were deeper.

Table 2.4 A summary of selected results from breed comparisons for *M. longissimus* characteristics (*M. longissimus* area (EMA) (cm^2); *M. longissimus* width (A); *M. longissimus* depth (B) (mm) including the Texel as a sire breed[1]

Author/s; Country; Comments	Breed	EMA	A	B
More O'Ferrall, (1974)	T x	12.0		
Ireland	DH x	11.6		
No specified adjustments	H x	11.2		
~16.9kg CWT	OD x	11.1		
	DD x	11.9		
Wolf et al. (1980)	DD x (c)	10.9^{ab}		
Scotland	O x (c)	10.5^{bc}		
Adjusted to a constant LWT	S x (c)	10.6^{bc}		
~16kg CWT	T x (c)	11.3^{a}		
Hanrahan et al. (1986)	S	bigger		
Ireland	T			
No specified adjustments				
~14-30kg CWT				
Kempster et al. (1987)	DD x (b)		55.1^{ns}	26.0^{a}
England	HD x (b)		56.2	25.5^{a}
Adjusted for SCF	OD x (b)		58.1	26.3^{a}
~19kg CWT	SD x (b)		55.3	25.8^{a}
	S x (b)		58	26.4^{a}
	T x (b)		57.7	27.4^{b}
Clarke et al. (1988)	O x R and C	15.0	56.7	26.5
New Zealand	T x R and C	14.4	54.5	26.0
Adjusted for CWT	S x R and C	14.0	54.0	26.0
~ weight unknown	(rsd)	(1.3)	(3.0)	(2.0)
McEwan et al. (1988)	T (Ram)	19.4		
Ireland	S (Ram)	17.8		
Adjusted to CWT	(av sem)	(0.7)		
~27.8kg for young	T (Ram) (Mature)	22.2		
~40kg for mature	S (Ram) (Mature)	20.2		
	T (Ewe) (Mature)	18.6		
	S (Ewe) (Mature)	17.4		
	(av sem)	(1.3)		
Ward et al. (1992)	Tx Cp		30.7^{ns}	15.8^{a}
New Zealand	O x Cp		29.1	14.1^{b}
Adj. for CWT				
? CWT				
Leymaster and Jenkins (1993)	T x Cl	16.2		
USA	S x Cl	15.8		
Adjusted to fixed age of 189 days	(sem)	(0.43)		
~28.0kg CWT				

Author/s; Country; Comments	Breed	EMA	A	B
Kirton et al. (1995)	O x R	11.0[ns]		
New Zealand	T x R	11.9		
Adjusted for CWT	(sed)	(0.99)		
~17.0kg CWT				
Hopkins et al. (1996)	T x Mer	16.2		
Australia	PD x Mer	16.6		
Adjusted to 28.5kg CWT	T x (BL x Mer)	17.9		
~28.5kg CW	PD x (BL x Mer)	17.1		
	(sed)	(0.1)		
Wolf and Jones (1996)	HC	20.8***		
Wales	Normal	18.3		
Adjusted to constant SLGWT				
~41.2kg LWT				
Ellis et al. (1997)	Cha x M		58.6[a]	28.1
England	S x M		60.0[b]	27.6
Adjusted to constant fat depth	T x M		61.1[b]	28.8
~18.6kg CWT				
Hopkins et al. (1997)	T x (BL x Mer) (Ram)	15.6[a]		
Australia	D x (BL x Mer) (Ram)	15.7[a]		
Adjusted to 24.8kg CWT for Ram lambs	T x Mer (ram)	15.9[a]		
Adjusted to 17.6kg CWT for Ewe lambs	D x Mer (ram)	15.5[a]		
	T x (BL x Mer) (Ewe)	12.1[a]		
	D x (BL x Mer) (Ewe)	11.8[ab]		
	T x Mer (ewe)	12.1[a]		
	D x Mer (ewe)	11.8[ab]		
Kirton et al. (1997)	T x	12.8	55.1	31.0
New Zealand	PD x	14.4	55.2	35.2
Adjusted to 19.5kg CWT	OD x	12.3	54.2	30.1
	DD x	12.2	53.7	30.8
	SD Fat x	13.4	54.0	32.3
	SD Lean x	12.6	52.0	31.4
	S x	12.5	55.9	30.3
	(approx sed)	(0.46)	(1.4)	(0.8)
Hopkins and Fogarty (1998)	T x (BL x Mer) (Crypt)	15.1[ab]		
Australia	D x (BL x Mer) (Crypt)	15.8[ab]		
Adjusted to 23.7kg CWT for Crypt	T x Mer (Crypt)	15.9[a]		
Adjusted to 17.6kg CWT for Ewe	D x Mer (Crypt)	15.5[ab]		
	T x (BL x Mer) (Ewe)	13.1[a]		
	D x (BL x Mer) (Ewe)	12.7[a]		
	T x Mer (Ewe)	13.2[a]		
	D x Mer (Ewe)	12.6[a]		
Scales et al. (2000)	PD x Mer	16.0	56.0	28.1
New Zealand	T x Mer	16.0	56.0	27.9
Adjusted to 19.5kg CWT	OD x Mer	15.0	56.0	26.9
~19.5kg CWT	S x Mer	15.0	57.0	26.6
	(LSD (P<0.05))	(0.5)	(1.0)	(0.8)
Fogarty et al. (2000)	D x (BL x Mer) (Ram)	15.1[bc]	63.21[b]	29.7[c]
Australia	T x (BL x Mer) (Ram)	15.6[b]	63.6[b]	30.7[a]
Adjusted to 19kg CWT for Ewe lambs	T x Mer (Ram)	16.0[a]	64.7[a]	30.6[ab]
	D x (BL x Mer) (Ewe)	13.2[bc]	60.8[b]	27.0[ab]
	T x (BL x Mer) (Ewe)	13.7[b]	61.3[b]	27.6[a]
	D x Mer (Ewe)	13.3[b]	61.3[ab]	27.0[ab]
	T x Mer (Ewe)	14.0[a]	62.3[a]	28.1[a]

Author/s; Country; Comments	Breed	EMA	A	B
Wolf et al. (2001)	LT		74.2	34.0
Wales	CT		75.5	33.4
Adjusted to constant slaughter age				
~43.2kg LWT				

[1] Abbreviations are defined in Table 2.1

Linear measurements

The linear measurements summarised in Table 2.5 varied widely between studies. For a given carcass weight, Texel-sired lambs have been shown to have significantly shorter carcasses than other terminal-sired-lambs. However, Hanrahan (1976) found no difference in carcass length between the Texel- and Suffolk-sired lambs and Ellis et al. (1997) found the Texel-sired lambs to be longer than the Suffolk-sired lambs. In the majority of studies where leg length has been considered, the Texel-sired lambs tended to have shorter legs than other breeds, although in some comparisons with Suffolk-sired lambs, as in the work of Hanrahan (1976) and Ellis et al. (1997), the difference was not significant. Most other linear traits such as shoulder width, gigot width and circumference have been considered in only one or two studies, with the Texel-sired lambs being not significantly different to other terminally-sired lambs for these traits.

Table 2.5 A summary of selected results from breed comparisons for linear measurements (carcass length (CcLe); leg length (LgLe); gigot width (GWi); chest width (ChWi)) (cm) including the Texel as a sire breed[1]

Author/s; Country; Comments	Breed	CcLe	LgLe	GWi	ChWi
Hanrahan et al. (1986)	S	[ns]	[ns]		
Ireland	T				
No specified adjustments					
~14-3kg CWT					
More O'Ferrall and Timon (1977b)	S x (a)	95.1^{c}	24.9^{a}		
Ireland	T x (a)	94.5^{ab}	25.6^{b}		
No specified adjustments	DH x (a)	95.3^{c}	25.0^{a}		
~16.9kg CWT	H x (a)	94.4^{ab}	24.7^{a}		
	OD x (a)	96.3^{d}	25.8^{b}		
	DD x (a)	94.9^{bc}	24.8^{a}		
Latif and Owen (1980)	T x (FLxDH) (Ram)		21.0^{ns}		
England	T x (FLxDH) (Ewe)		20.6		
No specified adjustments	S x (FLxDH) (Ram)		21.0		
~17kg CWT	S x (FLxDH) (Ewe)		19.9		
Clarke et al. (1988)	O x (R and C)	97.0	43.5		
New Zealand	T x (R and C)	98.0	43.6		
Adjusted for CWT	S x (R and C)	98.0	44.0		
	(rsd)	(1.7)	(1.3)		
Leymaster and Jenkins (1993)	T x (C1)	60.4			
USA	S x (C1)	63.5***			
Adjusted to fixed age of 189 days	(av sem)	(0.6)			
~28.0kg CWT					

Author/s; Country; Comments	Breed	CcLe	LgLe	GWi	ChWi
Wolf and Jones (1996)	CT	52.7			
Wales	Nor	54.9			
Constant SLGWT	(sed)	(5.79)			
~41.2kg LWT					
Ellis et al. (1997)	Cha x M	60.1[a]	26.1[ns]	65.5[ns]	23.6[ns]
England	S x M	53.8[b]	25.8	65.4	24.0
Adjusted to constant fat	T x M	60.4[a]	25.3	65.1	24.2
~18.6kg CWT					
Gigot Circumference					
Scales et al. (2000)	PD x Mer		22.8[a]	19.4[a]	
New Zealand	T x Mer		23.3[b]	19.9[b]	
Adjusted to 19.5kg CWT	OD x Mer		23.4[b]	20.0[b]	
	S x Mer		24.0[d]	19.6[a]	
Wolf et al. (2001)	LT	57.5	21.2	67.6	
Wales	CT	56.4	21.0	68.0	
Adjusted to constant slaughter age	(sed)	(9.6)	(1.62)	(5.48)	
~43.2kg LWT					

[1] Abbreviations are defined in Table 2.1

Carcass conformation

Conformation scores in the studies have been based on either the EUROP conformation score or on point scales where higher points indicate better conformation (Table 2.6). Generally differences between Texel-sired lambs and other terminal-sired-lambs were not significant, however, the Texel-sired lambs in most cases ranked near the top.

Conformation as assessed by muscularity indexes was considered by Jones et al. (2002) who showed Texel purebred lambs to have higher muscularity scores (using the formula developed by Purchas (1991)) than either Suffolk or Charollais purebred lambs. Hopkins et al. (1997) also reported muscularity values, but found no difference between Texel-sired and Poll Dorset-sired lambs.

Table 2.6 A summary of selected results from breed comparisons for conformation assessments (carcass conformation; EUROP conformation (1-5)) including the Texel as a sire breed[1]

Author/s; Country; Comments	Breed	Conf	EUROP Other	Other
Kempster et al. (1981)	DD x (b)	8.4		
England	HD x (b)	8.3		
Adjusted for SCF	OD x (b)	7.9		
~16kg CWT	SD x (b)	9.3		
15 point scale (15 top)	S x (b)	8.4		
	T x (b)	7.8		
Cameron and Drury (1985)	SBF x Chm	9.1		
Scotland	SBF x T	8.1		
Adjusted for DOB	SBF x Cha	9.0		
~16.7kg CWT	SBF x O	8.3		
15 point scale (15 top)				

Author/s; Country; Comments	Breed	Conf	EUROP	Other	Other
Kempster et al. (1987)	DD x (b)	7.6^{bc}			
England	OD x (b)	6.9^{a}			
Adjusted for SCF	SD x (b)	8.3^{c}			
~19kg CWT	S x (b)	7.8^{c}			
15 point scale (15 top)	T x (b)	7.9^{c}			
Hanrahan (1988)	S	2.9*			
Ireland	T	2.7			
No specified adjustments					
~ 18.2kg CWT					
5 point scale (5 top)					
Flanagan and Hanrahan (1992)	S	3.3^{ns}			
Ireland	T	3.3			
No specified adjustments					
~17.4kg CWT					
5 point scale (5 top)					
Leymaster and Jenkins (1993)	T x (C1)	12.4*			
USA	S x (C1)	11.8			
Adjusted to a constant age (189 days)					
~28.0kg CWT					
Conformation 14 point scale (14 top)					
Hopkins et al. (1996)	T x Mer		2.6^{ab}		
Australia	PD x Mer		2.9^{b}		
No specified adjustments	T x (BL x Mer)		2.2^{a}		
~ 28.5kg CWT	PD x (BL x Mer)		2.5^{ab}		
Wolf and Jones (1996)		Leg C			
Wales	CT	Better***			
Constant SLGWT	Nor				
~41.2kg LWT					
Ellis et al. (1997)		*C Score*			
England	Cha x M	2.9^{ns}			
Adjusted to SCF 100g/kg	S x M	2.8			
~18.6kg CWT	T x M	2.9			
5 point scale (5 top)					
Hopkins et al. (1997)	T x (BL x Mer) (Ram)		2.5^{a}		
Australia	D x (BL x Mer) (Ram)		2.6^{ab}		
Adjusted to 24.2kg CWT for R	T x Mer (Ram)		2.8^{ab}		
Adjusted to 17.1kg CWT for E	D x Mer (Ram)		3.0^{abc}		
	T x (BL x Mer) (Ewe)		2.3^{a}		
	D x (BL x Mer) (Ewe)		2.9^{b}		
	T x Mer (Ewe)		2.8^{b}		
	D x Mer (Ewe)		3.0^{bc}		
Hopkins and Fogarty (1998)	T x (BL x Mer) (Crypt)		2.6^{a}		
Australia	D x (BL x Mer) (Crypt)		2.8^{a}		
Adjusted to 23.7kg CWT for R	T x Mer (Crypt)		2.8^{a}		
Adjusted to 17.6kg CWT for E	D x Mer (Crypt)		2.2^{bd}		
	T x (BL x Mer) (Ewe)		2.5^{a}		
	D x (BL x Mer) (Ewe)		3.0^{bc}		
	T x Mer (Ewe)		2.8^{ab}		
	D x Mer (Ewe)		3.2^{ce}		
Hanrahan (1999)				*E+U (%)*	*R (%)*
Ireland	S (Low)			54	44
No specified adjustments	S (High)			52	43
~19kg CWT	T (Low)			60	35
	T (High)			52	43

Author/s; Country; Comments	Breed	Conf	EUROP	Other	Other
Fogarty et al. (2000)	D x (BL x Mer) (Ram)		2.9[d]		
Australia	T x (BL x Mer) (Ram)		2.5[e]		
Adjusted to 24kg CWT for Ram lambs	D x Mer (Ram)		3.1[c]		
Adjusted to 19kg CWT for Ewe lambs	T x Mer (Ram)		2.8[d]		
	D x (BL x Mer) (Ewe)		3.0[d]		
	T x (BL x Mer) (Ewe)		2.7[e]		
	D x Mer (Ewe)		3.2[c]		
	T x Mer (Ewe)		2.9[d]		
Scales et al. (2000)				*E (%)*	*R (%)*
New Zealand	PD x Mer			29.0	26.5
Adjusted to 19.5kg CWT	T x Mer			65.7	31.4
	OD x Mer			60.0	40.0
	S x Mer			31.8	56.0
Dawson and Carson (2001)	S	2.89***			
Northern Ireland	T	3.14			
Adjusted to 19kg CWT					
5 point scale (5 top)					
Wolf et al. (2001)	LT	12.5			
Wales	CT	13.3**			
Adjusted to constant slaughter age					
~43.2kg LWT					
15 point scale (15 top)					

[1] Abbreviations are defined in Table 2.1

Cut distribution

Measurements of cut distribution have varied between studies, and only selected cuts are presented in Table 2.7 to give an indication of the distribution. The Texel, appears to have a lower proportion of total weight in the fore-quarter and loin, with higher proportions in the leg. In the studies of Kempster et al. (1987) and Croston et al. (1987) the Texel-sired lambs appear to have a lower percentage of weight in the high-priced-cuts. However, the work of Cruickshank et al. (1996) found the Texel to have a higher percentage of weight in the high-priced-cuts, and Latif and Owen (1980) found no difference between the breeds.

Table 2.7 A summary of selected results from breed comparisons for cut distribution including the Texel as a sire breed.[1]

Author/s; Country; Comments	Breed	Results				
More O'Ferrall & Timon (1977b)		Hind	Fore			
Ireland	S x (a)	47.3[ab]	36.8[b]			
No specified adjustments	T x (a)	47.2[b]	37.1[a]			
~36-49kg LWT	DH x (a)	47.9[ab]	36.7[ab]			
% carcass	H x (a)	47.1[b]	36.3[b]			
	OD x (a)	47.3[ab]	36.8[ab]			
	DD (a)	47.6[ab]	35.4[c]			
Latif and Owen, (1980)		leg	chump	loin	Shlder	HPC
England	T x (FLxDH) (Ram)	2.1**	0.7[ns]	1.0[ns]	1.7*	3.8[ns]
No specified adjustments	T x (FLxDH) (Ewe)	1.8	0.7	0.9	1.4	3.3
~17kg CWT	S x (FLxDH) (Ram)	2.0	0.7	1.0	1.7	3.9
Weight of cut (kg)	S x (FLxDH) (Ewe)	1.7	0.6	0.9	1.5	3.5

Author/s; Country; Comments	Breed	Results				
Cameron and Drury (1985)		Leg	Chump	Loin	Shlder	
Scotland	SBF x Chm	24.7^{b}	8.3^{ns}	10.3^{a}	21.2^{a}	
Adjusted for Birthday Dev.	SBF x T	26.3^{c}	8.2	9.6^{b}	22.0^{b}	
~16.8kg	SBF x Cha	25.5^{ab}	8.2	10.2^{a}	21.7^{ab}	
%	SBF x O	25.1^{ab}	8.3	10.3^{ab}	21.6^{abc}	
Croston et al. (1987)		Leg	Chump	Loin	Shlder	HPC
Wales	DD x (b)	29.7^{cd}	8.3^{ns}	10.7^{abc}	20.3^{ab}	55.4^{abc}
Adjusted to constant fat	HD x (b)	29.4^{abcd}	8.4	11.3^{c}	20.1^{a}	56.0^{c}
~20.9kg CWT	OD x (b)	29.2^{abc}	8.3	10.9^{abc}	20.6^{bc}	55.1^{ab}
(g/kg)	SD x (b)	29.3^{abcd}	8.3	11.2^{bc}	20.2^{ab}	55.6^{bc}
	S x (b)	29.4^{abcd}	8.3	11.1^{abc}	20.6^{bc}	55.3^{abc}
	T x (b)	29.4^{abcd}	8.4	10.6^{ab}	20.9^{c}	54.7^{b}
Clarke and Kirton (1990)		Leg	Shlder	Rack	Loin	Other
New Zealand	T x	3.0	2.3	0.8	0.8	2.0
Adjusted to side weight *of 9kg*	O x	2.9	2.2	0.9	0.8	2.1
~18kg CWT	S x	2.9	2.3	0.9	0.8	1.9
Weight of cut (kg)						
Cruickshank et al. (1996)		HPC				
New Zealand	T x	40.2^{a}				
Adjusted to 19.2kg CWT	O x	40.0^{a}				
% CWT	S x	39.5^{b}				
Hopkins and Fogarty (1998)		Chump	Round	Loin		
Australia	T x (BL x Mer) (Crypt)	6.0^{a}	3.8^{ab}	5.0^{a}		
Adjusted to GR of 13.5mm (cyrpt)	D x (BL x Mer) (Crypt)	5.9^{a}	3.8^{bc}	5.3^{b}		
~23.7kg CWT for crypt	T x Mer (Crypt)	6.0^{a}	3.9^{a}	4.9^{a}		
Adjusted to GR of 10.3mm (ewe)	D x Mer (Crypt)	5.8^{ab}	3.8^{bc}	5.3^{b}		
~17.5kg CWT for ewe	T x (BL x Mer) (Ewe)	6.4^{ab}	4.2^{a}	4.9^{a}		
%	D x (BL x Mer) (Ewe)	6.2^{bc}	4^{bc}	5.3^{b}		
	T x Mer (Ewe)	6.6^{a}	4.2^{a}	5.0^{a}		
	D x M (Ewe)	6.3^{bc}	4.1^{abc}	5.2^{b}	2.7^{ab}	5.3^{a}
Scales et al. (2000)		Shlder	Leg	Loin	Rack	
New Zealand	PD x Mer	6.0	6.1	1.8	1.9	
Adjusted to 19.5kg CWT	T x Mer	6.1	5.9	1.8	1.8	
Weight (kg)	OD x Mer	6.1	6.0	1.8	1.9	
	S x Mer	6.0	6.0	1.9	1.9	
	LSD (P<0.05)	(0.14)	(0.11)	(0.11)	(0.11)	

[1] Abbreviations are defined in Table 2.1

Lean-meat yield

Measurements of lean-meat-yield differed between the studies summarised in Table 2.8. The results of the studies, however measured, have all shown similar results with Texel-sired lambs having a higher lean-meat yield than other terminal-sired-lambs. The only exception was Kirton et al. (1997) where Poll Dorset-sired lambs had a lean-meat-yield not significantly different to that of Texel-sired lambs.

Table 2.8 A summary of selected results from breed comparisons for lean-meat yield (% lean (%lean); saleable meat yield (SMY); % lean in loin (%LLoin); % lean in leg (%LLeg) including the Texel as a sire breed.[1]

Author/s; Country; Comments	Breed	%	SMY	%LLoin	%LLeg
More O'Ferrall and Timon (1977b)	S	55.9[bc]			
Ireland	T	59.9[a]			
No specified adjustments	DH	57.4[b]			
~36-49kg LWT	H	55.7[bc]			
	OD	56.1[bc]			
	DD	54.7[c]			
Latif and Owen (1979)	T x (FLx DH)	63.6**			
England	S x (FLx DH)	59.6			
No specified adjustments					
~16kg CWT					
Latif and Owen, (1980)	T x (FLxDH) (Ram)	61.7			
England	T x (FLxDH) (Ewe)	58.8			
No specified adjustments	Sx (FLxDH) (Ram)	57.9			
~17kg CWT	Sx (FLxDH) (Ewe)	56.8			
Clarke and Kirton (1990)	T x			60.6	
New Zealand	O x			57.2	
Adjusted to 800g Loin	S x			57.2	
~ 18kg CWT					
Cruickshank et al. (1996)	T x		64.7[a]		
New Zealand	O x		62.9[b]		
Adjusted to 19.2kg CWT	S x		63.4[b]		
Kirton et al. (1995)	T x (Ram)				67.4
New Zealand	O x (Ram)				66.2
Adjusted to same CWT	(sed)				(0.99)
~17.0kg CWT					
Kirton et al. (1997)	T x				66.5
New Zealand	PD x				66.3
Adjusted to 19.5kg CWT	OD x				65.5
	DD x				64.6
	SD Fat x				63.0
	SD Lean x				63.5
	S x				63.7
	(approx sed)				(1.3)
Wolf et al. (1980)	DD x (c)			54.7[a]	
Scotland	O x (c)			56.3[bc]	
Adjusted to constant LWT	S x (c)			56.3[bc]	
~ 16kg CWT	T x (c)			60.5[d]	
Kempster et al. (1987)	DD x (b)	55.7[a]			
England	HD x (b)	56.2[ab]			
No specified adjustments	OD x (b)	56.0[ab]			
~ 19kg CWT	SD x (b)	55.9[ab]			
	S x (b)	56.7[ab]			
	T x (b)	59.0[c]			
Hopkins and Fogarty (1998)	T x (BL x Mer) (Ram)		69.2[b]		
Australia	D x (BL x Mer) (Ram)		68.9[b]		
Adjusted to 23.7kg CWT for Ram lambs	T x Mer (Ram)		70.5[a]		
Adjusted to 18kg CWT for Ewe lambs	D x Mer (Ram)		69.6[ab]		
	T x (BL x Mer) (Ewe)		71.1[b]		
	D x (BL x Mer) (Ewe)		70.0[d]		
	T x Mer (Ewe)		72.2[a]		
	D x Mer (Ewe)		70.8[bd]		

Author/s; Country; Comments	Breed	%	SMY	%LLoin	%LLeg
Wolf and Jones (1996)	CT	66.2[ns]			
Wales	Nor	66.6			
Adjusted to constant SLGWT					
~41.2kg LWT					
Croston et al. (1987)	DD x (b)	56.0[a]			
Wales	HD x (b)	56.4[a]			
Adjusted to constant fat -125g/kg SF depth	OD x (b)	55.8[a]			
~ 20.9kg CWT	SD x (b)	56.6[a]			
	S x (b)	56.2[a]			
	T x (b)	57.9[b]			
Cameron and Drury (1985)	SBF x Chm	52.6[a]			
Scotland	SBF x T	58.8[b]			
Adjusted for DOB as slaughter	SBF x Cha	55.4[c]			
~16.8kg CWT	SBF x O	53.8[ac]			
Wolf et al. (2001)					
Wales					
Adjusted to constant SLGAGE	LT	64.3**			
~ 21.5kg CWT	CT	56.8			
	(sed)	(5.82)			

[1] Abbreviations are defined in Table 2.1

Fat traits

Although the studies in Table 2.9 have used different slaughter ages and slaughter weights, this does not seem to have had much effect on the between breed differences. Most studies have shown Texel-sired lambs to have the lowest percentage fat in the carcass of any of the terminal-breed-sired lambs, although Croston et al. (1987) did not find significant effects on fat percentage. For GR, the Texel sits mid-range in breed comparisons, and is not significantly different to other breeds. For other methods of fat assessment, no clear conclusions can be drawn, although they are generally consistent with the measures discussed above.

Texel-sired lambs have been shown to have a greater proportion of internal fat relative to Suffolks (Leymaster and Jenkins 1993); and Charolais (Ellis et al. 1997) (data not presented Table 2.9). Their ratio of subcutaneous fat to intermuscular fat is also higher than that of the Suffolk (Wolf et al. (1980); Leymaster and Jenkins (1993); and Ellis et al. (1997)) (data not presented Table 2.9).

Table 2.9 A summary of selected results from breed comparisons for fat traits (percentage fat in carcass (%Fat); fat depth over 12th rib, 110mm from midline (GR; mm); fat depth over *M. longissimus* (C; mm); other (specified in comments) including the Texel as a sire breed.[1]

Author/s; Country; Comments	Breed	FAT %	GR[1]	C[1]	Other
More O'Farrell, (1974)	S x	27.4			
Ireland	T x	22.7			
No specified adjustments	DH x	25.8			
~16.9kg CWT	H x	27.6			
	OD x	26.6			
	DD x	29.8			
More O'Farrell and Timon (1977b)	S x (a)	25.8[b]			
Ireland	T x (a)	22.7[c]			
No specified adjustments	DH x (a)	25.8[b]			
~ 36-45kg LWT	H x (a)	27.6[b]			
	OD x (a)	26.6[b]			
	DD (a)	29.8[a]			
Latif and Owen (1979)	T x (FLx DH)	16.0 (2.61)**			
England	S x (FLx DH)	22.3 (2.16)			
No specified adjustments					
(SD in brackets)					
~16kg CWT					
Latif and Owen, (1980)	T x (FLxDH) (Ram)	21.5**			
England	T x (FLxDH) (Ewe)	24.5			
~17kg CWT	Sx (FLxDH) (Ram)	25.7			
	Sx (FLxDH) (Ewe)	27.9			
	(sem)	(1.18)			
Wolf et al. (1980)	DD x (c)	27.9[a]			1.1[a]
Scotland	O x (c)	24.6[cd]			1.1[ab]
Adj. to cnst LWT	S x (c)	25.0[bd]			1.0[c]
~ 16kg CWT	T x (c)	21.5[e]			1.1[abc]
Other - SCF:IMF					
Cameron and Drury (1985)	SBF x Chm	31.1[a]			
Scotland	SBF x T	24.1[b]			
Adj. for DOB	SBF x Cha	28.0[cd]			
~ 16.8kg CWT	SBF x O	28.5[ac]			
Hanrahan et al. (1986)	T (Ram)				7.7
Ireland	T (Ewe)				16.1
Adj. for CWT	S (Ram)				11.9
~40kg CWT (adult)	S (Ewe)				18.4
Other – SCF(g)	(se)				(1.28)
Croston et al. (1987)	DD x (b)	26.1[b]			1.04[a]
Wales	HD x (b)	26.0[b]			1.02[a]
Adj. to cnst fat - 125g/kg SFd	OD x (b)	26.0[b]			1.03[a]
~20.9kg CWT	SD x (b)	26.2[b]			1.01[a]
Other - SCF:IMF	S x (b)	26.1[b]			1.04[a]
	T x (b)	25.4[ab]			1.09[b]
Kempster et al. (1987)	DD x (b)	26.7[cd]			
England	HD x (b)	26.3[bcd]			
Adj. for weight *of SCF*	OD x (b)	25.8[bc]			
	SD x (b)	27.3[d]			
	S x (b)	25.2[b]			
	T x (b)	24.0[a]			
Clarke et al. (1988)	O x (R and C)		5.3	1.8	
New Zealand	T x (R and C)		6.8	2.5	
~ weight unknown	S x (R and C)		7.3	2.9	
No specified adjustments	(rsd)		(2.1)	(0.3)	

Author/s; Country; Comments	Breed	FAT %	GR[1]	C[1]	Other
McEwan et al. (1988)	T (Ram)		4.4	1.2	
Ireland	S (Ram)		7	2.3	
~ 18.8kg CWT for young	(Av sem)		(1.4)	(0.5)	
~ 40kg CWT for mature animals	T (Ram) (Mature)		8.7	22.2	
	S (Ram) (Mature)		14.4	20.2	
	(Av sem)		(1.7)	(1.3)	
	T (Ewe) (Mature)		18.9	18.6	
	S (Ewe) (Mature)		19.4	17.4	
	(Av sem)		(1.7)	(1.3)	
Clarke and Kirton (1990)	T x				184.8
New Zealand	O x				203.3
Adj. to 800g loin	S x				215.6
~ 18kg CWT					
Other - loin fat (g)					
Ward et al. (1992)	T x Cp		8.6^{ns}	2.4^{ns}	
New Zealand	O x Cp		7.0	1.7	
Adj. to constant CWT					
? carcass weight					
Leymaster and Jenkins (1993)	T x Cl	30.6+	7.1^{ns}		
USA	S x Cl	32.5	6.6		
Adj. to a fixed age of 189 days	(Av SEM)	(0.65)	(0.03)		
~28.0kg CWT					
Young et al. (1993)	OD x Mer		11.5^{b}		
New Zealand	S x Mer		12.6^{bc}		
No specified adjustments	PD x Mer		12.5^{bc}		
~ 19.24kg CWT	T x Mer		13.1^{bc}		
Kirton et al. (1995)	O x R	16.6^{ns}			
New Zealand	T x R	15.9			
Adj. to a constant CWT	(sed)	(1.17)			
~17.6kg CWT					
% fat in leg					
Hopkins et al. (1996)	T x Mer		18.1^{a}	3.9^{a}	
Australia	PD x Mer		17.1^{a}	4.0^{a}	
Adj. to 28.5kg CWT	T x (BL x Mer)		15.6^{b}	2.4^{b}	
	PD x (BL x Mer)		15.8^{b}	2.7^{b}	
Wolf and Jones (1996)	CT	18.7^{ns}			
Wales	Nor	17.5			
Adj. to a constant SLGWT					
~41.2kg LWT					
Ellis et al. (1997)	Cha x M		3.2^{ns}	3.0^{ns}	
Adj. to cnst fat	S x M		3.1	2.8	
~18.6kg CWT	T x M		3.2	3.0	
Hopkins et al. (1997)	T x (BL x Mer) (Ram)		15.4^{ns}		18.0^{ns}
Australia	D x (BL x Mer) (Ram)		15.7		19.9
Adj. to 24.2kg CWT for ram lambs	T x Mer (Ram)		14.9		18.0
Adj. to 17.1kg CWT for ewe lambs	D x Mer (Ram)		13.9		18.7
Other - % fat of the leg	T x (BL x Mer) (Ewe)		10.6^{a}		16.8^{a}
	D x (BL x Mer) (Ewe)		11.7^{a}		17.9^{ab}
	T x Mer (Ewe)		10.9^{a}		16.5^{a}
	D x Mer (Ewe)		10.8^{a}		16.6^{a}

Author/s; Country; Comments	Breed	FAT %	GR[1]	C[1]	Other
Kirton et al. (1997)	T x		8.8	2.7	16.4
New Zealand	PD x		8.7	2.4	16.7
Adj. to 19.5kg CWT	OD x		9.2	2.9	16.7
~ 19.2kg CWT	DD x		10.7	2.3	18.2
Other - %fat leg	SD Fat x		12.7	4.3	20.8
	SD Lean x		11.3	3.0	20.2
	S x		9.2	2.9	18.7
	(approx sed)		(1.17)	(0.46)	(1.2)
Wylie et al. (1997)	T x GF (Ram)		6.0		
Ireland	T x GF (Wether)		6.5		
Adj. for cnst SLGWT	T x GF (Ewe)		7.9		
~ 19.5kg CWT	S x GF (Ram)		6.5		
	S x GF (Wether)		8.9		
	S x GF (Ewe)		8.5		
	(se no=30)		(0.305)		
Hop. and Fogarty (1998)	T x (BL x Mer) (Crypt)		14.4^{b}		
Australia	D x (BL x Mer) (Crypt)		14.2^{ab}		
Adj. to 23.7kg CWT for ram lambs	T x Mer (Crypt)		13.7^{ab}		
Adj. to 17.6kg CWT for ewe lambs	D x Mer (Crypt)		13^{a}		
	T x (BL x Mer) (Ewe)		10.5^{a}		
	D x (BL x Mer) (Ewe)		11.1^{a}		
	T x Mer (Ewe)		10^{ab}		
	D x Mer (Ewe)		10.5^{a}		
Nicoll et al. (1998)	S x (LC)		13.9^{a}		
New Zealand	T x (LC)		13.1^{a}		
Adj. for CWT					
~20.6kg CWT					
Fogarty et al. (2000)	D x (BL x Mer) (Ram)		13.1^{a}	3.1^{bc}	
Australia	T x (BL x Mer) (Ram)		13.1^{a}	3.4^{b}	
Adj. to 24kg CWT for ram lambs	D x Mer (Ram)		12.2^{b}	2.3^{e}	
Adj. to 19kg CWT for ewe lambs	T x Mer (Ram)		12.1^{b}	2.8^{cd}	
	D x (BL x Mer) (Ewe)		12.0^{a}	9.3^{a}	
	T x (BL x Mer) (Ewe)		11.4^{ab}	9.4^{a}	
	D x Mer (Ewe)		11.0^{bc}	8.3^{b}	
	T x Mer (Ewe)		10.7^{c}	8.3^{b}	
Scales et al. (2000)	PD x Mer		12.0^{bc}		
New Zealand	T x Mer		13.0^{b}		
Adj. to a 19.5 CWT	OD x Mer		11.0^{b}		
	S x Mer		12.0^{bc}		
Dawson and Carson (2001)	S			2.5^{ns}	
Northern Ireland	T			2.4	
Adj to 19kg CWT					
Safari et al. (2001)	T x (BL x Mer)		16.9 (3.0)		
Australia	D x (BL x Mer)		15.6 (2.9)		
No specified adjustments	T x Mer		13.9 (1.3)		
~23.8kg CWT (sd)	D x Mer		13.9 (1.3)		
Wolf et al. (2001)					
Wales					
Adj. to a cnst SLGAGE	LT	20.4*		5.8	1.5^{ns}
~21.5kg CWT	CT	18.9		5.6	1.1
Other - SCF:IMF	(sed)	(0.619)		(0.42)	(0.061)

[1] Abbreviations are defined in Table 2.1

Muscle to bone ratio (MTB)

Texel-sired lambs rank near the top relative to other terminal-sired-lambs for muscle to bone ratio (Table 2.10), although differences were not always statistically significant.

Table 2.10 A summary of selected results from breed comparisons for muscle to bone ratio (MTB) including the Texel as a sire breed.[1]

Authors; Country; Comments	Breed	MTB
More O'Ferrall, (1974)	S x	3.4
Ireland	T x	3.6
No specified adjustments	DH x	3.5
~ 17kg CWT	H x	3.4
	OD x	3.3
	DD x	3.6
More O'Ferrall and Timon, (1977b)	S x (a)	3.4^{bcd}
Ireland	T x (a)	3.6^{a}
No specified adjustments	DH x (a)	3.5^{abcd}
~ 36-40kg LWT	H x (a)	3.4^{bd}
	OD x (a)	3.3^{de}
	DD (a)	3.6^{a}
Croston et al. (1987)	BL x (b)	3.5^{a}
Wales	DD x (b)	3.5^{a}
Adjusted to constant fat - 125g/kg SFd	HD x (b)	3.6^{a}
~20.9kg CWT	OD x (b)	3.4^{a}
	SD x (b)	3.7^{b}
	S x (b)	3.6^{a}
	T x (b)	3.9^{c}
Latif and Owen (1979)	T x (FLx DH)	3.1
England	S x (FLx DH)	3.3
No specified adjustments		
~16kg CWT		
Wolf et al. (1980)	DD x (c)	3.5^{a}
Scotland	O x (c)	3.2^{b}
Adjusted to constant LWT	S x (c)	3.3^{b}
~ 16kg CWT	T x (c)	3.7^{c}
Ward et al. (1992)	T x Cp	8.1^{a}
New Zealand	O x Cp	6.1^{b}
Adj. for carcass weight		
? CWT		
Kirton et al. (1995)	T x (R)	4.6 +
New Zealand	O x (R)	4.3
Adjusted to the same CWT	(sed)	(0.16)
~ 17.0kg CWT		
Hopkins et al. (1997)	T x (BL x Mer) (Ram)	4.1^{ab}
Australia	D x (BL x Mer) (Ram)	3.9^{b}
hind leg	T x Mer (Ram)	4.1^{a}
Adjusted to 24.2kg for Ram lambs	D x Mer (Ram)	3.9^{ab}
Adjusted to 17.1kg for Ewe lambs	T x (BL x Mer) (Ewe)	4.2^{a}
	D x (BL x Mer) (Ewe)	4.2^{ab}
	T x Mer (Ewe)	4.4^{a}
	D x Mer (Ewe)	4.1^{ab}

Authors; Country; Comments	Breed	MTB
Wolf et al. (2001)		
Wales		
Adjusted to constant SLGAGE	LT	4.7*
~21.5kg CWT	CT	4.9
Adjusted to constant SLGWT	(sed)	(0.083)
~21.5kg CWT	LT	4.7*
	CT	4.9
	(sed)	(0.083)

[1] Abbreviations are defined in Table 2.1

Meat quality traits

Results of a number of studies that have examined the effects of breed on meat quality are summarised in Table 2.11 and Table 2.12. Within Table 2.11 the time at which the measurements were taken varied, and thus between studies the range of values often differs. The majority of these studies have found no differences between Texel-sired lambs and other terminal sired breeds for meat quality measurements. Occasionally differences, which are more likely due to attributes of the other breeds involved rather than positive attributes of the Texel-sired lambs, were detected. Likewise, no differences in sensory quality have been detected between the Texel-sired lambs and other terminal-sired lambs.

Table 2.11 A summary of selected results from breed comparisons for meat quality traits (ultimate pH (pHult); colour L* (L*); colour a (a*); colour b (b*); Warner Bratzler assessed tenderness (WB; kg); cooking loss percent (CL%) including the Texel as a sire breed.[1]

Authors; Country; Comments	Breed	pHult	L*	a*	b*	WB	CL(%)
Dransfield et al. (1990)	T x GF	5.8(0.2)	34.9(2)				
England	DD x GF	5.8 (0.2)	35.1(1.9)				
(sd)	S x GF	5.7 (0.1)	34.5(1.4)				
No specified adjustments	O x GF	5.8 (0.1)	35.5(2.0)				
~11-25kg CWT	SD x GF	5.9(0.1)	32.3(1.4)				
Measured on M. longissimus							
Hopkins and Fogarty (1988)	T x (BL x Mer) (Crypt)	5.6^{ab}	38.5^{a}	17.8^{a}	6.2^{a}	2.3^{a}	32.8^{ab}
Australia	PD x (BL x Mer) (Crypt)	5.6^{a}	38.7^{a}	17.7^{a}	7.0^{a}	2.6^{a}	33.1^{ab}
Meat temp. covariate for clr	T x Mer (Crypt)	5.6^{ab}	38.6^{a}	18.1^{a}	6.8^{a}	2.4^{a}	33.8^{a}
~24.7kg CWT for Ram lambs	PD x Mer (Crypt)	5.6^{ab}	38.8^{a}	18.1^{a}	6.6^{a}	3.3^{a}	33.5^{a}
~18.7kg CWT for ewe lambs3	T x (BL x Mer) (Ewe)	5.5^{ab}	38.0^{ab}	20.8^{a}	7.3^{a}	2.5^{a}	33.7^{a}
Measured on M. longissimus	PD x (BL x Mer) (Ewe)	5.5^{b}	37.3^{a}	20.8^{a}	7.3^{ab}	2.5^{a}	32.6^{a}
	T x Mer (Ewe)	5.6^{a}	38.0^{ab}	19.8^{a}	6.5^{ab}	2.6^{a}	33.3^{a}
Young et al. (1993)	OD x M	6.0^{b}					
New Zealand	S x Mer	6.1^{b}					
No specified adjustments	PD x Mer	5.9^{b}					
~ 19.2kg CWT	T x Mer	6.0^{b}					
except M ~13.2kg CW	(LSD at P=0.05)	(0.16)					
Measured on M. longissimus							

Authors; Country; Comments	Breed	pHult	L*	a*	b*	WB	CL(%)
Hopkins (1996)	T x Mer		38.1^{ns}	17.6^{ns}	10.3^{ab}		
Australia	PD x Mer		37.7	17.3	9.6^{a}		
Adjusted to 28.5kg CWT	T x (BL x Mer)		37.7	18.6	10.7^{b}		
Measured on tail	PD x (BL x Mer)		37.3	17.7	11.1^{b}		
Fogarty et al. (2000)	D x (BL x Mer) (Ram)	5.6^{d}	37.5^{a}	17.9^{b}	7.4^{a}		
Australia	T x (BL x Mer) (Ram)	5.6^{bc}	37.9^{a}	17.9^{b}	7.4^{a}		
Adjusted to 21.7kg CWT	D x Mer (Ram)	5.6^{cd}	37.7^{a}	18.5^{ab}	7.6^{a}		
and 215 days slaughter	T x Mer (Ram)	5.6^{cd}	38.0^{a}	18.2^{ab}	7.4^{a}		
Safari et al. (2001)	T x (BL x Mer)	5.6^{a}				2.0^{a}	32.7^{ab}
Australia	D x (BL x Mer)	5.6^{a}				2.6^{a}	33.8^{a}
No specified adjustments	T x Mer	5.6^{a}				2.8^{a}	33.1^{a}
~23.8kg CWT	D x Mer	5.6^{a}				2.5^{a}	33.6^{a}
Measured on M. longissimus							

[1] Abbreviations are defined in Table 2.1

Table 2.12 A summary of selected results from breed comparisons for meat quality as assessed by sensory evaluations (Tenderness (Ten); Juiciness (Jui); Flavour (Fla) and overall) including the Texel as a sire breed.[1]

Authors; Country; Comments	Breed	Ten	Jui	Fla	Overall
Ellis et al. (1997)	Ch x M	2.8	3.7	3.7	2.3
England	S x M	3.1	3.8	3.6	2.5
No specified adjustments	T x M	3.1	3.7	3.7	2.5
~18.6kg CWT	(av see)	(0.118)	(0.089)	(0.042)	(0.091)
8 point scale (1=high)					
Safari et al. (2001)	T x (BL x Mer)	64.6^{ns}	51.2^{ns}	59.2^{ns}	53.4^{ns}
Australia	D x (BL x Mer)	55.7	21.7	59.2	54.2
No specified adjustments	T x Mer	56.2	49.3	59.0	52.8
~23.8kg CWT	D x Mer	57.3	50.6	56.8	52.7
100 point scale (100= high)					

[1] Abbreviations are defined in Table 2.1

2.4 Gender of Choice for Prime Lamb Production

Producers currently have only limited ability to manipulate the sex of lambs born. Although this will become a possibility with sexed semen, it is likely to remain too expensive to use in sheep production systems. Current options for manipulation post-birth include castration of entire ram lambs (creating wethers) by removing the testes, and hence the production of testosterone or inducing cryptorchidism by pushing the testes into or against the body cavity, rendering the animal infertile, but still capable of testosterone production.

Each option (entire, wethers or cryptorchids) offers benefits and disadvantages to producers, and which option a producer chooses is often influenced by their production system. The type of carcass produced for each option varies, and has implications for the value of the carcass. With the move towards yield-based payment and likely eventual inclusion of "quality" measures, choices relating to sex of lamb produced are

likely to become increasingly important. The purpose here is to review studies in which gender groups (those differing in sex or castration status) have been compared for composition and meat quality traits that are of importance to the industry.

2.4.1 Possible Reasons for Differences Between Gender Groups

Differences in the physical attributes of the sexes are related to hormone production. The major sex hormones for ewes are oestrogens, whilst for rams it is testosterone (which belongs to a group of male hormones referred to androgens). Associated with these steroid hormones (oestrogen and testosterone) are generalized effects on growth and development. Two possible modes of action of these hormones were proposed by Heitzman (1981). The first is a direct effect on protein synthesis and/or degradation, mediated by direct entry of hormones into the muscle cells, and the second is an indirect effect via other hormones of the hypothalamus, pituitary, gonads, pancreas or the thyroid, which then exert an anabolic effect on muscle. From this information, however, it is unclear whether or not the same growth would be expected in rams and ewes, given that both hormones do influence muscle growth. However, it is clear that the two sexes have different growth paths (see below), so there are further differences.

The composition of ewes and rams differs at maturity (Thompson (1983) in Butterfield (1988)). This is an important consideration as previous work had suggested that differences in composition of ewes and rams at any weight could be largely attributed to differences in mature liveweight (McClelland et al., 1976).

2.4.2 Comparison of the Composition Characteristics and Meat Quality Between Gender Groups of Lambs

The papers summarised here are a selection that specifically set out to compare gender groups, and papers that were collected for Section 2.3 that included gender group comparisons. Comparisons between all gender groups (ewe, ram, wether and cryptorchid) are included.

Average daily gain (ADG)

The purpose of this review was to look at carcass characteristics, and thus many of the papers reviewed did not include details of growth rate. However, all that did showed ram lambs (and cryptorchids) to have higher growth rates than either wether or ewe

lambs (Corbett et al., 1973; Mahgoub & Lodge, 1998; Wellington et al., 2003). Differences between wether and ewe lambs were not as clear, with Wellington et al. (2003) showing no difference between the two gender groups, whilst Mahgoub and Lodge (1998) and Corbett et al. (1973) showed wether lambs to grow faster.

Dressing-out percentage (DO%)

Ram lambs generally have a lower DO% than either ewe or wether lambs (Table 2.13), although the results of Everitt and Jury (1966a) showed there to be no significant differences between the gender groups. No significant differences have been reported between ewe and wether lambs for DO%. In the one study that has included cryptorchids (Corbett et al., 1973), a similar pattern to rams was observed. This result is in part attributable to both the ram lambs and cryptorchids possessing an "extra" non-carcass component in the form of testes.

Table 2.13 A summary of selected results from gender group comparisons for dressing-out percentage (DO%).[1]

Author/s; Country; Comments	Gender	DO%
Everitt and Jury (1966b)	Ram	51.7^{ns}
No specified adjustments	Wether	50.1
~15kg CWT	Ewe	51.4
Fourie et al. (1970)	Ram	56.1^{ns}
No specified adjustments	Ewe	57.3
~ 29kg CWT		
Corbett et al. (1973)	Cryptorchid	45.8***
No specified adjustments	Wether	47.5
~13.5kg CWT	Ewe	47.8
	(se)	(0.18)
Kemp et al. (1981a)	Ewe	50.8^{ns}
No specified adjustments	Wether	50.0
Liveweights of 32, 41 and 50 kg	Wether	50.0
Butterfield et al. (1984a)	Ram	54.3**
No specified adjustments	Wether	57.2
~54kg CWT		
Bennett et al. (1991)	Ewe	49.4^{ns}
No specified adjustments	Wether	49.3
~13kg CWT		
Ellis et al. (1997)	Wether	45.0^{ns}
Adjusted to estimated SCF of 100g/kg	Ewe	44.7
~18.7kg CWT		
Mahgoub and Lodge (1998)	Ram	54.6
No specified adjustments	Wether	58.1
~28kg LWT	Ewe	58.4
	(se)	(0.96)
Carson et al. (1999)	Ram	45.3***
Adjusted to 22kg CWT	Ewe	48.2

Author/s; Country; Comments	Gender	DO%
Vergara et al. (1999)	Ram	48.7*
No specified adjustments	Ewe	50.8
Medium Weight *Data Used ~ 13.7kg*		
Wolf et al. (2001)	Ram	49.4[ns]
Adjusted to constant SLGWT	Ewe	50.9
~21kg CWT		
Wellington et al. (2003)	Rams	51.7[a]
Adjusted for SLGWT	Wether	55.0[d]
Adjusted for SLGWT	Ewes	56.7[d]

[t] Abbreviations are defined in Table 2.1

M. longissimus dimensions

Results for *M. longissimus* dimensions are harder to interpret than some other traits because covariate adjustments have varied between studies (Table 2.14). For those studies adjusted to a constant weight there was no difference between ewe and wether lambs, but cryptorchids tended to have wider *M. longissimus* than ewe lambs. Stanford et al. (2001) found no significant differences between ram and ewe lambs at the same age, whilst Wolf et al. (2001) found the muscle of ram lambs to be significantly wider. However, in both of these studies the ram lambs were heavier than the ewes so at a fixed weight the ewe lambs would likely have had larger *M. longissimus* dimensions.

Table 2.14 A summary of selected results from gender group comparisons for *M. longissimus* characteristics (*M. longissimus* area (EMA; (mm^2)); *M. longissimus* width (A; (mm)); *M. longissimus* depth (B (mm)).[t]

Author/s; Country; Comments	Gender	EMA	A	B
Everitt and Jury (1966a)	Ram	13.8[ns]		
Adjusted for CWT	Wether	15.4		
~15kg CWT	Ewe	11.2		
Corbett et al. (1973)	Cryptorchid	10.3[ns]		
No specified adjustments	Wether	10.5		
~13.5kg CWT	Ewe	10.8		
Barton and Purchas (1974)	Ewe			29.7[ns]
No specified adjustments	Wether			29.5
~14.9 kg CWT				
Kemp et al. (1981a)	Ewe	29.4[ns]	54.9[ns]	29.4[ns]
No specified adjustments	Wether	28.5	55.0	28.5
Liveweights of 32, 41 and 50 kg				
Field et al. (1990)	Ewe	13.7[ns]		
No specified adjustments	Wether	14.1		
~29kg CWT				
Bennett et al. (1991)	Ewe	8.8*	47.6*	26.8*
No specified adjustments	Wether	9.1	49.1	27.0
~13kg CWT				
Hopkins (1996)	Cryptorchid	13.4[ns]	60.4*	28.3[ns]
Adjusted to 22.5kg CWT	Ewe	12.8	56.4	28.8
Ellis et al. (1997)	Wether		61.0[ns]	28.3[ns]
Adjusted to estimated SCF 100g/kg	Ewe		58.8	28.0
~18.7kg CWT				

Author/s; Country; Comments	Gender	EMA	A	B
Jeremiah et al. (1997a)	Ram	15.0^{ns}		
40.5-49.5kg Results Used	Wether	14.0		
6-9 Mnth Old Results Used	Ewe	14.0		
Stanford et al. (2001)	Ram	10.3^{ns}	53.9^{ns}	24.9^{ns}
Adjusted to a fixed AGE and LWT	Ewe	10.8	56.6	25.6
Rams 43kg LWT; Ewes 37.5kg LWT				
Wolf et al. (2001)	Ram		77.5***	34.2
adjusted to constant age at scanning	Ewe		73.4	33.7
Rams 48kg LWT; Ewes 41kg LWT				
Wellington et al. (2003)	Ram	13.8^{ns}		
Adjusted for SLGWT	Wether	13.6		
~23.5kg CWT	Ewe	13.5		

[1] Abbreviations are defined in Table 2.1

Linear measurements

Of the papers reviewed few have considered linear carcass measurements (Table 2.15), additionally the anatomical positions used varied for the different measurements. The results show ram and cryptorchid lambs to have longer carcass length than ewe lambs, however, Ellis et al. (1997) found no difference in carcass length between wether and ewe lambs. Leg length was shown to be longer in wether lambs than ewe lambs by Bennett et al. (1991) but not significantly different by Ellis et al. (1997) or Everitt and Jury (1966b), however, in all three of these studies the data were adjusted differently. Ellis et al. (1997) and Everitt and Jury (1966b) also considered chest width and again found no significant differences. Testosterone causes the closing of the epiphyseal plates of bones, through wethers having low testosterone production, this closure does not occur resulting in elongated bones. However, in the comparison of ram and wether lambs made by Everitt and Jury (1996b), there was no significant difference in leg length, possibly the weights at which the animals were compared was not sufficient for this to be evident.

Table 2.15 A summary of selected results from gender group comparisons for linear measurements (carcass length (CcLe); leg length (LgLe); gigot width (GWi); chest width (ChWi)) (cm).[1]

Author/s; Country; Comments	Gender	CcLe	LgLe	ChWi
Everitt and Jury (1966b)	Ram	53.9***	19.9^{ns}	19.2^{ns}
Adjusted for CWT	Wether	51.4	21.4	18.7
~15kg carcass weight	Ewe	50.7	21.2	19.0
	(sd)	(0.81)	(0.41)	(0.30)
Wood et al. (1980)	Ram	54.1^{ns}	26.1^{ns}	
Adjusted CWT	Ewe	53.7	25.9	
~17.9kg CWT				
Kemp et al. (1981a)	Ewe			18.0^{a}
No specified adjustments	Wether			17.6^{b}
Liveweights of 32, 41 and 50 kg				

Author/s; Country; Comments	Gender	CcLe	LgLe	ChWi
Bennett et al. (1991)	Ewe		33.7*	
No specified adjustments	Wether		34.1	
~13kg CWT				
Hopkins (1996)	Cryptorchid	99.5*		
Adjusted to 22.5kg CWT	Ewe	97.8		
Ellis et al. (1997)	Wether	59.6^{ns}	25.6^{ns}	24.0^{ns}
Adj to est. SCF 100g/kg	Ewe	59.6	25.8	23.8
~18.7kg CWT				
Carson et al. (1999)	Ram	52.7***	27.0^{ns}	21.7^{ns}
Adjusted to 22kg CWT	Ewe	51.4	26.5	21.7
Wolf et al. (2001)	Ram	58.1***		
Adjusted to constant SLGAGE	Ewe	55.5		
~21kg CWT				

[1] Abbreviations are defined in Table 2.1

Carcass conformation

For conformation, the majority of studies, whether adjusting for age or weight, have found ewe carcasses to have superior carcass conformation to ram carcasses, although this difference has not always been significant (Table 2.16). In comparisons between wether and ewe lambs, there has been no clear trend.

Conformation as assessed by muscularity indexes was considered by Hopkins et al. (1996 and 1997) who showed there to be no differences in this index between cryptorchid and ewe lambs, when adjusted for carcass weight.

Table 2.16 A summary of selected results from gender group comparisons for carcass conformation (see comments for scales).[1]

Author/s; Country; Comments	Gender	Conformation
Field et al. (1990)	Ewe	3.9^{ns}
No specified adjustments	Wether	3.8
~29kg CWT		
4 point scale (4=best conformation)		
McClure et al. (1994)	Ewe	Higher*
	Ram	
Ellis et al. (1997)	Wether	2.9^{ns}
Adj to estimated SCF 100g/kg	Ewe	2.8
~18.7kg CWT		
5 point scale (5= best conformation)		
Jeremiah et al. (1997a)	Ram	2.7^{b}
40.5-49.5kg Results Used	Wether	3.1^{a}
6-9 Mnth Old Results Used	Ewe	3.1^{a}
5 point scale (5= best conformation)		

Author/s; Country; Comments	Gender	Conformation
Vergara et al.(1999)	Ram	5.4***
No specified adjustments	Ewe	7.2
~13.7kg CWT		
8 point scale (8= best conformation)		
Wolf et al. (2001)	Ram	12.8[ns]
adjusted to constant SLGAGE	Ewe	13.1
~21kg CWT		
15 point scale (15= best conformation)		

[1] Abbreviations are defined in Table 2.1

Cut distribution

Interpretation of cut distribution is difficult given that the way the results were reported varied considerably, and the limited number of comparisons between any combination of sexes (Table 2.17). As a result no conclusions can be made about distribution of cuts, because for example in the study which compared ram and ewe lambs, the ram lambs had heavier leg cuts than the ewes (McClure et al. 1994), whilst Seebeck (1968) found no difference.

Table 2.17 A summary of sex comparison studies for carcass cut distribution.[1]

Author/s; Country; Comments	Gender	Results				
Seebeck (1968)		Neck	Thorax	Loin/Flank	Shlder	Leg
13.5kg-35kg LWT	Ram	100[ns]	100[ns]	100[ns]	100[ns]	100[ns]
Adjusted to constant side muscle weight	Wether	99.1	97.9	102.7	102.1	99.3
Coefficients relative to rams	Ewe	94.5	97.9	103.6	100	100.9
Barton and Purchas (1974)		Leg	Loin			
No adjustment specified	Ewe	29.3[a]	27.1[a]			
~14.9 kg CWT	Wether	29.8[b]	26.1[b]			
% carcass						
Butterfield et al. (1984b)		Prox Pelvic	Dis Pelvic	Neck		
% carcass	Ram	27.0***	4.3*	16.7***		
	Wether	28.2	4.4	14.3		
Kemp et al. (1981a)		Leg	Loin	Shlder	Neck	
No specified adjustments	Ewe	30.1[ns]	10.4[ns]	23.1[a]	2.9[a]	
Liveweights of 32, 41 and 50 kg	Wether	29.8	10.2	23.8[b]	3.1[b]	
% carcass						
McClure et al. (1994)		Leg				
No specified adjustments	Ram	Heavier*				
~37-48kg LWT	Ewe					

[1] Abbreviations are defined in Table 2.1

Lean-meat-yield (LMY)

All studies reviewed found ram lambs to have a significantly higher LMY than ewe lambs and wether lambs when compared at the same weight (Table 2.18). No significant differences were reported for LMY between ewe and wether lambs.

Table 2.18 A summary of selected results from gender group comparisons for lean-meat-yield (% lean). Abbreviations are defined in Table 2.1

Author/s; Country; Comments	Gender	% Lean
Fourie et al. (1970)	Ram	53.8
No specified adjustments	Ewe	49.5
~ 30kg CWT		
McClelland et al. (1976)	Ram	54.3 ^{ns}
Adjusted to same proportion mature weight	Ewe	54.9
Rams ~17.2kg CWT; Ewes ~13.5kg CWT		
Wood et al. (1980)	Ram	57.0***
Adjusted for CWT	Ewe	55.5
~17.9kg CWT		
Butterfield et al. (1984b)	Ram	45.7*
	Wether	40.0
Hopkins et al. (1996)	Cryptorchid	57.5**
Adjusted to 22.5kg CWT	Ewe	53.9
Ellis et al. (1997)	Wether	55.6^{ns}
Adjusted to estimated SCF 100g/kg	Ewe	55.1
~18.7kg CWT		
Mahgoub and Lodge (1998)	Ram	58.0
Adjusted for CWT	Wether	55.0
~28kg LWT	Ewe	55.0
	(se)	(1.13)
Carson et al. (1999)	Ram	56.6*
Adjusted to 22kg CWT	Ewe	55.1
Wolf et al. (2001)	Ram	67.8***
Adjusted to 21kg SLGWT	Ewe	63.1
Díaz et al. (2003)	Ram	54.6**
~10-14kg LWT	Ewe	52.8
Wellington et al. (2003)	Ram	45.1^{a}
Adjusted for SLGWT	Wether	40.0^{b}
~ 23.5kg CWT	Ewe	38.0^{b}

† Abbreviations are defined in Table 2.1

Fat traits

The majority of studies (Table 2.19) have shown ewe and wether lambs to be fatter than ram or cryptorchid lambs for a given carcass weight. The exceptions to these results tend to be in studies where the sexes were at different slaughter weights (Stanford et al. 2001) in which case the differences were non-significant. However, in these studies the ewe lambs were always considerably lighter, and thus it is likely that at a constant weight the ewes would have been fatter than the ram lambs.

Kempster et al. (1987) also considered the distribution of fat, and showed ewes to have significantly more internal fat and less intermuscular fat when compared to wethers.

Table 2.19 A summary of selected results from gender group comparisons for fat traits (percentage fat in carcass (%Fat); fat depth over 12th rib, 110mm from midline (GR; mm); fat depth over *M. longissimus* (C; mm).[1]

Author/s; Country; Comments	Gender	FAT %	GR	C
Fourie et al. (1970)	Ram	33.9		
No specified adjustments	Ewe	41.6		
~ 29kg CWT				
Everitt and Jury (1966b)	Ram			2.6***
Adjusted to carcass weight	Wether			4.6
~15kg CWT	Ewe			4.7
	(sd)			(0.49)
Corbett et al. (1973)	Cryptorchid			2.6[ns]
No specified adjustments	Wether			2.9
~13.5kg CWT	Ewe			2.9
Barton and Purchas (1974)	Ewe			3.9[a]
No specified adjustments	Wether			3.4[b]
~14.9 kg CWT				
McClelland et al. (1976)	Ram	27.2[ns]		
Adjusted to same proportion mature weight	Ewe	27.8		
Rams ~17.2kg; Ewes ~13.5kg CWT				
Wood et al. (1980)	Ram	30.1***		
Adjusted for CWT	Ewe	32.2		
~17.9kg CWT				
Kemp et al. (1981a)	Ewe			5.1[ns]
No specified adjustments	Wether			4.6
LWT of 32, 41 and 50 kg				
Butterfield et al. (1984b)	Ram	45.6***		
	Wether	52.4		
Field et al. (1990)	Ewe		7.1[ns]	
~29kg CWT	Wether		6.6	
No specified adjustments				
Bennett et al. (1991)	Ewe	28.2*		2.6[ns]
No specified adjustments	Wether	27.1		2.5
~13kg CWT				
McClure et al. (1994)	Ewe			higher *
	Ram			
Afonso and Thompson (1996b)	Ewe			higher ***
	Ram			
Hopkins (1996)	Cryptorchid	26.8*	10.4**	2.0**
Adjusted for 22.5kg CWT	Ewe	30.9	15.0	4.1
Ellis et al. (1997)	Wether	23.2[ns]	3.00[ns]	2.88[ns]
Adjusted to estimated SCF 100g/kg	Ewe	23.8	3.25	3.10
~18.7kg				
Mahgoub and Lodge (1998)	Ram	23.7		
Adjusted for CWT	Wether	29.5		
~28kg LWT	Ewe	29.2		
	(se)	(1.36)		
Carson et al. (1999)	Ram	25.8***		2.9***
Adjusted for 22kg CWT	Ewe	29.4		3.7
Hopkins et al. (2001)	Cryptorchid		12.8[a]	3.5[a]
Adjusted to ~21.5kg CWT	Wether		15.5[b]	4.3[b]
	Ewe		15.1[b]	4.3[b]
Stanford et al. (2001)	Ram			4.50[ns]
Adjusted for age and SLGWT	Ewe			3.80
Rams 43kg; Ewes 37.5kg LWT				

Author/s; Country; Comments	Gender	FAT %	GR	C
Wolf et al. (2001)	Ram	16.1***		4.9***
adjusted 21kg CWT	Ewe	22.5		6.4
C measurement at constant scan age				
Díaz et al. (2003)	Ram	14.1***		
~10-14kg LWT	Ewe	19.7		
Wellington et al. (2003)	Ram	31.3[a]		
Adjusted for CWT	Wether	40.3[b]		
~23.5kg CWT	Ewe	43.9[b]		

[1] Abbreviations are defined in Table 2.1

Muscle to bone ratio (MTB)

Only seven of the papers reviewed considered sex comparisons for MTB (Table 2.20). For the studies comparing ewe and ram lambs, the ewe lambs had significantly higher MTB, except, in the comparison of Hopkins et al. (1996) where cryptorchid lambs had similar values to the ewe lambs. No differences were detected between ewe and wether lambs by Ellis et al. (1997).

Table 2.20 A summary of selected results from gender group comparisons muscle to bone ratio (MTB).[1]

Author/s; Country; Comments	Gender	MTB
Fourie et al. (1970)	Ram	5.9
No specified adjustments	Ewe	6.5
~ 29kg CWT		
Wood et al. (1980)	Ram	4.5*
Adjusted for CWT	Ewe	4.6
~17.9kg CWT		
Hopkins (1996)	Cryptorchid	3.7[ns]
Adjusted 22.5kg CWT	Ewe	3.6
Ellis et al. (1997)	Wether	2.9[ns]
Adjusted to estimated SCF 100g/kg	Ewe	2.9
~18.7kg CWT		
Mahgoub and Lodge (1998)	Ram	4.1
Adjusted for CWT	Wether	4.5
~28kg LWT	Ewe	4.6
	(se)	(0.13)
Carson et al. (1999)	Ram	3.7***
Adjusted to 22kg CWT	Ewe	4.0
Wolf et al. (2001)	Ram	4.7**
adjusted to constant SLGWT	Ewe	5.0
~21kg CWT		
Díaz et al. (2003)	Ram	2.2**
10-14kg LWT	Ewe	2.4

[1] Abbreviations are defined in Table 2.1

Meat quality traits

With the exception of Bickerstaffe et al. (2000), no differences between the genders were reported for any meat quality traits (Table 2.21 and Table 2.22). The majority of trials did not involve particularly heavy weights, and thus it is likely that testosterone production of the ram lambs had not peaked. If the animals had been heavier and older greater differences may have been found. This is supported by Misock et al. (1976) who showed that tenderness declined with increasing weights in ram lambs. Bickerstaffe et al. (2000) found differences between the mean pH of ram and ewe lambs that had been yarded together, but when they were kept separate there was no difference, this suggests that ram lambs when around ewe lambs, exert more "mating behaviours" depleting more muscle glycogen prior to slaughter.

In addition to the studies presented in Table 2.21 and Table 2.22, Jeremiah et al. (1993), carried out a retail survey to determine the retail acceptability of lamb rib chops from animals that differed in gender (ram, ewe, wether), and slaughter weight (40.5 – 76.8kg). The predominant reason retailers gave for not wanting to purchase any given product was its level of fat, which was evident for the ewe and wether lambs, but also the ram lambs at higher weights. The colour of the chops was also used by some retailers as a reason why they would not buy the product, especially for the lighter weight ram lambs.

Table 2.21 A summary of selected results from gender group comparisons for meat quality traits (ultimate pH (pHult); colour L* (L*); colour a (a*); colour b (b*); Warner Bratzler assessed tenderness (WB; kg); cooking loss percent (CL%).[1]

Author/s; Country; Comments	Gender	pH_{ult}	L*	a*	b*	CL%	WB[2]
Corbett et al. (1973)	Cryptorchid	5.7^{ns}					3.7^{ns}
No specified adjustments	Wether	5.7					4.1
~13.5kg CWT	Ewe	5.7					4.5
Misock et al. (1976)	Rams (29kg)						6.4^{b}
No specified adjustments	Rams (39kg)						8.0^{c}
	Rams (43kg)						8.2^{c}
	Wethers (46kg)						7.2^{bc}
Kemp et al. (1981a)	Ewe						4.2^{ns}
Longissimus Muscle	Wether						4.2
Liveweights of 32, 41 and 50 kg							
Kemp et al. (1981b)	Ewe						5.5^{ns}
No specified adjustments	Wether						5.5
Liveweights of 41 and 50 kg							
Dransfield et al. (1990)	Ram	5.7^{ns}	30.3^{ns}	14.9^{ns}	8.8^{ns}	18.5^{ns}	
No specified adjustments	Wether	5.6	31.3	14.6	9.2	18.7	
~18.3kg CWT	Ewe	5.7	30.9	14	8.7	19.4	

Author/s; Country; Comments	Gender	pH_{ult}	L*	a*	b*	CL%	WB[2]
Jeremiah et al. (1997b)	Ram		30.8[a]	6.4[ns]	4.3[ab]		
Adjusted for SLGWT	Wether		29.2[b]	6.5	4.2[b]		
40.5-49.5kg Results Used	Ewe		30.6[a]	6.4	4.6[a]		
Carson et al. (1999)	Ewe	5.7[ns]	39.3[ns]	16.1[ns]	10.3[ns]	21.3***	1.9[ns]
Adjusted to 22kg CCW	Ram	5.7	38.7	15.4	10.7	19.9	1.7
Vergara et al. (1999)	Ram		47.0[ns]	23.5[ns]	8.7[ns]		4.2[ns]
No specified adjustments	Ewe		48.4	22.6	9.0		4.1
~ 13.7kg CWT							
Bickerstaffe et al. (2000)	Rams (mixed)	6.1[a]					5.2[a]
Four treatment groups. For later 2	Ewes (mixed)	5.7[b]					6.1[a]
sexes kept separate at works	Rams (sep.)	5.7[b]					
	Ewes (sep.)	5.63[b]					
Díaz et al. (2003)	Ram	6.1[ns]	46.7[ns]	13.4[ns]	6.2[ns]		
10-14kg LWT	Ewe	6.2	46.2	13.4	6.3		

[1] Abbreviations are defined in Table 2.1

[2] W-B with the exception of Bickerstaff et al. (2000) who reported tenderness measurements using a MIRNZ Tenderometer

Table 2.22 A summary of selected results from gender group comparisons for meat quality as assessed by sensory evaluations (Tenderness (Ten); Juiciness (Jui); Flavour (Fla) and overall).[1]

Author/s; Country; Comments	Gender	Ten[1]	Jui[1]	Fla[1]	Overall[1]
Corbett et al. (1973)	Cryptorchid				4.1[ns]
No specified adjustments	Wether				4.0
~13.5kg CWT	Ewe				4.0
6 point scale (6=good)					
Misock et al. (1976)	Rams (29kg)	6.8[c]	6.1[bc]	5.8[c]	
No specified adjustments	Rams (39kg)	5.7[b]	5.8[b]	5.3[b]	
9 point scale (9=good)	Rams (43kg)	5.3[b]	5.8[b]	5.6[bc]	
	Wethers (46kg)	6.9[c]	6.3[c]	6.4[d]	
Kemp et al. (1981b)	Wether	7.2[ns]	7.0[ns]	6.8[ns]	6.8[ns]
No specified adjustments	Ram	7.2	7.0	6.8	6.7
LWT of 41 and 50 kg					
9 point scale (9=good)					
Kirton et al. (1983)	Ram (year)	7.1[ns]	6.5[ns]	7.2[ns]	7.5[ns]
No specified adjustments	Ewe (year)	7.1	6.4	7.1	7.4
10 point scale (10=good)	Ram (old)	5.9[ns]	6.0[ns]	6.8[ns]	6.5[ns]
	Ewe (old)	5.7	5.9	6.8	6.6
Dransfield et al. (1990)	Ram		1.1[ns]	1.7[ns]	2.2[ns]
~18.3 kg CWT	Wether		1.1	1.6	2.2
Overall – scale -7-7 (higher better)	Ewe		1.1	1.6	1.7
Others – scale 0-4 (higher better)					
Ellis et al. (1997)	Wether	3.1[ns]	3.8[ns]	3.7[ns]	2.5[ns]
Adjusted to estimated SCF 100g/kg	Ewe	3.0	3.7	3.6	2.4
~18.7kg CWT					
8 point scale (1=high)					
Arsenos et al. (2002)	Ram 45%	5.2[ns]	4.5[ns]	6.0[ns]	6.3[ns]
% mature body weight	Ewe 45%	5.1	4.5	5.9	6.1
Scale 1-8 (8=high)	Ram 90%	4.8[ns]	4.4[ns]	3.1[b]	5.7[ns]
	Ewe 90%	4.8	4.3	5.1[a]	5.8

[1] Abbreviations are defined in Table 2.1

2.5 Development of Quantitative Trait Loci Technology

Historically studies of genetic inheritance have involved consideration of single locus traits, these types of traits are qualitative traits and are easily assigned a phenotype e.g. horned or polled (not horned) (Mather, 1941). The concept of Quantitative Trait Loci (QTL) came from work of Sax (1923) (in Tanksley et al. (1982)), who demonstrated linkage between a Mendelian marker and a QTL using garden beans, where there was an association between colour alleles (markers) and seed size (quantitative trait). By the 1940's it was known that most traits are quantitative, that is they have gradual change between two extremes in a population (Mather, 1941). It was with this understanding that the concept of the infinitesimal model was developed, whereby it is hypothesised that quantitative traits are controlled by an indefinitely large number of genes (polygenes), all of which have small but cumulative effects. The challenge of identifying the location of these genes followed, with one of the first descriptions of the process involving the formation of linkage maps (Thoday, 1961).

2.5.1 Development of DNA Markers

The first attempt to use genetic markers as indicators of quantitative traits in livestock was carried out in dairy cattle by Neimann-Sorensen and Robertson (1961). In their work they considered whether or not blood group genes were associated with production characteristics in three breeds of Danish cattle. Although they found statistically significant associations between blood groups and production characteristics, the amount of variation in the production characteristic explained by the blood groups was not large enough to have any practical importance.

The blood group approach used by Neimann-Sorensen and Robertson (1961) meant that the number of markers was limited to the number of blood groups available. Blood groups are still used as a means of parentage testing for many species including cattle and horses. Over subsequent years most research relating to this field concentrated on the discovery of new marker types. Different types of markers have included the use of electrophoretic separation to distinguish differences between proteins (Lewontin & Hubby, 1966), polymorphisms in restriction enzyme sites (Solomon & Bodmer, 1979) and the use of naturally-occurring enzyme variation (Tanksley et al., 1982).

A major advance on these techniques was the discovery and reporting of what Southern

(1975) initially described as the detection of specific sequences among DNA fragments separated by gel electrophoresis. These later became known as Restriction Fragment Length Polymorphisms (RFLPs). The use of these DNA markers to create a genetic linkage map was first described by Botstein et al. (1980), who discussed their advantage as being their co-dominance. Through being co-dominant animals that were heterozygous and homozygous could be distinguished. Another advantage of RFLPs over previous markers was the large number available; this number is estimated in humans to be in the order of 3×10^5 (Beckmann & Soller, 1983). It was with these advantages in mind that Beckmann and Soller (1983) proposed the use of RFLP for detection and mapping of QTL, as was first carried out in tomatoes by Paterson et al. (1988).

The major discovery that was the precursor to modern day marker techniques was the development of the polymerase chain reaction (PCR) technique first described by Mullis et al. (1986). This technique involves the targeting of a DNA sequence using a pair of primers, this targeted sequence is then replicated through a series of temperature cycles which allows denaturing and replication of the targeted DNA sequence. This process is exponential in the number of copies of the targeted DNA sequence made.

The next advance in the use of DNA to provide markers was the discovery of minisatellite markers (or Variable Number Tandem Repeats (VNTR)) by Jeffreys et al. (1985). These multiple allele markers are tandem repeat markers (repeats of two nucleotide pairings e.g. GCGCGC), located mainly in telomeric regions of chromosomes. Although they are no longer used (superseded by microsatellites to be discussed below), highly informative minisatellites were discovered by Georges et al. (1990) and the first marker associated with the Callipyge locus in sheep was a minisatellite (Cockett et al., 1994).

Random Amplified Polymorphic DNA (RAPD) markers were first reported by Williams et al. (1990), and involved the amplification of random DNA segments with single primers of arbitrary nucleotide sequence. Although linkage maps can be generated using these markers, they are limited in that because of their randomness they do not specify a known locus, and a new map must be generated for each new pedigree.

The marker of choice during the 1990's and the early 2000's has been the microsatellite (also known as simple sequence repeats or SSR). Microsatellite markers were possible because of the development of the PCR technique and were first independently reported by three groups in 1989 (Litt & Lutty, 1989; Tautz, 1989; Weber & May, 1989). Microsatellite markers are characterized by simple sequence repeats. In most livestock species AC/GT nucleotide repeats occur randomly throughout the genome. Variation in microsatellites is due to differences in the number of repeats for a given marker resulting in different alleles. These markers are isolated by creating primers for each side of the repeats, such that each primer is a unique DNA sequence and isolates a particular position every time. They are advantageous in that they are co-dominant, and multiple alleles often exist for any marker in any population. Microsatellite markers have, since their discovery, superseded most other markers as the marker of choice for livestock linkage maps and QTL searches. For example, currently 77% of markers used in the ovine linkage map are microsatellites (http://www.thearkdb.org, May 2003).

Single-Stranded-Conformation-Polymorphisms (SSCP) are an alternative marker, based on the electrophoretic separation of single-stranded nucleic acids with subtle differences in sequence (often a single base pair). These differences result in a different secondary structure and a measurable difference in mobility through a gel (Orita et al., 1989).

Although it has long been known that changes in the nucleotide sequence of a gene results in mutations which can change the function of the gene, an even greater number of nucleotide sequence changes exist which do not change the function of the gene. An example would be a DNA sequence CGC which codes for the amino acid Arginine, a single nucleotide change to give CGG would still result in Arginine and therefore not change the function. With the development of the PCR technique these changes can be more readily discovered and are referred to as Single Nucleotide Polymorphisms (SNPs). Unlike microsatellites there is likely to be limited variation in SNPs with only two alleles at any one site within a population.

2.5.2 Development of Linkage Maps

Linkage maps can be constructed because some markers/genes (loci) are located sufficiently close enough together on chromosomes that their inheritance is not independent from one another. Such loci on the same chromosome (Chr) may show co-

segregation, i.e. progeny often inherit alleles that are of the same grandparental origin. The recombination fraction for a pair of loci represents the proportion of times that alleles at those loci do not have the same grandparental origin.

One of the first reports of modern day linkage maps using DNA was by Botstein et al. (1980) who discussed the need to be able to systematically map genes. In this work they used linkage relationships among RFLP markers using human pedigree. Creating linkage maps is considerably easier in livestock species when there are often large numbers of half-sib pedigree structures.

The first use of linkage maps to identify QTL was carried out by Paterson et al. (1988) using a complete RFLP linkage map of the tomato to successfully search for QTL. Lander and Botstein (1989) discussed in detail the benefits of using RFLP linkage maps for QTL detection in livestock as a step forward from the traditional mapping approach of studying genetic markers one-at-a-time (see 2.5.4).

Optimum mapping is achieved when the markers used are highly polymorphic (have multiple alleles at one locus), thus microsatellites are ideal markers (Montgomery et al., 1995). RFLP markers do, however, have a role because they are associated with gene sequences conserved across mammalian species, and they are therefore useful in defining regions of conserved synteny (Montgomery et al., 1995).

The first published linkage map in livestock was in cattle (Barendse et al., 1994). In this first report 202 loci were genotyped across cattle reference families. Four kinds of polymorphisms were used, minisatellites, SSCP, RFLPs and microsatellites. Conserved regions were detected between the bovine map and the human map of the time. A linkage map in sheep was published in the following year (Crawford et al., 1995). In this first report 246 loci, the majority of which were microsatellites derived from sheep or cattle that were genotyped through the international mapping flock. Extensive conservation of both order and distance between markers on the ovine and bovine maps was detected. Subsequently numerous updated maps have been published for cattle (Kappes et al., 1997; Sonstegard et al., 1997b) and sheep (de Gortari et al., 1998; Maddox et al., 2001), although more recent reports have been published at the chromosome level for cattle (e.g. (Kurar et al., 2002)) and sheep (e.g. (van Stijn et al.,

2003)). In May 2003 the bovine online linkage map contained 2725 loci and the sheep map 1722 loci (http://www.thearkdb.org/browser?species=cow&objtype=stats).

2.5.3 Experimental Designs for QTL searches

Crossing systems

The type of analysis required for detection of QTL, given phenotype and marker information, differs depending on the experimental design. There are a number of different types of experimental design that are implemented in QTL experiments, each of which offers different advantages and opportunities for any given trait. The different types of designs are summarised in Figure 2.2.

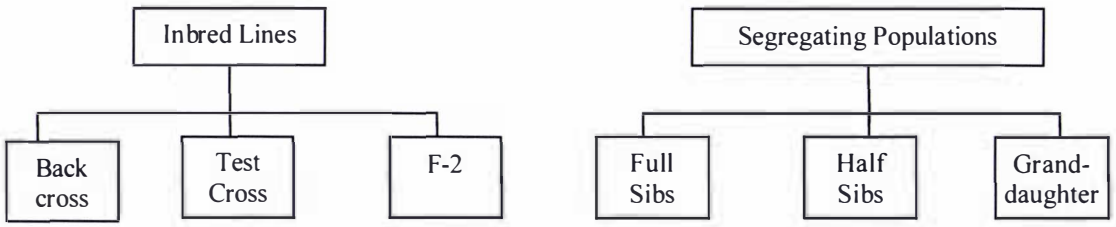


Figure 2.2 Experimental designs for QTL detection (adapted from Weller (2001))

Inbred lines have been widely used in QTL studies. Inbred lines are assumed to have fixed alleles for given loci. The underlying principle of such studies is to take two diverse populations that differ in performance for given traits, and by crossing them cause segregation of different alleles for genes of interest. This enables the search for marker allele/phenotype association studies. Such studies first involve the generation of a first cross population (F1) by the mating of the inbred parental strains. Backcrossing involves mating F1 individuals back to one of the parental strains. Testcrossing involves mating F1 individuals to another (3^{rd}) inbred strain. By crossing F1 individuals a second cross population (F2) can be created. One of the first reported uses of the F2 design to search for QTL in domestic livestock species was in pigs as described by Andersson et al. (1994). In their work they crossed European wild boar and domesticated Large White pigs to generate F1s in the search for QTL for growth and fat traits and then interbred them to generate the F2 population. Since this time the F2 design has been used extensively in pigs (e.g. Walling et al. (1998) and Perez-Enciso (2000)). Other alternatives have also been used in pigs and have involved combinations of the basic design such as backcrossing the F2's to the parental breeds

(Marklund et al., 1999). The F2 design has also been used in cattle, although not as extensively (Napolitano et al., 2001). The F2 design offers the ability to generate both homozygotes and heterozygotes within a population, and thus observe the effect of receiving one or two copies of any given QTL.

Inbred lines do not exist for many species (humans are an example), and thus an alternative design is to use segregating populations and to use existing or created family structures as the framework for QTL analysis. Half-sib designs, which most often involve a common sire, were first described by Neimann-Sorensen and Robertson (1961) in their searches for associations between blood groups and production traits in dairy cattle. In order for the half-sib design to be effective, the sire used must be heterozygous at the markers used so that approximately half the resulting progeny are carrying each of the alternate alleles of the sire. Furthermore, it must be possible to deduce which allele was inherited from the sire, instances where this is not the case are where the progeny are heterozygous for the same alleles as the sire. Such animals are defined as "uninformative". Half-sib designs have, been used in a number of studies to search for QTL, predominately in cattle (Beever et al., 1990; Napolitano et al., 1996; Casas et al., 2000). The term daughter design also refers to a half-sib design and was first used by Weller et al. (1990) in his description of options for QTL experiments in the dairy industry, in which a sire and all of his daughters are genotyped and the daughters phenotyped.

The full-sib design is less common because of the relatively small number of full-sibs that can be generated for most species. Statistical methods were theoretically developed which can accurately analyse even small full-sib data sets (Haseman & Elston, 1972). As was the case for the half-sib design ideally both the sire and dam are heterozygous, preferably for different alleles, so that alleles originating from the sire and dam can unequivocally be determined. If the above does not hold true, an animal will be uninformative.

An alternative to the daughter design is the granddaughter design (Weller et al., 1990) which was specifically developed for use in the dairy industry and involves a sire and his progeny test sons being genotyped, with the phenotypic data collected on the daughters of the progeny-tested sons but used to detect QTL in the original sire

(grandsire). This type of design has been used to detect QTL in North American Holstein Friesian populations (Heyen et al., 1999) and the New Zealand and Dutch dairy populations (Spelman et al., 1996; Grisart et al., 2002), as the phenotypes are already generated from the progeny testing of bulls for use in artificial insemination.

Animals to use

Quantitative Trait Loci of interest are likely to be found only in certain breeds, or lines within breeds, as they require a differential allele to exist, which is associated with a certain phenotype. Ideally QTL experiments involve animals that are diverse for the phenotype of interest in the first instance, as this increases the likelihood of detecting a QTL. For beef cattle many QTL searches have involved crosses between *Bos taurus* and *Bos indicus* breeds, for pig QTL searches, crosses between diverse domesticated breeds (e.g. Large White crossed with Meishan) and for dairy cattle breeds such as Jersey and Friesian (Spelman et al., 1998). Reports of non-associations between a particular marker/gene and a phenotype must be interpreted within that particular population only. Likewise if QTL are reported, consideration needs be given as to whether it is within the breed or limited to the specific population in which the search was carried out.

2.5.4 Analysis of Data From Experiments Designed to Search for QTL

Methods for QTL analysis have been developed since the early 1970's, with different types of analysis being required for different experimental designs. Jayakar (1970) described a statistical approach for detection and estimation of linkage for a locus affecting a quantitative character. In their example of a backcross between M_1M_1 and M_1M_2 individuals, they showed that in the absence of linkage there was a mixture of normal distributions in the phenotypes for the trait of interest, all with means of zero (a symmetric unimodal distribution). However, if linkage existed between the QTL and the phenotypes, the distribution was multi-modal. In this early work, the markers were blood groups, which as previously discussed, limited the amount of information available and success of analysis. Weller (1986) described the use of a maximum likelihood technique for mapping QTL in a segregating F-2 generation of inbred founder lines. This technique was based on recombination frequencies between two loci and the means and variances of the genotypes, it, however, suffered the limitation of not easily being able to detect QTL with small effects.

The limitations of all these early methods were that the effects were only estimated at the point of the markers, and thus underestimated the size of the effect if in fact the QTL lay between the markers (Paterson et al., 1988). This was overcome by the development of interval mapping, which allowed inference about points throughout the entire genome, by using information from flanking markers based on recombination rates (Paterson et al., 1988; Lander & Botstein, 1989). Options for interval mapping include least-squares or maximum likelihood. Although maximum likelihood interval mapping is useful, it is computationally demanding and requires specially written software. In comparisons between the two methods, Haley and Knott (1992) concluded that similar results were obtained using the two methods. Furthermore, the greatest amount of information relating to detection of a QTL came from the phenotypic differences between marker genotypes and relatively little from the genotype distribution. Haley et al. (1994) then went on to show that this least-squares approach could also be used for mapping QTL in outbred lines. Subsequently, a number of comparisons of the two methods have shown that the most significant difference is the greater computational time requirements of the maximum likelihood method. It also requires pre-processing of data to remove fixed effects, whereas the least-squares method enables fitting of fixed effects and the QTL probabilities at the same time (Knott et al., 1996; Kao, 2000).

Other statistical aspects that have become important to the estimation of QTL effects/associations include estimating a confidence interval associated with a QTL and estimating statistical thresholds for the data. Visscher et al. (1996) described the development of empirical confidence intervals around a QTL using the bootstrapping approach. Bootstrap samples were created through recreating the data set by sampling-with-replacement individuals from the original data set for a particular sire, with the number sampled equal to the original number and rerunning the analysis. They proposed the use of 500 replicates from which the 90 and 95% confidence intervals could be estimated. Setting of significance thresholds in QTL studies is important to eliminate the possibilities of false positives. Doerge and Churchill (1996) described the permutation-test approach for setting significance thresholds, that involves shuffling of the phenotypes among the genotypes and rerunning the analysis. After a user defined number of replications, the thresholds required to gain any significance level can be determined. Meanwhile Lander and Kruglyak (1995) and Kruglyak (1996) developed

conservative, independent guideline significance thresholds from which "suggestive", "significant", "highly significant" and "confirmed linkage" criteria for the existence of a QTL at a given locus could be determined. The thresholds varied depending on the type of design that was used to generate the progeny.

2.5.5 Marker Assisted Selection (MAS)

When QTL are detected, they can be exploited via MAS, through identification of a haplotype, in terms of certain alleles at specific markers, which can be tested for in chosen animals (Meuwissen & Goddard, 1996). The aim of MAS is to get early information on animals without awaiting phenotypes. Meuwissen and Goddard (1996) demonstrated through simulation, how using MAS could increase rates of genetic gain compared to traditional selection procedures (such as progeny testing), particularly for traits that have a low heritability (fertility traits), are sex limited (milk production) or when there is a long delay in obtaining phenotypic information (slaughter data). Although considerable literature exists considering the use of MAS from a theoretical point of view (Henshall & Goddard, 1997; Spelman & van Arendonk, 1997; Spelman et al., 1999a), there are very few reports of actual industry use of MAS. This is likely due to a shortage of suitable candidate QTL for MAS (as will be discussed in the next section), and commercial secrecy.

2.6 Quantitative Trait Loci Affecting Growth, Composition and Meat Quality Characteristics for Sheep and Cattle

Only QTL affecting growth and carcass traits in cattle and sheep will be considered here. The QTL reviewed are not an exhaustive list, rather it is mainly restricted to those published in refereed journals, as these are usually based on stronger evidence. However, references to relevant QTL from recent conference proceedings are included.

2.6.1 Cattle

Muscle-hypertrophy locus

The muscle-hypertrophy condition in the Belgian Blue cattle breed has long been recognised and the locus controlling this phenotype was designated *mh* (muscle hypertrophy) by Hanset and Michaux (1985). This trait represented one of the first attempts to determine the chromosomal location of the locus. Muscular hypertrophy is a good illustration of how an observable phenotype, can lead to identification of QTL

and then the underlying gene, a sequence of events that exists for very few traits.

The first reported scan of the genome to detect the location of the *mh* locus was described by Charlier et al. (1995), who screened the entire bovine genome using 213 microsatellite markers from a backcross population (Belgian Blue sires mated to black and white dams (F1); The F1 progeny were then mated back to Belgian Blue sires). This study was able to narrow down the region of the locus to the centromeric end of bovine Chr2. Additional markers in the region were mapped by Casas et al. (1998), who narrowed its position to between 2 and 6cM from the beginning of the linkage group (centromere), with Sonstegard et al. (1997a) showing it mapped close to the position of the alpha collagen type III locus (COL3A1) which was also mapped in humans. The work of Sonstegard et al. (1997a) showed that the region of interest on bovine Chr2, mapped to human Chr2 and showed conserved synteny for a number of genes in the region. At a similar time McPherron et al. (1997) discovered that deletions of the growth differentiation factor 8 gene (GDF8 or Myostatin) in mice caused an increased muscling phenotype similar to that seen in Belgian Blue. Smith et al. (1997) showed that GDF8 mapped to the same region as the *mh* locus in cattle and was therefore a strong candidate gene.

Kambadur et al. (1997), after sequencing the GDF8 gene, compared the sequence from Belgian Blue and Friesian (non hyper-muscled) cattle, and found an 11-base pair deletion in the coding region, which effectively disables the genes ability to control muscle growth. This was also independently verified by Grobet et al. (1997). Subsequently a number of different mutations to the gene have been shown to be responsible for increased muscling phenotypes in other breeds of cattle including the Asturiana de los Valles (Dunner et al., 1997), Piedmontese (Kambadur et al., 1997), Charolais, Gasconne, Maine-Anjou, Parthenaise, Asturiana, and Rubia Gallega (Grobet et al., 1998). An interesting breed in which evidence for mutations to GDF8 have been found is the South Devon as described by Smith et al. (2000a), a breed selected for good muscle conformation but against double muscling. It was hypothesised that the South Devon animals were predominately heterozygous with an intermediate muscle phenotype. Specific trials have also investigated the exact effects of the *mh* locus, with Casas et al. (1998) looking at the inheritance of one copy of the *mh* allele on birth and carcass traits. Relative to the alternative allele, the *mh* allele was shown to increase rib-

M. longissimus area, retail product yield and birth weight, whist decreasing marbling, yield grade, fat percentage and pH. The exact sizes of the effects were not reported.

Loci affecting tenderness

The only other locus with which the associated gene has been discovered is associated with beef tenderness. Two initial QTL searches led to the discovery of this gene. The first by Keele et al. (1999) was a genome scan (196 markers) for QTL affecting tenderness as assessed by Warner-Bratzler measurements in a population derived from crossing Brahman x Hereford bulls to *Bos taurus* cows. This study identified a region of bovine Chr15 influencing tenderness, which explained 26% of the phenotypic variation. In the following year, Casas et al. (2000) carried out a genome scan searching for QTL affecting many traits in two half sib families (Belgian Blue cross MARCIII (¼ Angus, ¼ Hereford, ¼ Red Poll, ¼ Pinzgauer) and Piedmontese cross Angus). A region of bovine Chr29 which accounted for variation in tenderness was of particular interest, although the exact proportion accounted for was not reported. A potential candidate gene for tenderness was identified from the human genome as being micromolar calcium-activated neutral protease (CAPN1 or Calpain-I). This gene maps to human Chr11, but because of interruptions in the conserved synteny in the region of this gene it could either map to bovine Chr15 or 29 (Smith et al., 2000b).

Smith et al. (2000b) went on to show that the CAPN1 gene maps to bovine Chr29, in the middle of the region identified by Casas et al. (2000). That CAPN1 is the gene responsible for differences in tenderness was confirmed by Page et al. (2002), who sequenced the CAPN1 gene from phenotypic extremes for meat tenderness using two populations (Piedmontese cross Angus and Limousin cross Jersey), and showed two SNPs to be responsible for the difference. The sire and dam effects, together, accounted for over 30% of the residual variance in tenderness (Cullen et al., 2003). The QTL that was detected on Chr15 has also been followed up, and although a candidate gene, MYOD1, has been hypothesised its involvement has not yet been confirmed (Rexroad III et al., 2001).

Other QTL affecting growth and carcass traits

No other QTL in cattle affecting growth or carcass traits have been followed up to the point of discovering the underlying gene mutation. Indeed, a number of QTL that have

been discovered, have not been followed up in subsequent work or have been followed up commercially (e.g. tests for marbling and tenderness are commercially available from Genetics Solutions, Australia www.geneticsolutions.com.au).

One of the first QTL searches for growth and carcass traits in cattle was that of Beever et al. (1990) using a paternal half-sib family of Angus cattle. Although "modern markers" such as microsatellites had been developed by this time, they were not in widespread use, and this work used polymorphisms in BoLA-A (class I major histocompatibility complex), B, C, and F blood systems, serum transferrin (Tf) and vitamin D binding protein. Associations between polymorphisms for the B blood group and weights and carcass fatness were identified, as was an association between BoLA and EMA. However, there are no reports of follow up work.

Rocha et al. (1992) used restriction fragment length polymorphisms (RFLPs) associated with genes known to be involved with growth including growth hormone (GH), prolactin, osteonectin and parathyroid hormone to search for QTL. They used half-sib populations (Charolais and Red Poll sires) and a diallele experiment involving five breeds (Angus, Brahman, Hereford, Holstein, and Jersey). They found an association between polymorphisms in GH and shoulder width at birth and maternal effects on birth and weaning weights. Associations between variations in parathyroid hormone alleles and measures of body size were also detected. This work was followed up by Taylor et al. (1998), who used growth hormone one (GH1) which had been mapped to bovine Chr19, as a starting point (candidate gene) for developing microsatellite markers to search for QTL affecting growth and composition traits in a *Bos indicus* cross *Bos taurus* population. Two distinct QTL on Chr19 were detected. The first QTL was in the region of the GH1 gene, with an association between the locus and subcutaneous fat thickness; with the effect approximately 0.4 of the adjusted standard deviation. The second QTL lay outside the region of GH1, this QTL influencing the unsaturated to saturated fatty acid profile of subcutaneous fat over the *M. longissimus*, with the effect equal to approximately 0.5 of the adjusted standard deviation. However, the work of Di Stasio et al. (2002), although finding variation in the genotypes, failed to find any association between GH1 (and an additional gene for pituitary transcription factor Pit-1 (POU1F1)) and fourteen traits including conformation traits. This work was carried out using a Piedmontese cross population, whilst that of Taylor et al. (1998) involved a

reciprocal backcross population of Angus and Brahman animals. The use of very different populations could explain why different results were obtained.

Moody et al. (1996), like Rocha et al. (1992), used loci known to be involved in growth to search for QTL within three populations of Hereford cattle. In this work Kappa-casein, beta-lactoglobulin and insulin-like growth factor 1 polymorphisms were shown to be associated with birth weight and daily gain to weaning, whilst pituitary transcription factor 1 polymorphisms were associated with birth weight. However, there have been no follow up reports of this work.

Napolitano et al. (1996) searched for QTL in a Chianina cross Piedmontese population using only three specific markers IDVGA-2 (Chr2); IDVGA-3 (Chr11) and IDVGA-46 (Chr19). They found evidence for an association between polymorphisms at marker IDVGA-46 and variation in dimension traits (such as height at withers, width of chest and length of rump). Associations between polymorphisms and height at the withers were found for IDVGA-2 and IDVGA-3. The association of dimension traits with IDVGA-46 was again confirmed in work by Napolitano et al. (2001).

A number of genome scans carried out by Casas et al. (1998; 2000; 2001) have attempted to identify regions other than GDF8 which contribute to variation in meat and growth traits using Belgian Blue cross and Piedmontese cross populations. This work has identified suggestive QTL, which were unique within each breed. For the Belgian Blue population, QTL for marbling score and retail product yield were identified on Chr3. For birth weight, yearling weight, hot carcass weight and *M. longissimus* area QTL were detected on Chr4. Quantitative Trait Loci were detected on Chr6 for carcass weight and on Chr4 for post-weaning average-daily-gain. Furthermore, QTL for marbling were detected on Chr8, 10, 17 and 27, with an additional fat QTL on Chr8, and finally a QTL for Warner-Bratzler shear force was detected on Chr9. For the Piedmontese population, suggestive QTL for fat depth, retail product yield and USDA yield grade were detected on Chr5, with an additional QTL for fat depth on Chr8, whilst QTL for Warner-Bratzler measurements were detected on Chr29. This latter QTL affecting tenderness was later confirmed as the CAPN1 gene, discussed above.

Stone et al. (1999) and Casas et al. (2003) have reported the search for QTL affecting

growth and carcass composition using a *Bos indicus* x *Bos taurus* (Brahman x Hereford), mated to *Bos taurus* cows. Stone et al. (1999) reported strong evidence for QTL affecting dressing-out percentage and rib bone on Chr5, and suggestive evidence for QTL affecting retail product yield on Chr2 and Chr13, *M. longissimus* area on Chr14 and birth weight on Chr1. The QTL on Chr2 mapped between markers TEXAN2 and ILST30 (distinct from GDF8). Casas et al. (2003) reported the complete scan on the same population, with a considerably different outcome to Stone et al. (1999). Casas et al. (2003) reported significant QTL for birth weight on Chr5 and Chr 21, *M. longissimus* area on Chr5 and Chr6, retail product yield on Chr9, and marbling score on Chr23. Twenty-seven further suggestive QTL were reported. The estimated position of the retail product yield QTL on Chr2 changed, with it reported as mapping to the region of marker RM356, approximately 20cM from the original estimate of Stone et al. (1999).

No other reports of cattle carcass trait QTL have been located in published journals. There is a great deal of variation throughout the genome that is likely to account for variation in most important meat traits. The fact that QTL are only detected in some populations is to be expected, as diverse phenotypes are required to identify them. This is why crosses between diverse breeds such as *Bos taurus* and *Bos indicus* breeds have been so useful in detecting QTL, likewise, with crosses between Belgian Blues and other breeds because of their extreme muscling phenotype.

2.6.2 Sheep

Relative to cattle, few QTL influencing meat production and quality traits have been mapped and published in sheep. All that have been published, have stemmed from notable phenotypic differences. However, in each case, there has been considerable follow-up work. The locus for which the most work has been carried out is the Callipyge, the other three are the Rib-Eye Muscling locus and loci associated with increased muscling phenotypes in the Texel and Suffolk.

Callipyge loci

The first to report an increased muscling phenotype in a Dorset flock of sheep in the United States was made by Jackson et al. (1993a; 1993b). Jackson and Green (1993) further characterised this phenotype by showing lambs exhibiting this phenotype did not grow faster, but had improved feed efficiency. The background of these animals was

described by Cockett et al. (1994), who reported how in 1983 a sheep producer identified a ram with extreme muscling, a phenotype which was passed on to some of his progeny and descendents, which suggested an heritable mutation. At this time the phenotype was named Callipyge (Greek for beautiful buttocks; CLPG). Cockett et al. (1994) carried out an initial genome search using four bovine minisatellite markers, as relatively few markers had been mapped to the ovine genome at the time. There was an association between marker GMBT16 and the phenotype. This marker mapped to bovine Chr21 and ovine Chr18. Further markers in the region of GMBT16 were mapped, and the region of the CLPG locus narrowed to a 14.5cM region near markers CSSM18 and TGLA122.

An interesting point about the inheritance of CLPG that was first reported by Georges and Cockett (1996), was that only heterozygous individuals having inherited the CLPG mutation from their sire expressed the phenotype. This unusual mode of inheritance was described by Cockett et al. (1996) as resulting from "Polar Overdominance". This mode of inheritance was confirmed by Freking et al. (1998a), who also mapped further markers to the region of interest to refine the chromosomal location of the gene. This work confirmed the location of the QTL to be at the telomeric end of Chr18, specifically within a 3.9cM interval. This region is conserved between species, and by chance imprinted genes DLK1/GTL2 were mapped to this region in humans (Fahrenkrug et al., 2000; Wylie et al., 2000), providing an excellent starting point for determining the underlying genes in Callipyge lambs. That the Callipyge locus maps to this area was confirmed by Berghams et al. (2001) who mapped the locus to within a 450 kilobase chromosome segment, a segment which also contained the DLK1 and GTL2 genes.

Charlier et al. (2001) demonstrated that additional imprinted genes, MEG8 and PEG11 also mapped to this region, and that DLK1 and PEG11 were paternally expressed, whilst GTL2 and MEG8 were maternally expressed. Freking et al. (2002) reported the identification of a single base change upstream from GTL2 that was consistent in animals known to have inherited the genotype. Smit et al. (2003) genotyped the founding sire, and discovered an inconsistent allele frequency at the site of the single base change, a phenomenon not seen in any of his progeny. This suggested that the point mutation occurred during the rams early embryonic development, and resulted in the ram being mosaic. Furthermore, it was estimated that only 10% of the 150 offspring

produced by the sire expressed the Callipyge phenotype, suggesting that he was also germline mosaic. A hypothesis to explain how this mutation and its imprinted nature causes the phenotype has recently been presented by Georges et al. (2003), however, the details of this are complex and outside the scope of this review.

Considerable phenotypic data have been collected on animals expressing the Callipyge phenotype versus those that are not. All studies have shown Callipyge lambs to have significantly ($P<0.01$) more muscle than their non-Callipyge counterparts as assessed by muscle weight (Jackson et al., 1993a; Koohmaraie et al., 1995; Carpenter et al., 1996; Jackson et al., 1997a; Freking et al., 1998b; Goodson et al., 2001; Abdulkhaliq et al., 2002) and *M. longissimus* dimensions and areas (Leymaster & Freking, 1976; Jackson et al., 1994b; Snowder et al., 1994; Carpenter et al., 1996; Field et al., 1996; Jackson et al., 1997a; Leymaster & Freking, 1998; Abdulkhaliq et al., 2002). As a result of the increased muscle, there is significantly less fat in Callipyge lambs (Jackson et al., 1993b; Busboom et al., 1994; Snowder et al., 1994; Jackson et al., 1997a; Freking et al., 1998b; Goodson et al., 2001; Abdulkhaliq et al., 2002). Callipyge lambs do not differ in growth rates (Jackson & Green, 1993; Abdulkhaliq et al., 2002), however, for a given liveweight, they have heavier carcasses and therefore a higher DO% (Jackson & Green, 1993; Jackson et al., 1993b; Snowder et al., 1994; Koohmaraie et al., 1995; Field et al., 1996; Jackson et al., 1997a; Freking et al., 1998b; Goodson et al., 2001; Abdulkhaliq et al., 2002). A problem first identified by Jackson et al. (1994a), was that meat from Callipyge lambs was significantly less tender ($P<0.001$) than lambs of normal phenotype, as shown by Warner-Bratzler shear tests and taste panels. This result has since been confirmed in a number of experiments (Kerth et al., 1995; Koohmaraie et al., 1995; Field et al., 1996; Shackelford et al., 1997; Freking et al., 1999; Goodson et al., 2001; Abdulkhaliq et al., 2002). Although Shackelford et al. (1997) demonstrated there is variation between muscles as to the difference in tenderness, with M. *longissimus* most affected, but others such as M. *quadriceps* less affected. The exact reason for the increased toughness is not known, however, it is hypothesised that it is influenced by the post-mortem stability of myofibrils, which is likely to involve calpastatin activity (Taylor & Koohmaraie, 1998; Duckett et al., 2000).

Rib-Eye Muscling Locus

Information on the Carwell QTL has only been reported at conferences. Banks (1997)

was the first to report an increased *M. longissimus* dimension phenotype in an Australian stud Poll Dorset flock. Given that the phenotype resembled a less extreme version of the Callipyge animals discussed above, the telomeric portion of ovine Chromosome 18 was considered as a candidate region for the QTL (Nicoll et al., 1998). Work by Nicoll et al. (1998) confirmed that the Carwell locus, like the Callipyge, mapped to Chr18, and at the time it was suggested that the two were possibly different allelic forms at the same locus. Jopson et al. (2001) renamed the locus the Rib-Eye Muscling (REM) locus, based on the work of Nicoll et al. (1998), which determined that the effect of the locus was limited to *M. longissimus* muscle, and gave an increase in *M. longissimus* weight of 8% relative to normal animals for the same weight.

Given that it was hypothesised that the REM locus and Callipyge locus were the same, an important consideration was whether or not the REM locus, like the Callipyge had a negative effect on meat quality. Jopson et al. (2001), found that although the meat from REM animals was slightly tougher than that from normal lambs, this effect was overcome with the use of commercial New Zealand aging techniques. Jopson et al. (2001) also considered the mode of inheritance and showed, that muscling was increased, irrespective of which parent the allele was inherited from. This suggested that it followed a normal mode of inheritance, in contrast to the polar overdominance inheritance pattern of the Callipyge locus. In light of the different modes of inheritance and types of effects, it was thought unlikely that the two were different allelic versions of the same locus. This has recently been confirmed, with the REM locus having been fine-mapped to an approximately 500kbp region that does not overlap with either the Callipyge mutation or the associated cluster of imprinted genes (McLaren et al., 2003).

Variation in muscling within the Texel breed

The following is a review of the studies that led to the experimental work reported in this thesis.

The Texel is known for its superior muscling phenotype relative to most other terminal-sire breeds (see Section 2.3.3). Although not as extreme as that seen in Belgian Blue cattle, it was hypothesised that mutations to the GDF8 gene could be responsible (Marcq et al., 1998). To test this hypothesis, Marcq et al. (1998) compared the sequences of the GDF8 gene between Texels and Romanovs (non-muscular breed). No

sequence differences were found. In follow up work in which an F2 population of Texel x Romanovs were generated, there was considerable evidence (lod score >3) for a QTL affecting muscling, for two of the 39 traits tested (one of which was conformation), in the region of GDF8. This information stimulated three research groups to follow up the search for a QTL affecting muscling in the Texel breed of sheep in the region of GDF8.

Broad et al. (2000) carried out a screen of the purebred New Zealand Texel population, looking for associations between ultrasound measurements (*M. longissimus* dimensions and fat depth) and markers located in the region of GDF8 on ovine Chr2. As the animals used were purebred, it enabled some observations showing that the level of polymorphism around the GDF8 locus was limited, with a number of the rams homozygous for all markers in the region (24 out of 26). Of the twelve sires showing heterozygosity, only three showed evidence for a QTL affecting muscle and fat in the region. The size of the QTL detected explained at most 10% of the phenotypic standard deviation for the traits concerned. Further details are in the introduction to Chapter 4.

Walling et al. (2001) carried out a search for QTL within the purebred British Texel population, using markers on Chr2 and Chr18. These were chosen because of their association with increased muscling phenotypes in other species/breeds (GDF8 and Callipyge). Ultrasound measures of *M. longissimus* dimensions and fat depth above M. *longissimus* were recorded at eight months of age. This work provided evidence for a QTL affecting muscling on ovine Chr2 at approximately 60cM and a QTL affecting muscling and fat at 170cM. A QTL affecting muscling and fat was also identified on Chr18, in the region of the Callipyge and REM loci. Walling et al. (2002) reported results of scans based on the same animals on five further chromosomes (3, 4, 5, 11 and 20). This work provided evidence for two further QTL affecting total muscle on Chr3 and Chr20, whilst there was evidence for a QTL affecting fat on Chr4. The regions identified are known to include important genes involved in growth, with IGF1 mapping to Chr3, leptin to Chr4 and the major histocompatibility complex to Chr20.

Marcq et al. (2002) followed up on their original work by generating more F2 animals (Texel x Romanov) from which carcass measurements and weights were collected. Preliminary results presented for chromosomes 1, 2, 9, 14, 18 and 21 provided strong

evidence based on 12 of the measurements taken (e.g. muscle weight, muscle percentage, fat weight and fat percentage) for a QTL affecting muscle and fat on Chr2, and in particular in the region known to contain the GDF8 gene. The GDF8 gene of the Texels and Romanovs were sequenced, including upstream and downstream sections, but, no single polymorphism could explain the phenotypic differences. Furthermore, they found no differences between the breeds in the expression of the GDF8 gene at the mRNA level in skeletal muscle. Screening on a backcross Texel x Romanov population also failed to provide significant evidence for a QTL, thus suggesting that there may not be a simple mode of inheritance.

Variation in muscling within the Suffolk breed

The work of Walling et al. (2002), in addition to involving a search for QTL in the purebred British Texel population, also involved a search within the purebred British Suffolk population. The same chromosomes as were scanned in the Texels were used (2, 3, 4, 5, 11, 18 and 20), and in addition chromosomes 1 and 6 were scanned. Only one QTL affecting muscling was detected in the Suffolk population on Chr1, whilst two affecting fat were found on chromosomes 3 and 20. The size of these effects varied between 0.5 and 0.8 of a phenotypic standard deviation. At the time of writing this literature review there had been no follow up reports to the discovery of these QTL.

2.7 Conclusions

The New Zealand sheep industry

The New Zealand sheep industry is characterised by the following:

- 40 million sheep in New Zealand, 70% of which are breeding ewes.
- 95% of New Zealand lamb is exported, accounting for 10% of NZ export income.
- Terminal sire breeds (e.g. Suffolk or Texel) used for prime lamb production.
- Move towards export of fresh, processed into saleable cut product.
- Consumers preferring increased lean and improved quality.
- Meat processors investigating yield-based payment, and monitoring of meat quality.

Based on the above characteristics, prime lamb production is important in New Zealand. Meat processors should implement yield-based payment to provide better market signals to producers, as to the requirements of the consumer.

Texel-sired lambs versus other terminal-sired-lambs

The Texel has been included as a terminal sire breed in a number of breed comparison studies. However, results have been variable, and it is difficult to draw firm conclusions about the attributes of the breed. Relative to other terminal sire breeds Texels show:

- Lower growth rates
- Higher dressing-out percentages, although often not statistically significant.
- Shorter carcasses.
- Larger *M. longissimus* dimensions.
- Improved conformation scores, although often not statistically significant.
- Less weight in neck and loin region, and more in the leg.
- Higher lean-meat yield.
- Lower carcass fat percentage.
- Similar GR measurements.
- No differences in meat quality characteristics.

Based on the above attributes, Texel-sired lambs are likely to have only limited advantage over other breeds with the existing payment system based on carcass weight and GR. However, with the proposed introduction of yield-based payment, Texel-sired lambs are likely to have an advantage over other terminal-sired lambs.

Gender differences in composition characteristics and meat quality

The review of gender comparisons revealed some consistent differences, however, there are also some traits where the direction and size of the difference were not clear. Consistent differences were:

- Dressing-out percentage: ram and cryptorchid lambs have significantly lower DO% than ewe or wether lambs.
- Carcass conformation: ewe lambs had better conformation scores than ram lambs (although no significant differences were found between ewe and wether lambs).
- Lean-meat yield: ram lambs had higher meat yield than ewe lambs (although no differences were found in a comparison of ewe and wether lambs).
- Fatness: ewe lambs were fatter, than ram lambs at a constant carcass weight.

Unclear differences were:

- Linear measurements: ram and cryptorchid lambs tended to be longer than ewe lambs at the same weight.
- *M. longissimus* dimensions: data adjustment often differed, but it seems that at a constant weight ewe lambs have larger *M. longissimus* relative to ram lambs.
- Cut distribution: this varied between studies and was not often considered.
- Meat quality: tended to be no significant differences, although one study did show decreasing tenderness, with increasing weight in ram and another showed higher pH for ram lambs.

Based on the above results, ram lambs have had, and will continue to have an advantage over ewe lambs, based on either the current or proposed new payment systems. The only potential draw back is possible poorer meat quality with ram lambs, although these were not consistent, they warrant further consideration.

Development of Quantitative Trait Loci technology

There have been milestones in the development of QTL technology which have led to the modern day ability to carry out detailed QTL searches. These milestones have included:

- The development of markers:
  Blood groups → Restriction fragment length polymorphisms → Polymerase chain reaction technique, allowing amplification of specific DNA sequences → Microsatellite marker → Single nucleotide polymorphisms.
- The development of linkage maps:
  - First using RFLP markers in tomatoes, now available for most species using a variety of markers and genes.
- The development of QTL experimental designs:
  - Can use inbred or segregating populations.
  - Can be based on full-sib, half-sib, or grandparental design.
  - Ideally diverse phenotype animals should be used.
- The development of QTL analysis techniques:
  - Maximum likelihood approaches vs least squares.
  - Single marker analysis vs Interval mapping.

- Permutation tests to estimate significance thresholds.
- Bootstrapping to estimate confidence intervals for QTL position.

Over the past decade the ability to carry out QTL searches has improved considerably. This trend is likely to continue, as more markers, experimental design and analysis techniques are developed. Using existing technology, however, the opportunity exists to carry out searches for most given populations and traits.

Existing QTL for sheep and cattle

A number of QTL affecting growth, composition and meat quality characteristics have been detected in both sheep and cattle, although only a few have been followed up in detail. For cattle these include:

- GDF8: Maps to Chr2 and is associated with increased muscling, specific mutations to the gene have been identified.
- Calpain 1 (CAPN1): Maps to Chr29 and is associated with tenderness, specific mutations to the gene have been identified.

For sheep these include:

- Callipyge: Maps to Chr18 and is associated with increased muscling, but decreased tenderness, the genes involved are currently being discovered.
- Rib-Eye Muscling Locus: Maps to Chr18 (distinct from Callipyge) and is associated with increased *M. longissimus* area, work is continuing to identify the gene involved.

A number of other QTL have been detected in cattle, however, there are limited reports of follow up work on the majority of these QTL.

Worldwide, three research groups are attempting to identify QTL which explain the increased muscling phenotype in the Texel breed of sheep. During the course of the study, associated with this literature review, one team provided strong evidence for a QTL in the region of GDF8 on Chr2. However, they did not consider muscle/fat distribution within the leg (one of the more valuable parts of the carcass), nor meat quality. Thus a number of questions remain unanswered regarding this QTL.

Chapter 3

A Comparison of the Carcass Composition and Meat Quality of Ram and Ewe Lambs

3.1 Introduction

New Zealand meat companies are currently looking to implement lamb payment to growers based on lean-meat yield, and through the use of DNA technology establishing traceability systems for monitoring quality. Under such systems a range of information will be required by breeders to make management decisions to optimise returns under yield-based payment systems and maximise meat quality. One important factor is the sex of lamb produced. Although farmers have limited control over sex ratio, information on sex effects may influence the preferential treatment of one sex over another if it is more profitable.

Factors that determine lean-meat yield (LMY%) are carcass fat percentage (FAT%) and muscle to bone ratio (MTB), as described by the function (LMY%) = (100-fat%) $((MTB)(MTB+1)^{-1})$ (Purchas, 1993). Differences in FAT% between lamb sexes are well documented with ewe lambs having a higher FAT%, however, MTB values also tend to be higher for ewe lambs in most studies (Carson et al., 1999; Wolf et al., 2001), although this difference is not always significant (Hopkins, 1996). Purchas et al. (2002) noted that estimation of MTB often has relied on a positive relationship with carcass shape. This is commonly assessed by *M. longissimus* muscle dimensions or subjective estimations of carcass muscularity. Carcass muscularity, anecdotally describes those meat animals with a blocky conformation (due either to muscle or fat), however, objectively muscularity has been defined as the ratio of muscle depth relative to skeletal dimensions (Purchas et al., 1991). Purchas et al. (2002) showed however, that at a common muscle plus bone weight, muscularity values were higher for carcasses of bulls than heifers, but when the data were adjusted for MTB as well, the values were generally higher for carcasses of heifers than bulls. Thus for the same muscularity values, the MTB and therefore LMY% the advantage of bulls over heifers was increased.

Differences between meat from ewe and ram lambs in quality characteristics including pH, colour and tenderness have generally not been found to be statistically significant (Corbett et al., 1973; Dransfield et al., 1990; Field et al., 1990; Ellis et al., 1997; Arsenos et al., 2002), the exception being Bickerstaffe et al. (2000). Considerable unpublished New Zealand meat processor industry data suggests significant differences between the sexes exist (John McEwan pers comm.).

The objective of the research reported here is to evaluate sex differences in carcass composition and meat quality characteristics and to investigate interrelationships between carcass composition and meat quality traits.

3.2 Materials and Methods

The lambs described were generated as part of a trial searching for Quantitative Trait Loci (QTL) for composition traits in the Texel.

3.2.1 Ethical Approval

Ethical approval for this experiment was obtained from the Animal Ethics Committee, Massey University, New Zealand. Protocol number 00/167.

3.2.2 Animals

The 543 lambs in this trial were sired by six sires on three properties. Property 1 ran one purebred Texel sire and two Texel/Coopworth-cross sires, Property 2 ran two purebred Texel sires and Property 3 ran one purebred Texel sire. The lambs were born in spring of 2001. All ewes and lambs within a property were run together on pasture from birth. Ram lambs were left entire. The mean age at weaning ranged between 68 days for Property 1 and 96 days on Property 3. Post weaning all lambs within a property were run together on pasture. Lambs from each property were assigned within sex to one of two slaughter groups which were balanced for sex. The first slaughter group for each property included ram and ewe lambs that were at least 38kg and 35kg liveweight respectively. The mean age at slaughter varied and was 143 days and 180 days, respectively, for slaughter groups one and two on Property 1; 156 and 193 days, respectively, for Property 2, whilst on Property 3 it was 176 and 251 days, respectively. This difference in slaughter date reflected different lamb growth rates on the three properties. Details of the numbers of animals per sub group are given in Table 3.1.

3.2.3 Slaughter Procedure

Lambs from Properties 1 and 2 were slaughtered at Alliance's Smithfield, Timaru, and lambs from Property 3 were slaughtered at the Takapau plant of Richmonds Co. All lambs were weighed off pasture two days prior to trucking to obtain a final liveweight. Transport to the processing plant took place the morning prior to slaughter, with the

lambs held overnight at the processing plant.

Table 3.1 Details of the number of lambs per different sub-class

	Property 1						Property 2				Property 3		
	Sire One		Sire Two		Sire Three		Sire Four		Sire Five		Sire Six		
Slaughter Group	1	2	1	2	1	2	1	2	1	2	3	4	**Total per Class**
Sex													
Ewe	4	41	8	36	9	36	19	26	20	25	11	33	**268**
Ram	4	42	8	37	9	35	19	26	20	25	23	27	**275**
Rearing Rank													
1	5	18	8	12	11	10				1	26	25	**116**
2	3	65	8	61	7	61	38	52	40	49	8	35	**427**
Dissector													
1	7	31	9	13	9	26	21	22	18	19	12	12	**199**
2		23		23	1	15	2	10	2	11	9	19	**115**
3		17	3	12		10	5	5	8	7	3	11	**81**
4											10	18	**28**
5	1	12	4	25	8	20	10	15	12	13			**120**
Total per Group	**8**	**83**	**16**	**73**	**18**	**71**	**38**	**52**	**40**	**50**	**34**	**60**	

Lambs were slaughtered and dressed under normal commercial conditions. Specifically for Properties 1 and 2, the animals were stunned at 400V for two seconds at one amp, this was followed by immobilization at 40V for 43 seconds, no post-dressing high voltage was used. These carcasses were chilled at 9°C for 8 hours, after which time they were taken down to 0.5°C prior to cutting. For Property 3, the animals were stunned at 400V for one second at one amp, this was followed by immobilization at 70V for 20 seconds, post-dressing the carcass were given 1110V for 92 seconds at two amps. These carcasses were chilled to less than 13°C within eight hours, and less than 7°C within 18 hours and less than 1°C within 24 hours.

Measurements of carcass dimensions (Table 3.2) were taken by trial personnel on the day of slaughter. They were measured on the cooling floor after electrical stimulation. GR was measured by works personnel. On the day following slaughter the carcasses were cut up and the right leg and loin (Table 3.2) of each carcass were packaged in plastic bags and frozen and stored for 1-5 months at -18°C.

Table 3.2 Abbreviations and description of measurements made

Description	Abbreviation
Measurements Made at the Processing plant	
Dressed cold carcass weight (kg)	CCW
Carcass length from between the hind legs to the front of the neck (cm)	CLNG
Forequarter width at the widest part of the forequarter (cm)	WF
The minimum width of the thorax (behind the shoulders) (cm)	WTH
The maximum width at the gigots (cm)	G
Soft tissue depth, measured at the 12th rib, 110mm from the mid-line (mm)	GR
Weight of chump on, shank on, aitch bone in (long leg) (g)	LG
Weight of no-rib, short loin with flaps removed (g)	LN
Maximum width of LL at cranial end of loin (mm)	A
Maximum depth of LL at right angles to A, at cranial end of loin (mm)	B
Subcutaneous fat over B (mm)	C
M. longissimus muscle area from tracing of cranial end of loin (cm^2)	EMA
Measurements Made During Dissection	
Trimmed leg weight (g)	TLG
M. semimembranosus (g)	SM
M. semitendinosus (g)	ST
M. biceps femoris (g)	BF
M. adductor femoris (g)	AD
M. quadriceps femoris (g)	QD
M. gluteus medius (g)	GM
M. longissimus et lumborum	LL
Leg muscle except the above six muscles (g)	MST
Five muscle weight (BF, ST, SM, AD and QD)	$5MS_{WT}$
Six muscle weight (BF, ST, SM, AD, QD and GM)	$6MS_{WT}$
Leg subcutaneous fat (g)	SCF
Leg intermuscular fat (g)	IMF
Femur bone (g)	FM
Pelvic bone (g)	PV
Leg bone (includes femur and pelvic) (g)	TLBN
Femur length (mm)	FMLNG
Meat Quality Measurements	
Warner Bratzler peak force *M. semimembranosus* (kg)	WBPKSM
Warner Bratzler yield force *M. semimembranosus* (kg)	WBYFSM
Warner Bratzler peak force *M. longissimus* (kg)	WBPKLL
Warner Bratzler yield force *M. longissimus* (kg)	WBYFLL
Cooking loss *M. semimembranosus* (%)	CKSM
Cooking loss *M. longissimus* (%)	CKLL
Sarcomere length *M. semimembranosus* (μm)	SLSM
Sarcomere length *M. longissimus* (μm)	SLLL
Colour "L*" *M. semimembranosus*	CLSM
Colour "a*" *M. semimembranosus*	CASM
Colour "b*" *M. semimembranosus*	CBSM
Colour "L*" *M. longissimus*	CLLL
Colour "a*" *M. longissimus*	CALL
Colour "b*" *M. longissimus*	CBLL
Ultimate pH *M. semimembranosus*	PHSM
Ultimate pH *M. longissimus*	PHLL
Other Traits	
Leg muscularity (see definition 3.2.7)	LGMUSC
Femur muscle to bone (see definition 3.2.7)	MTB
Percentage fat in leg	LGFT%
Percentage lean in leg	LGMS%

3.2.4 Dissection Procedure

Frozen legs and loins were thawed at ambient temperature (10-20°C) for approximately 18 hours prior to dissection. Dissection was carried out by a team of five people and legs were randomly allocated to dissectors on the day. Prior to dissection the legs were trimmed to a consistent level by removing loose fat inside of the pelvic bone (to the level of the back bone), and removing the loose flap on the cranial edge, with a vertical cut back to the edge of *M. tensor fasciae latae* and *M. quadriceps femoris* muscle. Dissection of the leg involved removing the muscles, fat depots and bones described in Table 3.2. Other components such as tendons, blood vessels and lymph nodes were removed and weighed separately when visible (often within fat depot and so not detected). Prior to dissection of the loin, measurements of *M. longissimus* (A, B, EMA (Table 3.2)) and fat depth C were taken. Only partial dissection of the loin was carried out with removal of subcutaneous fat and *M. longissimus* (Table 3.2).

3.2.5 Preparation of Meat Samples for Quality Analysis

For each SM and LL muscle starting from the proximal end an initial transverse cut was made to provide a flat face. From there:

- A 10mm slice was cut and placed in a labelled bag and frozen for subsequent colour and pH measurements.
- Three 25mm slices were cut, and placed in a plastic bag for cooking (outlined below).
- A small slither of muscle parallel to the fibres was removed and placed in a test-tube that was capped and placed in a chiller for subsequent measurement of sarcomere length.

3.2.6 Meat Quality Analysis

Warner-Bratzler shear force

The following measurements were carried out for both the SM and LL samples using the procedure described by Purchas and Aungsupakorn, (1993):

- On the day of dissection the slices identified above were weighed (excluding the weight of the bag).
- The samples were then cooked by suspending the bag so that the samples were immersed in a preheated water bath at 70°C for 90 minutes.

- After cooking any liquid in the bag was poured out.
- The samples were placed in the chiller for cooling at 2-4°C overnight (16 hours).
- On the following day the samples were reweighed and cooking loss as a percentage of initial weight was calculated.
- Six 13x13 mm cores were cut parallel to the orientation of the muscle fibres.
- The Warner-Bratzler machine, with a square blade attached was used to shear each core twice at approximately one third and two thirds of its length.
- The following measurements were recorded:-
- Peak Force (PF)
- Mean (MN)
    - The mean value of the, on average, 447 Force values collected during each shear.
- Initial yield force (YF)
    - The first break (peak or shoulder) to form in the force-deformation curve.
- The difference between the peak force and the yield force was estimated (PF - YF)

Cooking loss

Cooking loss (CK) was estimated for both the SM and LL

Using the weights recorded pre and post cooking, described above:

$$\text{Cooking loss}\,(\%) = \frac{\text{Before Cooking Weight - After Cooking Weight}}{\text{Before Cooking Weight}} \times 100$$

Sarcomere length

Sarcomere length was determined the day following dissection for both the SM and LL using a method similar to the laser diffraction method described by Cross et al. (1980-81):

- A small sliver of muscle was teased out on a microscope slide using scalpel blades to provide a thin layer with fibres approximately parallel (~1x2x5mm).
- Several drops of a buffered sucrose solution were added to the slide (0.25M sucrose, 0.05M Tris, 1mM EDTA adjusted to a pH of 7.6).
- A cover slip was placed on and pressure steadily applied for 15-20 seconds with

some movement to flatten out the sample.

- The slide was then placed under a helium-neon laser beam to display an array of diffraction bands on a screen, 100 mm below the sample..
- 12 measurements were made for each muscle.
- The average of these values was calculated and converted using the following formula

$$SL\ (\mu m) = 0.6328\left[\frac{\sqrt{(D/10/2)^2+100}}{(D/10/2)}\right]$$

Where D=distance between the first-order diffraction bands in mm

Colour

Colour was determined for SM and LL samples as follows:

- The samples were removed from the freezer approximately 18 hours prior to measurement and placed in a chiller to thaw (0-1°C).
- Once thawed the meat samples were sliced in half along the grain to expose a fresh surface and were exposed to air in the chiller for 30 minutes.
- A Minolta ChromaMeter was calibrated to a white standard, using the L*, a*, b* scale.
- Measurements were taken through a clear petri dish pushed flush against the meat to provide a contact surface for the ChromaMeter.
- L* (lightness), a* (redness) and b* (yellowness) were recorded at two points of the muscle surface and the values averaged.

pH

pH was determined for both the SM and LL samples. These measurements were taken at the same time that colour measurements were made.

- The meat samples once used above for the colour were turned over to reveal muscle that had not been exposed to the air.
- A sensorex spear tip pH probe was calibrated to pH 4 and 7 using buffer standards.
- The probe was inserted into the muscle samples and held until a steady pH reading was obtained.

- pH was recorded on two points of the muscle surface.

3.2.7 Muscularity and Muscle to Bone Ratios

MUSC was calculated as described by Purchas et al. (1991) using the weight of the five main muscles ($5M_{WT}$) surrounding the femur (BF, ST, SM, AD and QD) and FMLNG as follows:

$$MUSC = \left[\frac{\sqrt{[(5MS_{WT})/(FMLNG/10)]}}{(FMLNG/10)} \right]$$

Femur MTB (MTB) was also calculated by dividing $5M_{WT}$ by the FMWT. Leg MTB (LMTB) was calculated by dividing total leg muscle weight by the weight of TLBN. For statistical analysis purposes described below the weight of the leg (LGWT) was taken as sum of the component parts (LGWT was less than TLGWT by an average of 17.1g or 0.6%). The percentage fat in the leg (%FAT) was obtained by expressing the weight of IMF plus SCF as a percentage of LGWT.

3.2.8 Statistical Analysis

Carcass composition

Data were analysed using the General Linear Model procedure of SAS (SAS, 1991). For carcass traits the fixed effects were sex (ewe or ram), rearing rank (RR as single or twin) and group (GRP – (12 groups with two slaughter lots within each sire; property was completely confounded with sire)), CCW was fitted as a covariate. For the dissection traits the fixed effects were sex, RR, dissector and GRP. LGWT or 6 muscle weight ($6MS_{WT}$: BF, ST, SM, AD, QD and GM) were used as covariates for the leg traits and CCW for LL weight. The fixed effects fitted for LGMUSC and ratio traits were the same as those for the dissection traits, LGWT was fitted as the covariate. An additional model was fitted for LGMUSC which included MTB and LGWT as covariates to determine whether the relationship between LGMUSC and MTB differed between the sex groups. For each analysis all interactions were fitted (fixed effects and covariates), followed by backwards elimination of non-significant effects. No interactions reached the 5% threshold, and therefore no interactions were included in the final models.

Meat quality

Meat quality data were analysed using the General Linear Model procedure of SAS (SAS, 1991). Two models were fitted, both included the fixed effects of sex, RR and GRP, the first model did not include covariates, whilst the second included the covariates of pH and pH^2.

To determine whether or not differences in the meat quality results existed for the two muscles used (SM and LL), pair-wise t-tests were applied within sex using the paired option in the T-Test procedure of SAS (SAS, 1991).

Correlations between carcass composition and meat quality traits

To determine whether or not correlations existed between carcass composition data and meat quality data, the correlation procedure of SAS (SAS, 1991) was used. The data were first analysed using the GLM procedure. The models used were the same as those previously described and took account of the fixed effects and covariates. The resulting residuals were stored and then used in the correlation procedure, within sex.

3.3 Results

The main focus of this analysis was to determine sex differences for carcass composition and meat quality traits. The results of other fixed effects (RR, GRP and dissector) will not be presented. Briefly however, rearing rank was only significant for fat traits with singletons having larger values than twins. Group had a significant effect on carcass weight, however, inclusion of carcass weight or leg weight as a covariate for other traits removed most group effects. Dissector was significant for all dissection traits ($P<0.05$).

3.3.1 Carcass Composition Traits

The differences between sexes for carcass traits adjusted to a common carcass weight are presented in Table 3.3. Carcass weight fitted as a covariate was significant ($P<0.001$) for all traits with a positive relationship in each case. Sex was significant for most carcass traits. For a given carcass weight, ram lambs were longer, and had greater measurements of A, while ewe lambs were wider at WTH, wider at G, had greater GR and C measurements, deeper B measurements, larger EMA and heavier legs.

Table 3.3 Least-squares means for lamb carcass characteristics for ewe and ram lambs. All variables except carcass weight are adjusted for carcass weight differences

	Sex			Carcass Weight		R^2%
	Ewe	Ram	Effect[a]	Effect [a]	Estimate[b]	(RSD)
Number of animals	269	275				
Carcass weight (kg)	16.7	17.6	***			33 (1.2)
Dressing-out %	42.0	40.0	***	***	0.90	62 (1.6)
Carcass length (cm)	80.3	81.0	***	***	1.00	50 (1.7)
Maximum width of shoulders (cm)	17.3	17.3	NS	***	0.33	48 (0.7)
Minimum width of shoulders (cm)	16.7	16.3	***	***	0.35	47 (0.6)
Maximum width of gigots (cm)	22.5	22.2	***	***	0.35	61 (0.5)
M. longissimus width (A) (mm)	55.2	58.1	***	***	0.73	40 (3.2)
M. longissimus depth (B) (mm)	27.2	26.7	*	***	0.68	33 (2.1)
M. longissimus area (cm^2)	12.2	11.8	***	***	0.40	44 (12.4)
Fat depth C (mm)	3.0	2.1	***	***	0.30	28 (1.0)
Fat depth at 12th rib (GR) (mm)	8.0	5.7	***	***	0.68	59 (1.8)
Leg weight (g)	2,899	2,864	**	***	159.59	86 (89.5)

[a] NS: non-significant; P>0.05; * P<0.05; ** P<0.01; *** P<0.001
[b] Regression coefficient

3.3.2 Leg Composition Traits

Differences between sexes for leg traits adjusted to a common LGWT (Table 3.4) indicated that all traits increased with increasing LGWT (P<0.001). Sex was significant for the all leg traits, except the $5MS_{WT}$ and LL. For a given leg weight ram lambs had heavier total muscle weight FM, PV, TLBN and longer femurs, whilst ewe lambs had heavier IMF, SCF and total fat weight.

Table 3.4 Least squares means for lamb leg traits observing differences between sexes

	Sex			Leg Weight[a]		R^2%
	Ewe	Ram	Effect[b]	Effect[b]	Estimate[d]	(RSD)
M. longissimus weight[a] (g)	200.0	202.3	NS	***	10.10	64 (15.91)
Five muscle weight[c] (g)	1,069.7	1,067.1	NS	***	0.32	91 (33.18)
Total muscle in leg weight (g)	1,889.3	1,901.5	**	***	0.59	96 (39.34)
Leg intermuscular fat weight (g)	146.3	136.5	**	NS	0.02	40 (23.22)
Leg subcutaneous fat weight (g)	225.4	181.1	**	***	0.08	44 (37.19)
Total fat weight in leg (g)	371.7	317.5	**	***	0.10	46 (49.00)
Femur bone weight (g)	144.1	156.1	**	***	0.03	66 (9.33)
Total bone in leg (g)	406.2	432.7	**	***	0.09	66 (24.91)
Femur bone length (mm)	171.0	172.7	**	***	0.01	49 (3.80)

[a] leg weight fitted as a covariate, except for *M. longissimus* for which carcass weight was fitted as a covariate
[b] NS: non-significant; P>0.05; * P<0.05; ** P<0.01; *** P<0.001
[c] weight of *M. semimembranosus*, *M. semitendinosus*, *M. adductor*, *M. quadriceps* and *M. biceps femoris* muscles
[d] Regression coefficient

Differences between sexes for leg muscles adjusted to a common $6MS_{WT}$ are presented in Table 3.5. $6MS_{WT}$ fitted as a covariate was significant (P<0.001) for all these traits, each of which was positively correlated with $6MS_{WT}$. Sex was significant for all but the QD. For a given $6MS_{WT}$ ewe lambs had heavier BF and AD weights and ram lambs had heavier SM and ST weights.

Table 3.5 Least-squares means for weight-adjusted lamb leg muscles adjusted for leg six-muscle weight as measures of muscle distribution within the leg

	Sex			6 Muscle Weight[a]		R^2%
	Ewe	Ram	Effect[b]	Effect[b]	Estimate[c]	(RSD)
M. semimembranosus (g)	249.8	252.7	**	***	0.2	86 (10.6)
M. semitendinosus (g)	89.1	93.5	***	***	0.1	77 (6.3)
M. gluteus medius (g)	166.9	166.3	NS	***	0.2	73 (10.8)
M. biceps femoris (g)	246.0	243.8	*	***	0.2	89 (9.9)
M. quadriceps femoris (g)	363.6	363.1	NS	***	0.3	88 (13.4)
M. adductor (g)	120.6	116.4	**	***	0.1	69 (7.8)

[a] 6 muscle weight fitted as a covariate (*M. semimembranosus*, *M. semitendinosus*, *M. adductor*, *M. quadriceps*, *M. gluteus medius* and *M. biceps femoris*)
[b] NS P>0.05; * P<0.05; ** P<0.01; *** P<0.001
[c] Regression coefficient

3.3.3 Leg Characteristics

Differences between sexes for leg characteristics other than weights and lengths adjusted to a common LGWT (Table 3.6) were significant (P<0.001) for all leg characteristics. Ewe lambs had higher MTB, FMTB, LGFT% and LGMUSC for a given LGWT. All traits were positively correlated with leg weight, and leg weight fitted as a covariate was always significant.

Covariates were significant (P<0.001) for LGMUSC adjusted to both a common LGWT and MTB. Leg muscularity was positively correlated with both leg weight and MTB and for a given LGWT and MTB ram lambs had higher LGMUSC values whereas before adjustment to a constant MTB, LGMUSC values were lower for rams.

Table 3.6 Least-squares means for weight-adjusted leg characteristics in the form of percentages or ratios for ewe and ram lambs

	Sex			Leg Weight[a]		Femur		R%
	Ewe	Ram	Effect[b]	Effect[b]	Estimate[c]	Effect[b]	Estimate[c]	(RSD)
Leg fat percent	11.2	9.6	***	***	0.0001			46 (1.48)
Leg muscle:bone	4.7	4.4	***	***	0.0004			53 (0.33)
Femur muscle:bone	7.5	6.9	***	***	0.0007			63 (0.51)
Leg muscularity	0.463	0.455	***	***	3.3E-05			53 (0.02)
Leg muscularity adj for MTB	0.458	0.463	***	***	1.6E-05	***	0.022	74 (0.01)

[a] leg weight fitted as a covariate
[b] NS P>0.05; * P<0.05; ** P<0.01; *** P<0.001
[c] Regression coefficient

3.3.4 Meat Quality

The differences between sexes for meat quality characteristics for two muscles are given in Table 3.7. Sex was significant for most traits with the exception of CLSM, SLSM, SLLL and CKLL. For both muscles (SM and LL) pH was higher for the ram lambs, as were all Warner-Bratzler measures of tenderness. For both muscles (except CLSM) all

colour parameters were higher in ewe lambs than ram lambs. Cooking loss for SM muscle was higher in the ram lambs.

The recommended threshold for pH values is 5.8, 3% and 10% of ewe and ram lamb values for SM exceed this threshold, with the corresponding values for LL 4% and 32%. Based on the "New Zealand Beef and Lamb Quality Mark Standards" the threshold for mean shear force is 12kg (using Warner-Bratzler measurements). Although no Warner-Bratzler mean shear force values exceeded 12kg, for 22% and 41% of SM samples from ewe and ram lambs had Warner-Bratzler peak values greater than 12kg.

Table 3.7 Least-squares means for meat quality characteristics for meat from *M. semimembranosus* and *M. longissimus* muscles of ewe and ram lambs, observing differences between sexes

	M. semimembranosus				*M. longissimus*			
	Sex			R%	Sex			R%
	Ewe	Ram	Effect[a]	(RSD)	Ewe	Ram	Effect[a]	(RSD)
pH	5.6	5.7	***	13 (0.10)	5.6	5.7	***	32 (0.18)
Colour "L*" (Lightness)	31.9	31.8	NS	60 (1.70)	33.7	33.2	***	57 (2.11)
Colour "a*" (Redness)	15.0	14.2	***	51 (1.18)	14.8	13.8	***	39 (1.40)
Colour "b*" (Yellowness)	6.5	6.1	***	39 (0.72)	6.6	5.9	***	35 (0.97)
Cooking loss (%)	36.4	37.6	***	31 (1.21)	30.6	30.4	NS	19 (2.02)
Sarcomere length (μm)	1.9	1.9	NS	8 (0.15)	1.8	1.8	NS	15 (0.09)
Warner-Bratzler peak force (kg)	9.9	11.2	***	23 (2.83)	5.9	6.9	***	25 (1.68)
Warner-Bratzler yield force (kg)	7.6	8.4	***	25 (2.45)	4.9	5.5	***	25 (1.15)
Peak force – yield force	2.4	2.7	***	18 (0.83)	1.0	1.4	***	20 (0.81)

[a] NS P>0.05; * P<0.05; ** P<0.01; *** P<0.001

The differences between sexes for meat quality characteristics adjusted to a common pH are presented in Table 3.8. Fitting pH as a covariate was significant (P<0.001) for the all traits with the exception of CLSM and CKSM, whilst fitting pH^2 as a covariate was significant (P<0.001) for CLSM, CASM, and all LL traits and SLL which was significant at the 5% level. Fitting pH and pH^2 as covariates did not alter the results observed in Table 3.7, with the exception of CKLL. For CKLL, when unadjusted for pH there were no significant difference between the sexes, with if anything a slightly higher value for ewe lambs. However, after adjustment for pH the difference between the sexes was significant with ram lambs having a higher cooking loss.

The differences between the SM and LL by sex for meat quality characteristics are in Table 3.9. The difference between the two muscles was significant for all traits in the ram (P<0.001) and ewe lambs, although in the ewe lambs CA difference was only significant at the 1% level and pH and CB differences were only significant at the 5%

level. Excluding those traits that were not highly significant the trend remains the same between the ewe and ram lambs. For CA, CK, SL, and all Warner Bratzler all measurements were higher in the SM. Colour "L*" was the only the measurement lower in the SM than the LL. For those traits not significantly different between muscles in both sexes, there were inconsistent differences between the sexes in the direction of the differences. For pH the muscle with a higher pH in the ewe lambs was the SM, whilst for the ram lambs it was LL, and for CB, the muscle with a higher measurement in the ewe lambs was the LL, whilst for the ram lambs it was the SM.

Table 3.8 Least-squares means for lamb quality characteristics for two muscles from ewe and ram lambs after adjustment for differences in ultimate pH[a]

	M. semimembranosus			
	Sex			R%
	Ewe	Ram	Effect[b]	(RSD)
Colour "L*"	31.9	31.8	NS	61 (1.69)
Colour "a*"	14.9	14.3	***	60 (1.07)
Cooking loss (%)	36.4	37.6	***	34 (1.19)
Sarcomere length (μm)	1.9	1.9	NS	11 (0.15)
Warner Bratzler peak force (kg)	10.0	11.0	***	31 (2.70)
	M. longissimus			
	Sex			R%
	Ewe	Ram	Effect[b]	(RSD)
Colour "L*"	33.4	33.3	NS	64 (1.92)
Colour "a*"	14.5	13.9	***	53 (1.21)
Cooking loss (%)	30.1	30.7	***	53 (1.52)
Sarcomere length (μm)	1.8	1.8	**	19 (0.09)
Warner Bratzler peak force (kg)	6.0	6.8	***	30 (1.61)

[a] pH and pH^2 fitted as covariates
[b] NS P>0.05; * P<0.05; ** P<0.01; *** P<0.001

Table 3.9 Least-Squares means (± se) for the difference between *M. semimembranosus* (SM) and *M. longissimus* (LL) muscles for meat quality characteristics

	Ewe		Ram	
	Difference (SM-LL) ± stderr[a]	Significance	Difference (SM-LL) ± stderr[a]	Significance
pH	0.01 ± 0.004	*	-0.10 ± 0.013	***
Colour "L*"	-1.81 ± 0.081	***	-1.23 ± 0.115	***
Colour "a*"	0.22 ± 0.076	**	0.62 ± 0.087	***
Colour "b*"	-0.14 ± 0.047	*	0.34 ± 0.071	***
Cooking loss "%"	0.10 ± 0.011	***	0.13 ± 0.011	***
Sarcomere length (μm)	6.03 ± 0.089	***	7.67 ± 0.155	***
Warner Bratzler peak force (kg)	2.47 ± 0.048	***	2.89 ± 0.057	***
Warner Bratzler yield force (kg)	1.09 ± 0.042	***	1.54 ± 0.061	***
Peak force - yield force (kg)	1.37 ± 0.052	***	1.34 ± 0.063	***

[a] Paired t-test comparison of *M. semimembranosus* and *M. longissimus*.
[b] NS P>0.05; * P<0.05; ** P<0.01; *** P<0.001

Correlations between the leg characteristics and meat quality are presented by sex in Table 3.10. Any correlations between 0.12 and 0.16 are significant at the 5% level,

correlations between 0.16 and 0.20 are significant at the 1% level and correlations greater than 0.2 are significant at the 0.1% level. No correlations were above 0.3, and the results were not consistent between traits, sexes, and muscles. A negative correlation existed between the muscle ratios and pH for both muscles in the ram lambs, this effect was not seen in the ewe lambs. A positive correlation existed between the muscle ratios and cooking loss for the SM in both sexes, but not in LL. A positive correlation existed between sarcomere length and the muscle traits for the LL in ram lambs, whilst in the ewe lambs a negative correlation existed between sarcomere length and the muscle traits for the SM. WBPF and PF-YF were positively correlated with LMTB in LL of both sexes, and MTB in the ram lambs.

Table 3.10 Simple linear correlations between selected composition characteristics and meat quality traits after adjustment for leg weight for ewe and ram lambs. Shaded numbers are co-efficients which are significant (P<0.01)

	Ewe							
	M. semimembranosus				*M. longissimus*			
	Leg MTB	Femur MTB	Leg Musc	Leg Fat%	Leg MTB	Femur MTB	Leg Musc	Leg Fat%
pH	0.00	-0.05	-0.04	0.08	0.03	0.01	0.00	-0.01
Colour "L*"	0.07	-0.01	-0.01	0.07	0.08	-0.02	0.01	0.12
Colour "a*"	-0.15	-0.05	-0.07	0.16	-0.03	0.04	-0.01	0.06
Colour "b*"	-0.11	-0.04	0.00	0.10	-0.06	-0.02	-0.04	0.14
Cooking loss "%"	0.14	0.17	0.13	0.07	-0.07	-0.09	-0.10	0.12
Sarcomere length (μm)	-0.12	-0.14	-0.06	-0.17	0.06	0.03	0.05	-0.07
Warner Bratzler peak force (kg)	0.10	0.00	-0.01	-0.04	0.12	0.03	0.07	-0.12
Warner Bratzler yield force (kg)	0.10	0.00	-0.03	-0.02	0.07	0.02	0.05	-0.11
Peak force - yield force (kg)	0.07	0.01	0.03	-0.07	0.15	0.05	0.08	-0.10

	Ram							
	M. semimembranosus				*M. longissimus*			
	Leg MTB	Femur MTB	Leg Musc	Leg Fat%	Leg MTB	Femur MTB	Leg Musc	Leg Fat%
pH	-0.17	-0.17	-0.18	-0.01	-0.11	-0.13	-0.10	0.02
Colour "L*"	-0.01	-0.02	0.08	-0.01	0.00	0.00	0.06	0.08
Colour "a*"	0.03	0.02	0.10	0.12	0.04	0.06	0.02	0.03
Colour "b*"	0.07	0.08	0.15	0.02	0.03	0.08	0.05	0.07
Cooking loss "%"	0.19	0.18	0.22	-0.10	0.00	-0.06	0.04	-0.03
Sarcomere length (μm)	0.05	0.05	0.14	-0.08	0.22	0.20	0.18	-0.05
Warner Bratzler peak force (kg)	0.03	0.00	0.00	0.06	0.13	0.12	0.08	0.01
Warner Bratzler yield force (kg)	0.03	0.00	0.01	0.07	0.08	0.08	0.05	0.05
Peak force – yield force (kg)	0.03	0.00	-0.04	-0.01	0.15	0.13	0.08	-0.03

3.4 Discussion

The introduction of yield-based payment systems will change the "ideal" lamb, compared to the current system. Until the techniques for determining yield are developed by the industry, exactly what factors will influence the end price of a carcass are still unclear. However, it is likely that there will be increased returns for carcasses that have a higher proportion of their weight in the higher price cuts, and within those cuts more muscle and less fat (better cut yield). Ideally a trial would be conducted using these techniques to assess differences between sexes, however, given the techniques are still only in developmental phase this is not currently possible.

As discussed by Purchas (1993) LMY% is determined by FAT% and MTB, although as further discussed by Purchas et al. (2002) this approach relies on a relationship between MTB and carcass shape, a relationship which is not always consistent.

Only limited literature was available to compare the results against, as most gender comparison studies have been made either between ewes and wethers (castrated males) or between rams and wethers. Wethers because of their lack of primary sex hormone have a different growth path and final body composition relative to either of the entire sexes and it is therefore not appropriate to include comparisons made only to them. It is, however, appropriate to draw some conclusions from comparisons between cryptorchids and ewes, as cryptorchids still produce testosterone which is the key to the sex differences. Specifically the work of Hopkins and Hopkins et al. (1996) referenced below involved cryptorchid lambs as opposed to ram lambs in comparison with ewe lambs.

Rams have higher mature weights than ewes, but it is important to note that even at their mature weights, the composition of mature ewes and rams has been shown to differ (Thompson (1983) in Butterfield (1988) and Thonney et al. (1987)). This is an important consideration as work previous to Thompson suggested there to be no differences in composition of mature ewes and rams for their given percentage of maturity (McClelland et al., 1976). Thus even adjusting to the same level of maturity may not remove any differences seen. Irrespective of this, however, the reality is that meat companies ultimately have "optimum" weight ranges, which were achieved in this current study and so the differences in the degree of maturity reached by ewe and ram

lambs at the same weight, although they may aid in explaining the results seen, are of limited importance for providing recommendations and guidelines to producers as to ways to optimise returns.

3.4.1 Dressing-Out Percentage

Dressing-out percentage although not having a direct effect on LMY% (when LMY% is estimated on the basis of carcass weight) is an important indicator for producers as to whether or not their carcasses are going to meet desired weight ranges, a criterion likely to remain even under yield-based payment. Its importance lies in the fact that small changes in DO% can have large effects on the weight of the resulting carcass. In this study the DO% of the ewe lambs was significantly higher than that of the ram lambs, although the difference was only two percentage points, for a 40kg LW animal this equates to a 0.8kg difference in carcass weight. A number of authors have found a similar trend (Mahgoub & Lodge, 1998; Vergara et al., 1999; Wolf et al., 2001; Wellington et al., 2003), although Everitt and Jury (1966b) found no significant differences between the sexes. A possible explanation for the differences observed is that the ram lambs involved in this trial would have attained puberty and thus their testicles would have been developed (albeit not fully) resulting in, for any given liveweight, increased non-carcass component weight. However, the results of Gaili, (1992) when expressed as a proportion of empty body weight showed there to be no significant sex differences in the weight of any on the non-carcass components, however, the lambs Gaili's study were only at 30kg and perhaps not developing sexually.

3.4.2 Carcass Conformation Traits

Differences in body shape as assessed by weight-adjusted measures of length and width at three anatomical positions showed that ram lambs were longer and narrower than the ewe lambs (with the exception of WF). A number of other studies have also found ram lambs to be longer than ewe lambs (Hopkins et al. 1996). This is consistent with the rams ultimately having a larger frame (due to heavier weight) and so at any given weight will be longer.

3.4.3 M. longissimus Traits

Differences in *M. longissimus* dimensions were detected in this study, with ram lambs

having wider but shallower muscles and a reduced overall area compared to ewe lambs at the same weight. Despite this reduced area, no significant differences were detected between the weight of the LL muscle from the two sexes. A number of studies have found the same trends as observed in this current study (Purchas & Wilkin, 1995; Wolf et al., 2001). There have, however, been studies where differences have not been found when adjusting for carcass weight (Everitt and Jury 1996b; Hopkins et al. 1996)

3.4.4 Fat Traits

Measures of fat depths and fat weights were made in this study. For all measures of fatness assessed at a common weight the ewe lambs were fatter than the ram lambs. This is consistent with the results of other studies that have compared the sexes at a constant weight (Everitt & Jury, 1966b; Wood et al., 1980; Butterfield et al., 1984b; McClure et al., 1994; Afonso & Thompson, 1996b; Hopkins et al., 1996; Teixeira et al., 1996; Stanford et al., 2001; Wolf et al., 2001; Díaz et al., 2003; Wellington et al., 2003). In the work of Wolf et al. (2001), although they found that overall fat levels were higher in ewe lambs, they did not find differences between sexes in the intermuscular fat depot, whilst Afonso and Thompson (1996b) (based on CAT-scan estimates) found rams to have higher intermuscular fat at the same weight. Conversely Gaili (1992), reported no differences in the partitioning of subcutaneous and intermuscular fat between ram and ewe lambs. That the ewe lambs had a higher proportion of fat compared to the ram lambs was expected, given that in addition to the ewe lambs being more mature at the set carcass weights, mature ewes compared with rams have a higher proportion of fat in their body (Thompson (1983) in Butterfield et al. (1988)). Given that the carcass weights achieved roughly fall into the most desired range for meat companies, it means that if yield-based payment takes into account fat level, which it is likely to do, ewe lambs will be penalised relative to ram lambs.

3.4.5 Cut Distribution

A number of other studies have compared the distribution of cuts in a carcass between sexes, but in the current study the only cut that was weighed was the leg. The weight of the leg for a common carcass weight was higher for the ewe lambs than the ram lambs. Similar results have been shown by Jones et al. (2002), who also showed there to be no difference between sexes in the weight of the loin or shoulder but for rams to have a higher proportion of their weight in the best end neck and less in the leg, with similar

reports by Purchas and Wilkin (1995). The work of Butler-Hogg and Brown (1986) and Wolf et al. (2001) considered muscle (as opposed to whole cuts) distribution throughout the carcass, and also showed ewe lambs to have more of their weight in the leg region and less in the shoulder compared to ram lambs. However, the work of Seebeck (1968) found no significant differences between the sexes in the cut proportions throughout the carcass. That the ewe lambs had a higher proportion of weight in the leg, is more likely to do with the ram lambs having a lesser proportion of weight in the leg because of their increased weight in the forequarter, an attribute of rams related to masculization (Lohse, 1973; Butterfield, 1988). This provides the ewe lambs with an advantage over the ram lambs if the proposed yield-based payments system is calculated per cut, a direction that seems likely, in that in comparison with the shoulder and neck, the leg (when boned) is a more valuable cut and overall contributes more to the total value of the carcass than other cuts (Johnson et al., 2002).

3.4.6 Muscle Traits

Several muscle measurements were made within the leg. At the same trimmed leg weight the $5MS_{WT}$ was not significantly different between the sexes, but the amount of total muscle in the leg was significantly heavier in the ram lambs, although the absolute difference was only small. All other studies have shown ram lambs to have more muscle than ewe lambs for a given carcass weight (Wood et al., 1980; Butler-Hogg & Brown, 1986; Afonso & Thompson, 1996a; Hopkins et al., 1996; Teixeira et al., 1996; Carson et al., 1999; Wolf et al., 2001; Jones et al., 2002; Díaz et al., 2003). This would lead to ram lambs being rewarded relative to ewe lambs at the same weight under yield-based payment systems.

Depending on the system used to implement yield-based payment, there is the possibility that it could be to the level of individual muscles. As many overseas clients seek an "easy preparation" product, there may be a move away from cuts such as the whole leg and a move to packaging of individual muscles from the leg. When comparing the distribution of muscles within the leg between the sexes in this trial, there were no differences in the weight of the GM or QD muscles, but the SM and ST muscles were significantly heavier in the ram lambs, and the BF and AD muscles were significantly heavier in the ewe lambs. Other studies considering the distribution of muscles within the leg, have shown inconsistent results (Taylor et al., 1980; Butler-

Hogg & Brown, 1986; Wolf et al., 2001). Taylor et al. (1980) showed that the ST, SM, QD and GM (expressed as a percent of total muscle) were heavier in ewes, with the BF heavier in the rams; Butler-Hogg and Brown (1986) showed no difference in the ST, SM, or GM, but showed the AD to be heavier in ewe lambs and the BF to be heavier in ram lambs, whilst Wolf et al. (2001) showed the SM and BF to be heavier in the ewes.

Given the inconsistencies of these results, when compared with other studies further interpretation is limited. Different muscles in the leg are involved with different aspects of locomotion, but as both sexes, in the studies described, were run together they would have been exposed to the same terrain, and so if terrain differences were to result in specific development of a particular muscle it should have been consistent across both sexes. Differing distribution of muscles in the leg between sexes will become important if payment does occur at the muscle level, because muscles in the leg are likely to vary greatly in their value. For instance the BF and QD are likely to be less valuable because of the increased level of connective tissue found in these muscles relative to others in the leg, whilst the SM and ST are likely to be worth the most because of their uniformity. If this scenario were to unfold, then the ram lambs would have an advantage in that more of their weight is in the SM and ST specifically, with more of the ewe lambs weight in the BF. However, whether or not the differences would be commercially significant is unclear given the small difference in weight between the sexes.

3.4.7 Bone Traits

Differences between the sexes in leg bone weight and femur length for a given leg weight were significant in this study, with ram lambs having heavier and longer bones. Wolf et al. (2001) also showed ram lambs to have a significantly higher proportion of bone, whilst Purchas and Wilkin (1995) found no difference. Given the larger resulting weight and frame of ram lambs, that they have heavier bones is not surprising in that they require extra structure and strength to support their increased muscle and fat masses. This, however, gives the rams a disadvantage under a yield-based payment system in that bone contributes little to the value of a carcass.

3.4.8 Leg Characteristics

The leg and femur muscle to bone ratios were higher in the ewe lambs than the ram lambs, as was femur muscularity. However, when adjusted to a constant muscle to bone ratio the muscularity was significantly higher in the rams. Improved femur muscle to bone ratios in ewes have been reported in a number of studies (Wood et al., 1980; Purchas & Wilkin, 1995; Mahgoub & Lodge, 1998; Carson et al., 1999; Wolf et al., 2001; Jones et al., 2002), whilst Teixeria et al. (1996) and Hopkins (1996) found no differences. In reports of muscularity Wolf et al. (2001) and Hopkins (1996) found no differences between sexes whilst Jones et al. (2002) found no differences when using the standard formula of Purchas et al. (1991), but when using alternative indices involving side lengths found ram lambs to have higher muscularity than ewe lambs. The reason for the increased muscle to bone ratios of the ewes in this study, is that the rams have heavier bone weight and length relative to their muscle, so which ever way the ratio is defined the rams will have lower values.

The relationship between MTB and MUSC differed between the rams and ewes in a similar way to that reported for bulls and heifers by Purchas et al. (2002), so that at the same MTB the MUSC of the ram was higher than that of the ewes. This means that if MTB is being predicted from a measure of carcass shape such as MUSC, then ram carcasses will need to have a higher MUSC value in order to achieve the same MTB. Or looked at from another perspective, for carcasses of the same MUSC, those from ewe lambs will have a higher MTB and therefore a higher lean-meat yield if levels of fat percent are the same.

3.4.9 Meat Quality

Although significant results were found for meat quality traits, the difference between the sexes was often small, and the fact that they were significant is likely due to the large number of animals in this trial.

Differences between sexes in pH were detected for SM and LL, with ram lambs having a higher pH. In other studies comparing ram and ewe lambs no differences in pH were detected (Corbett et al., 1973; Dransfield et al., 1990; Jeremiah et al., 1997b; Díaz et al., 2003). The exception is Bickerstaffe et al. (2000), who considered the differences between keeping sexes together at the works versus not, and showed that ram lambs

kept with ewe lambs prior to slaughter had increased pH levels. Colour differences were not consistent between the muscles; however, the trend was the same (and significant in most cases) with muscles from ewe lambs lighter and redder than those from rams. This is in contrast to the results of Jeremiah et al. (1997b), Vergara et al. (1999) and Diaz et al. (2003) who found no differences in colour between the sexes. When the data were adjusted for pH and pH^2 the differences in L* no longer existed between the sexes, but the difference in a* remained. It is possible that these differences are due to differences in proportions of haem pigment in the sexes, with Ledward and Shorthose (1971) showing muscle from ewe lambs to have on average 20% more haem pigment than cryptorchid lambs. In terms of quality, visual appearance is an important consumer trait, ideally meat needs to be bright and red in colour. Based on these results it would appear that the meat from ewe lambs would have a visual advantage, whether these differences would be detectable by eye is unclear and follow up studies using sensory-panel assessment would have to be carried out.

For SM and LL indicators of meat toughness (Warner-Bratzler measurements) were significantly higher for the ram lambs than the ewe lambs, effects that remained even after adjustment for pH and pH^2. Bickerstaffe et al. (2000) also showed ewe lambs to be more tender than that from ram lambs. This is in contrast to the results of Corbett et al. (1973), Kemp et al. (1981a) and Vergara et al. (1999) who found no differences in shear force between the sexes and Dransfield et al. (1990) who showed the LL from ewes to be slighter tougher than those from rams. Additionally the studies of Corbett et al. (1973), Kemp et al. (1981) and Dransfield et al. (1990) involved sensory evaluation and found no differences between ewe and ram lambs for sensory traits. Arsenos et al. (2002) and Kirton et al. (1983) only conducted sensory evaluation and found no significant differences between sexes. In all of these studies the minimum carcass weight was 14kg although most were at least 18kg which makes them comparable to those in the current study, although some included in the study of Kirton et al. (1983) were likely to be larger as they were over two years of age (exact weight not reported).

Sex differences in tenderness for lamb meat have been small and non-significant. In cattle although few comparisons have been made directly between heifers and bulls (management problems), those that have, have shown there to be no significant differences between bulls, heifers and steers (equivalent to wether) (Wierbicki et al.,

1956). Comparisons between steers and bulls, and steers and heifers independently have shown the bull beef to be tougher than that from steers in some studies, but there to be few differences between steers and heifers. These differences are thought to arise from the increased levels of testosterone that occur in the male, which leads to their more aggressive nature and increased rates of glycogen depletion pre slaughter, and also to their lower fat levels. Production of testosterone is puberty dependent, however, from the weights described for the other trials one would have expected that the majority of the ram lambs described, would have been around puberty and thus producing testosterone, although this is not known for certain. It remains a fact though, that meat from ram lambs was significantly less tender in this trial, a result that has important meat quality implications for the future if quality control is to become an increased part of lamb production systems.

Increased muscling phenotypes such as the Callipyge in Dorset sheep are also associated with negative tenderness. Correlations between lamb leg composition and meat quality traits (Table 3.10) were considered, as there is an industry trend towards increased muscling and decreased fat. That no highly significant correlations existed between any of the traits is a positive sign, however, there were some correlations of moderate significance. For *M. longissimus* there was evidence that as the muscle to bone ratio increased, so did indicators of meat toughness, an effect that was more pronounced for the ram lambs than the ewe lambs. This has implications in that although consumers are demanding increased leanness in the products they are buying, this could have negative effects on meat quality. An example of this has been seen in the pig industry where selection for increased lean in pigs lead to PSE (pale, soft, exudative meat) (Fox et al., 1980; Moelich et al., 2003). Thus breeders may be placed in a situation where they may have to monitor and maintain meat quality while selected for increased muscling.

3.5 Conclusions

Many previously reported differences between ewe and ram lambs were confirmed in this study:-

- At the same carcass weight ram lambs had increased muscle relative to ewe lambs.
- At the same carcass weight ram lambs had decreased fat relative to ewe lambs.

- Leg weight (as proportion of carcass weight), increased in ewe lambs relative to ram lambs.

However, observations not consistent with the literature were:

- Muscle to bone ratio higher in ewe lambs than in ram lambs.
- Meat quality was poorer in ram lambs (as assessed by pH, colour and tenderness) relative to ewe lambs.

Based on this information, both ram and ewe lambs have some characteristics that will increase their carcass value under proposed new yield- and quality-based payments systems and others which will decrease it, specifically:-

- Ewe lambs will be disadvantaged by their increased fat levels, but will be advantaged in that slightly more of their weight is within the higher value leg cut. However, the fat level disadvantage could be overcome by slaughtering at a lighter weight.
- Ram lambs will be advantaged by their increased muscle and decreased fat, but will be disadvantaged if quality is considered in some way.

The net effect of the trade-offs discussed above can not be determined until meat companies reveal their exact methods of payment.

Consistent meat quality differences (across pH, colour and tenderness) have not previously been reported, but if these differences exist as shown in this study, they pose a problem for the New Zealand lamb industry as it aims for improved and more consistent quality. This is of particular concern because of the indication (albeit small) that there is a negative correlation between increased muscling and tenderness.

Thus further consideration needs to be given by meat companies to defining minimum standards. The industry also needs to be aware of the implications that may come from moving towards increased muscling in that it may have an impact on the quality of the meat produced, an area which will require ongoing monitoring.

Chapter 4

The Search for QTL Affecting Traits Contributing to Lamb Value

4.1 Introduction

The type of lamb which captures the highest returns will change as the New Zealand lamb meat industry moves towards yield-based payment. This change is necessary to meet the increasing demands of overseas consumers for a consistent, high-quality product, in which there is less fat and more muscle, particularly in the high-price cuts. Accurate measurement of improved quality is currently difficult, but will become a reality as plate-to-paddock traceability and automated yield measurement becomes part of the industry. Although some breeders are looking to move towards improving their animals in this direction with traditional selection techniques, faster rates of genetic gain are theoretically possible using genetic regions known as Quantitative Trait Loci (QTL) (Meuwissen & Goddard, 1996).

Most phenotypic traits are assumed to be influenced by a large number of allelic variants of genes involved with the trait, each of which have a small cumulative effect on the phenotype, but with some having larger effects than others. Quantitative Trait Loci are regions of DNA which explain a significant proportion of the variation in phenotypes. These QTL encompass the genes which have a major influence on metabolic or signalling pathways affecting the trait in question. Moderate correlations exist between most fat and muscle traits, because they share a number of metabolic or signalling pathways, so is it likely that a single QTL may effect both trait groups. If a QTL can be identified which has significant effects on either of these traits, the opportunity exists to base selection pressure on the genetic make up of the animal, rather than indirectly on its phenotype. The aim is to rapidly increase the frequency of animals carrying the favourable form of the QTL. This type of selection is referred to as Marker Assisted Selection (MAS), a method which is to be applied within the dairy industry for genetic improvement (Spelman & Garrick, 1997; Spelman et al., 2001). Although in the case of the dairy industry the increase in genetic gain is expected to be small, as it is being applied to already routinely measured traits such as lactation yields. Quantitative Trait Loci, are considered to be particularly useful as a selection criteria for traits that are difficult to measure, sex-limited, or are measurable only after slaughter (Meuwissen & Goddard, 1996).

In order to commence a QTL search, a population of animals is required which exhibits variation in the trait of interest. While Texel sheep are known for their improved

carcass attributes relative to other breeds (see Chapter 2), there appears to be considerable variation within the breed for muscle traits and conformation (Wolf et al. 2001). This suggests that there is considerable genetic variation for these traits, and so Texel sheep provide a good population to use in investigating underlying genetic causes of the muscling-related phenotypes.

Increased muscling within certain breeds of cattle has long been known (Culley, 1807 in Charlier et al., 1995). The most commonly described cattle breed in which this occurs is the Belgian Blue which is known for its "double-muscling". In 1995 the QTL causing this effect was mapped to a region of bovine Chromosome 2 (Chr2) referred to as the muscular hypertrophy locus (Charlier et al., 1995). Subsequently GDF8 (a member of the transforming growth factor-β superfamily) was mapped to this region and a polymorphism in the second intron of the gene was shown to be associated with the double-muscling phenotype in Belgian Blue cattle (Smith et al., 1997). Other polymorphisms in the GDF8 gene were identified by Grobet et al. (1998) as being associated with the double-muscling phenotype in other breeds of European cattle including the Limousin and Blonde d'Aquitaine. A similar double-muscling phenotype in mice was created through knocking out the GDF8 gene (McPherron et al., 1997). A further hyper-muscular phenotype (not as severe as the above double-muscled phenotype) in a line of mice referred to as "Compact", has also been shown to be caused by mutations to the GDF8 gene (Varga et al., 1997; Szabo et al., 1998).

Regions of DNA responsible for increased muscling seen in two lines of sheep have already been identified. The first was in a line of Dorset sheep in America that showed an increased muscling phenotype now known as the Callipyge phenotype (Greek for "beautiful buttocks"). The mutation causing this phenotype has been mapped to a region of ovine Chromosome 18 (Georges et al., 2003; Smit et al., 2003). A line of Poll Dorset sheep identified in Australia (Banks, 1997) have also been shown to have increased muscling. This effect has also been mapped to a region of Chromosome 18 close to but distinct from the Callipyge locus (McLaren et al., 2003). Also, the effects seen are quite different in that it only affects *M. longissimus* (compared with the whole body) (Nicoll et al., 1998). This locus is referred to as the Rib-Eye Muscling (REM) locus, with the Carwell allele at this locus responsible for the improved phenotype (Jopson et al., 2001). Further details of these loci are summarised in Chapter 2.

Two studies had been carried out searching for a QTL causing the increased muscling phenotype in Texels at the commencement of the current investigation. In the first Marcq et al. (1998) used GDF8 as a starting point. Although they found no sequence differences in the coding region of the GDF8 gene of Belgian Texels or Romanov controls, they felt that there could be a functional polymorphism outside the coding sequence. To test this theory they initiated a linkage analysis with microsatellite markers flanking the GDF8 gene in a (Texel x Romanov) x (Texel x Romanov) F_2 pedigree. Two of the 39 continuous traits tested (one of which was carcass conformation) gave LOD scores greater than 3, from which they concluded that a QTL affecting muscle development mapped to the proximal region of ovine chromosome 2q (Marcq et al., 1998). This provided sufficient evidence for New Zealand scientists to conclude that, if the mutation was present in the New Zealand Texel population, it would be detectable from the segregation pattern of progeny of a sire heterozygous in the region of interest (John McEwan pers comm.).

The first study to search for a QTL in the New Zealand Texel population was reported by Broad et al. (2000). In this study only purebred Texels were screened. Due to the small size of the New Zealand Texel stud population, only 36 sires over three properties met the minimum threshold of 40 or more progeny. Five markers in the region of GDF8 (BM81124, BY5, BULGE20, BULGE23 and INRA40) were initially genotyped. Only 12 of the 36 sires were heterozygous for one or more of the markers, with only two sires heterozygous at all markers. This suggested low levels of polymorphism around the GDF8 locus in Texels. Two sires were heterozygous at all five markers, one sire at four markers and three sires at three markers. Progeny of the 12 sires were genotyped. The data available on the progeny consisted of live weights and ultrasound scanning measurements including *M. longissimus* width (A), depth (B) and fat depth (C). Of the 12 sires, only three showed evidence for QTL segregating in the region of GDF8 when the data were liveweight-adjusted. For one sire the favourable form of the QTL was associated with an increase in *M. longissimus* depth of 2.4mm ($P<0.05$). For the second sire there was an decrease in *M. longissimus* fat depth of 1.45mm ($P<0.05$). For the third sire there was an increase in *M. longissimus* width of 2.3mm, depth of 1.4mm and area of 1.5cm^2 ($P<0.05$).

In unpublished follow-up work to Broad et al. (2000) a further 13 markers (BM4006,

OARFCB128, TGLA10, BY5, BM81124, BULGE23, BULGE20, INRA40, CP78, TEXAN2, TG377, HH30, ILSTS30, OARFCB20, RM356, BMS1126, BM6444, FCB110 (markers previously used are underlined)) were genotyped for those sires showing evidence of a QTL. This analysis provided further evidence that sire Waikite 295/97 was segregating for liveweight-adjusted *M. longissimus* dimensions, although fat depth C was significant, *M. longissimus* area only approached significance. The position of the QTL peak appeared to be around marker INRA40. There was a suggestion that sire Waikite 150/96 was segregating for a QTL for *M. longissimus* fat depth at marker FCB20, although this effect was not significant, and even less so when adjusted for liveweight. Sire Waikite 295/97 was the son of Waikite 150/96.

Based on the results of Broad et al. (2000) and subsequent analyses, there was sufficient evidence to justify follow-up experiments to confirm the location and to estimate the size of the QTL effect. In the previous trial purebred Texel animals were used, this meant that the dams were also purebred Texels and had similar alleles to the sires making identification of the sire inherited allele difficult. Furthermore, because the animals were purebred and being used within breeding schemes, only live-animal measurements were available. With this in mind it was decided that to increase the chances of detecting a QTL and estimating the size of its effects on carcass traits, commercial non-Texel dams would be used to generate the progeny using a half-sib family design. This assumed that the outcross dams were not segregating for the trait. This would reduce potential confounding dam effects on the QTL estimates and would also enable the progeny to be slaughtered, thereby allowing more detailed carcass and meat quality assessments to be collected.

The objectives of this work were to use the sires identified previously, or their sons, which were heterozygous for markers in the region of interest, to generate cross-bred progeny, which would be taken through to slaughter with as many traits recorded as possible, and compared these traits against genotypes. Attention focused not only on carcass traits, but also meat quality traits as previous work with muscling QTL has shown that they may also have detrimental effects on meat quality (e.g. Callipyge in Dorset sheep (Jackson et al., 1994a; Duckett et al., 2000); selection for increased lean in pigs leading to PSE (pale, soft, exudative meat) (Fox et al., 1980; Moelich et al., 2003)).

4.2 Materials and Methods

4.2.1 Ethical Approval

Ethical approval for this experiment was obtained from the Animal Ethics Committee, Massey University, New Zealand. Protocol number 00/167.

4.2.2 Trial Design

The trial design chosen for the experiment used half-sibs as first described by Neimann-Sorensen and Robertson (1961). The basis of the design is to generate a large number of progeny from a sire, heterozygous at the markers used, with approximately half the resulting progeny carrying one allele and the remainder the other allele.

4.2.3 Animals

Sires used

Based on the results of Broad et al. (2000) and follow up work, rams Waikite 295/97 and Waikite 150/96 were identified as sires that should be used. Sire Waikite 150/96 was used as a reference sire in the New Zealand Texel Sire Referencing Scheme in 1999, sons and grandsons of this sire were throughout Texel studs and on AgResearch properties. Sire Waikite 295/97 was a son of sire Waikite 150/96, but progeny of this sire only existed on one property. Only sire Waikite 150/96 was still alive at the time of the trial. Six purebred and 14 Coopworth-cross sons of sire Waikite 150/96 and five purebred sons of Waikite 295/97 were genotyped using the following eight markers OARFCB128, BM81124, BULGE20, INRA40, TEXAN2, ILSTS30, OARFCB20 and RM356. Choice of sires was based on heterozygosity of markers, particularly in the regions of interest, which for sons of sire Waikite 295/97 was in the region of marker INRA40, and for sons of Waikite 150/96 was in the region of marker OARFCB20.

Progeny numbers required

The trial design consisted of two parts, the first a chromosome arm test for presence of a QTL and the second a test of its effects at the QTL peak. The formula used to calculate the minimum number of progeny required to detect a QTL for each of these tests was:-

$$n = \frac{2\sigma^2(t_{1-\beta} + t_{\alpha/2})^2}{f\delta^2}$$

n is the number per marker group (assumed to be half the total number of offspring)

t is the critical value for a t distribution

α is the point-wise significance level

δ is the difference in marker group means [diverge*(1 – (θ*2))]

f is the proportion of informative offspring

σ^2 is the within marker group variance

β is the power required

θ is the largest distance between the point of analysis and the closest marker

diverge is the average effect of allele substitution

This formula is based on the power calculations of Steele and Torrie (1980), but was modified for use with QTL searches to consider the size of the QTL effect, the relative distances between markers and the likely informativeness of individuals.

Table 4.1 gives the values used to estimate the number of progeny required for a chromosome arm significance test for QTL detection and to detect statistically significant effects at a given QTL position.

Table 4.1 Values used to estimate the number of progeny required to detect a QTL within a Texel half-sib population

	Chromosome Arm Significance	Tests at the QTL peak
α	0.002	0.05
θ	0.1	0.05
diverge	1	0.8
β	0.8	0.8
δ	0.8	0.8
f	0.7	0.7
σ	1	1
df	98	62
$t_{1-\beta}$	3.17	2.0
$t_{\alpha/2}$	0.85	1.15
n	72	45
estimated number required	144	90

From Table 4.1 it was estimated that 144 progeny per sire would need to be generated, on which basic data would be collected, but that only 90 would be required for detailed carcass dissection measurements.

Generation of Progeny

Three properties were used in the trial; details on the properties are presented in Table 4.2. The summary details for number of lambs born per sire and class groups (sex, rearing rank and dam age) are in Table 4.3. To achieve the 145 progeny required, 145

dams were single sire mated to each of the sires. However, there was considerable variation in number of lambs born per sire (Table 4.3). For Property One this reflects the use of highly fecund ewes, but for the others, reflects low pregnancy rates.

Table 4.2 Details of properties and animals used

	Property 1	Property 2	Property 3
Farm Name	Woodlands Research	Tanbar	Skye
Location	Invercargill, South Island	Ohai, South Island	Dannevirke, North Island
Property description	Flat	Terrace/river flats	Steep hill country
Sires used	Waikite 535/98	Waikite 150/96	Premier 15/98
	Waikite 429/98	Woodlands 1170/00[2]	Premier 122/99
		Woodlands 1199/00[2]	
Dam breed	Coopworth	Romney	Perendale/Romney
Start of lambing	12/09/01	29/09/01	10/10/01
Lambing beats	Daily	Daily	Every two days[1]
Weaning	5/12/01	22/01/02	26/01/02
Scanning	22/02/02	18/02/02	06/04/02
Slaughter group one	23/02/02	23/02/02	
Slaughter group two	04/04/02	04/04/02	
Slaughter group three			08/04/02
Slaughter group four			01/07/02

[1] Due to the locality of the property daily lambing beats were not practical. Based on the work of Amer et al. (1999) in deer, it was decided that tagging of the lambs every two days would be adequate to estimate date of birth
[2] Note these two sires are Texel x Coopworth, while the balance are purebred Texels

Table 4.3 Number of lambs born per sire

		Sex		Rearing Rank				Age of Dam						
Sire	**Total**	**E**	**R**	**1**	**2**	**3**	.[1]	**2**	**3**	**4**	**5**	**6**	**7**	.[1]
1170/00	107	58	49	25	82				27	36	8	32	2	2
1199/00	113	53	60	21	79	12	1		35	49	5	23		1
150/96	121	53	68	24	91	6			18	63	8	24	7	1
122/99	83	37	46	56	27						42	41		
15/98	123	61	62	64	59						63	60		
429/98	211	121	90	31	171	9		111	33	37	18	12		
535/98	213	105	108	31	172	9	1	118	43	21	17	12	2	

[1] missing information

4.2.4 Measurements Made

Live animal measurements

Lambs were weighed at birth (BT), weaning (W), scanning (SC) and slaughter (SLG) (Table 4.2). Average daily weight gains were estimated between birth and weaning (ADGBW) and between weaning and scanning (ADGWSC). Measurements by ultrasound of *M. longissimus* width (UA), depth (UB) and fat depth over *M. longissimus* (UC) were made on all lambs prior to the first slaughter group for each property.

Slaughter procedure

Details of assignment for slaughter and slaughter procedures are given in Chapter 3,

Section 3.2.2. Briefly, there were two slaughter groups for each property, with assignment to the first slaughter group based on a minimum liveweight of 35kg and 38kg for ewe and ram lambs, respectively. This criterion enabled an evaluation to be undertaken on commercial properties, and was based on the observation by Broad et al. (2000) that the QTL could be detected after liveweight adjustment.

Measurements at the meat works

Measurements are described in Chapter 3, Section 3.2.3 and are listed in Table 4.4.

Assignment of lambs for further analysis

The right leg and loin from carcasses of 90 progeny per sire for six sires were selected for further analysis in the laboratory. No samples were collected for sire 122/99 (note from this point flock prefixes are omitted) due to insufficient progeny. Ideally all progeny born would have been further analysed, however, costs prevented this. The selection criteria for further analysis varied for the three properties, but the aim was to have those selected be representative of the sire. For Property One (sires 429/98 and 535/98) where there were predominately twin-reared lambs, with few single- or triplet-reared lambs, it was decided that the legs/loins for further analysis should only be selected from the twins. For Property Two (sires 1170/00, 1199/00 and 150/96), most lambs were single or twin born with few triplet born, so triplet born lambs were excluded from further selection. For Property Three (sires 15/98) there were no triplet born lambs, and the numbers of twin and single reared lambs were approximately equal so both were included for selection. Within these constraints 45 ewe and 45 ram lambs were selected at random. As this was carried out prior to genotyping, it was assumed that this would also ensure a representative sample of genotypes.

4.2.5 Dissection of Legs and Loins

Details of the dissection technique and subsequent measurements of meat quality for two muscles are described in Chapter 3 Section 3.2.4 and a list of the measurements is given in Table 4.4.

Table 4.4 Abbreviations and descriptions of carcass and meat quality measurements made

Description	Abbreviation
Measurements Made at the Processing plant	
Dressed carcass weight (kg)	CCW
Dressing-out percentage (Dressed carcass weight/slaughter weight)	DO
The distance taken from between the hind legs to the front of the neck (cm)	CLNG
The distance at the widest part of the forequarter (cm)	WF
The minimum width of the thorax (behind the shoulders) (cm)	WTH
The maximum width at the gigots (cm)	G
Soft tissue depth, measured at the 12th rib, 110mm from the mid-line (mm)	GR
Widest part of LL muscle at cranial end of loin (mm)	A
Deepest part of LL muscle, at right angle to A, at cranial end of loin (mm)	B
Subcutaneous fat depth over B (mm)	C
M. longissimus muscle area from tracing of cranial end of loin (mm^2)	EMA
Measurements Made During Dissection	
Trimmed leg (g)	TLG
M. semimembranosus (g)	SM
M. semitendinosus (g)	ST
M. biceps femoris (g)	BF
M. adductor femoris (g)	AD
M. quadriceps femoris (g)	QD
M. gluteus medius (g)	GM
All remaining muscle in leg not defined above (g)	MST
Total leg muscle (g)	TLMS
All leg subcutaneous fat, to level of muscle (g)	SCF
All leg intermuscular fat (g)	IMF
Total leg fat (g)	TLFT
M. longissimus (g)	LL
Subcutaneous fat of the loin (g)	LLFT
Femur bone (g)	FM
Pelvic bone (g)	PV
All bone in leg (includes femur and pelvic) (g)	TLBN
Length of femur bone (mm)	FMLNG
Meat Quality Measurements	
Warner Bratzler peak force *M. semimembranosus* (kg)	WBPFSM
Warner Bratzler yield force *M. semimembranosus* (kg)	WBYFSM
Warner Bratzler peak force *M. longissimus* (kg)	WBPKLL
Warner Bratzler yield force *M. longissimus* (kg)	WBYFLL
Cooking loss *M. semimembranosus* (%)	CKSM
Cooking loss *M. longissimus* (%)	CKLL
Sarcomere length *M. semimembranosus* (μm)	SLSM
Sarcomere length *M. longissimus* (μm)	SLLL
Colour "L*" *M. semimembranosus*	CLSM
Colour "a*" *M. semimembranosus*	CASM
Colour "b*" *M. semimembranosus*	CBSM
Colour "L*" *M. longissimus*	CLLL
Colour "a*" *M. longissimus*	CALL
Colour "b*" *M. longissimus*	CBLL
pH *M. semimembranosus*	PHSM
pH *M. longissimus*	PHLL
Leg muscularity (see definition 3.2.7)	LGMUSC
Femur muscle to bone (see definition 3.2.7)	MTB
Percentage fat in leg	LGFT%
Percentage lean in leg	LGMS%

4.2.6 Genotyping

Blood sampling

Blood samples were collected from all lambs using easiTrace™ DNA-labels (FTA paper with a glue backing) (Shackell et al., 2001). 10mm scratches were made on the inside of the lambs' ear, on an area free of dirt or lanolin build up. Once a well of blood had formed the FTA paper was put in contact with the blood. Labels were attached to numbered cards and the blood dried before being stored at room temperature.

DNA extraction

A full description of the DNA extraction technique is provided in Appendix 8.1.

Markers used

The same markers used for the genotyping of the potential sires were used for genotyping the trial progeny, with the addition of TGLA10 for the progeny of sire 15/98. Details of the markers used are described in Table 4.5. Note from this point on marker prefix OAR is omitted and ILSTS30 is referred as ILST30.

Table 4.5 Details of the primers used

Marker[1]	Primer A[2]	Primer B[2]	Gel Running Time (Hours)	Source of primers	Reference
FCB128	CAGCTGAGCAACTA AGACATACATGCG	ATTAAAGCATCTTCT CTTTATTTCCTCGC	1 ½	Macromolecular Resources	Buchanan et al. (1993)
TGLA10	CTAAATTTATCCCA CTGTGGCTCTT	CAATCTGCAGTAGCA TACATCCTTG	3	Genset Oligos	Georges and Massey (1992)
BM81124	GCTGTAAGAATCTT CATTAAGCACT	CCTGATACATGCTAA GGTTAAAAAC	2 ½	Gibco BRL	Stone et al. (1995)
BULGE20	CAGCAGGTCTGTTG AAGTGTATCAG	AGTGGTAGCATTCAC AGGTAGCCAG	2	Invitrogen Life Technologies	Grobet et al. (1997)
INRA40	TCAGTCTGGAGGAG AGAAAAC	CTCTGCCCTGGGGAT GATTG	3	Genset	Vaiman et al. (1994)
TEXAN2	ACATTGTCATGTGG TTGCTAAC	ACTCTGGGTATGTAT ATGTCAAG	2	Gibco BRL	Holder et al. (1994)
ILST30	CTGCAGTTCTGCAT ATGTGG	CTTAGACAACAGGG GTTTGG	2	Gibco BRL	Kemp et al. (1995)
FCB20	AAATGTGTTTAAGA TTCCATACAGTG	GGAAAACCCCCATA TATACCTATAC	1 ½	Invitrogen Life Technologies	Buchanan et al. (1993)
RM356	GCATCACTAACATC CACTGAGG	CCACTAGGAGAGGT CATTCCC	2	Gibco BRL	McGraw et al. (1997)

[1] NB that ILSTS30 is abbreviated to ILST30 and OARFCB markers abbreviated to FCB
[2] Primers are the sequence of base pairs that amplify the specific sequence of AC repeats in a marker

DNA amplification

Amplification of the DNA was carried out using the Polymerase Chain Reaction (PCR), which requires a radioactive label, cocktail and DNA, details of which, along with the PCR programme used are in Appendix 8.1.

Gel electrophoresis

Gels were run on homemade plates, with 48 lanes per gel. Details of the gel used are included in Appendix 8.1. The gel was poured into the plate ensuring that no air locks formed and was allowed to set. The plates were attached to plastic backing and top and bottom placed in 1:10 dilution of x10 TBE electrophoresis buffer and connected to an electrophoresis power pack. Then 10µl of azide was added to each well of amplified DNA, which was then heated at 95°C for four minutes prior to loading the gels. Four microlitres of amplified DNA was added to the lanes on the gels using a 12 channel Hamilton syringe. Electrophoresis took place at 38volts/cm, with each marker requiring a different running time as is shown in Table 4.5. Each gel contained four controls, made up of two positive controls of known genotypes, one negative control (blank) and the sire for the given progeny. Lanes were run for all marker/lamb combinations with the exception of marker TGLA10, which was only run for sire 15/98.

Processing the gels

After electrophoresis the gels were transferred onto card and allowed to dry. The cards were placed in radiographic cassette cases with yellow fast film added (Kodak X-OMAT-AR) in the dark room and were exposed over night (>12 hours). The film was developed automatically using a 100-Plus x-ray film processor.

Allele scoring

Alleles revealed on the gels were manually scored using the A to Z scale described in Table 4.6 which is unique to the AgResearch laboratory in which the genotyping work was carried out. In the first instance control wells were scored, if any controls were incorrect the gels were rerun. All gels were independently scored by two people.

Markers BULGE20, INRA40, BM81124, TEXAN2 and FCB128 ran well, with the gels clear and alleles easy to read and score. Marker ILST30 had doublets (double bands visible for each allele) on 70% of the gels and so distinguishing between the F and G alleles was at times difficult and double scoring identified some that needed careful rescoring. Marker FCB20 did not run well, attempts to improve the standard cocktail did not improve it and it was decided to only score those individuals where the results were unambiguous. Marker TGLA10 did not run well initially, with the controls not scoreable, but the use of new primers and cocktails (described in Appendix 8.1)

produced clear, scoreable gels for this marker.

Table 4.6 A-Z Scoring for markers used in the Texel QTL trial. Each letter corresponds to a different allele. Distance from top corresponds to number of AC repeats, with those nearer the top having fewer, and so travel a shorter distances along the gel

Microsatellite Markers								
FCB128	**TGLA10**	**BM81124**	**BULGE20**	**INRA40**	**TEXAN2**	**ILST30**	**FCB20**	**RM356**
M	A	A	A	N	S	M	A	P
A		B	B	M	M	A	Y	
B	B	C	C	A	N	B		A
C	C	D	D	B	O		B	M
	D	E	E	C			C	
	E	G	F	D		C	Z	B
		Z	G	E		D	D	C
D	F	H	H	F	T	E	X	D
E	G	S	I	G		F		E
		R	J	H		G	E	N
	H	Q	K		A		F	F
		P	L				G	G
		O	M		R		H	
	I	N			F		I	
F		M			B		J	
		L			C			
		K			D			
		I			L			
		F			P			
		J			E			
		T						
		U						
		V						
		W						
		X						
		Y						

4.2.7 Genotype Testing

Genotypes were tested using the following criteria, to determine if they followed the rules of Mendelian inheritance.

Pedigree

Through the laws of inheritance each progeny genotyped should have inherited one allele from its sire at each marker. For all individuals that appeared to have incorrect pedigrees the genotypes were re-scored. If, after re-scoring, two or more marker genotypes were inconsistent with that of the putative sire it was assumed that the animal did not belong to that sire and it was excluded from analysis. Where only one marker out of the eight had an incorrect genotype the individual remained in the data set, as mutations do occasionally occur. For these markers, the genotype was set to missing. Thirty-six animals were excluded on this basis.

Multiple recombinants

Double recombinants are unlikely to occur within a 30 centiMorgan (cM) region, and it is even less likely that it should occur within a 20 cM region. All instances of double-recombination at less than these distances were re-scored. Re-scoring did not eliminate all instances of double recombination, however, they occurred at an acceptably low frequency across the population (<0.05%).

Segregation

Under normal inheritance each sire allele should be inherited by approximately half of its progeny. Determining the allele inherited cannot be carried out where the progeny are heterozygous for the same alleles as its sire. For the remainder of genotypes, however, the inherited allele can be determined. The proportions of each allele were compared using a Chi-squared test. Any significant ($P<0.05$) deviations from the expected 50% were checked by re-scoring the progeny for that sire at the given marker.

Family differences in recombination fraction

Although differences in recombination fractions for different sire groups can exist, it was assumed that they would be similar and that any major differences indicated a problem in scoring. For each marker pairing, the number of instances of recombination and non-recombination (1 and 0) were determined for each sire and compared between sires using Likelihood Ratio Chi-Squared analysis. Cases with $P<0.01$ and within 40cM were checked by rescoring the affected families for the given makers.

4.2.8 Map Distances

The map used to obtain distances between marker was that previously used in the analysis by Broad et al. (2000). It was developed using the male map positions from de Gortari et al. (1998) and inserting additional markers in their relative positions, based on information from the International Mapping Flock (IMF) and the Broad flocks combined. Although distances could have been obtained from analysis of the current data set, it was felt that it was more suitable to use the distances generated previously as the minimum number of informative meiosis for any one marker was 137, whilst that for the current data set was 81 (Table 4.7). Additionally, in the previous mapping work more markers, were mapped over a wider region increasing the chance of detecting double recombinants relative to the current data set, thereby increasing its accuracy. An

alternative source of marker distances would have been the maps produced by Maddox et al. (2001; 2002). However, this source of distances is based on the IMF animals, and so was already included in the original analysis.

Comparison of the use of any of the three maps (original, current data or Maddox maps), showed that although the absolute (cM) positions of the markers varied, relative positions of the markers were approximately the same (Table 4.7).

Table 4.7 The position of markers on ovine chromosome two, and the respective number of informative meiosis on which the distances are based for three flock genotyping data sets

	1998 de Gortari		1998 Texel + IMF		Current Data	
	Position (cM)	No. Informative Meiosis	Position (cM)	No. Informative Meiosis	Position (cM)	No. Informative Meiosis
FCB128	0	354	0	215	0	349
TGLA10	15	182	26.2	220	30.8	81
BM81124	46	325	47.6	234	53	458
BULGE20			51.4	308	54	437
INRA40			54.5	328	58.7	745
TEXAN2	63	179	63.8	229	74.3	762
ILSTS30	78	137	79	185	91.6	522
FCB20	97	272	102	274	105.6	391
RM356	115	425	119.5	184	120.8	726

4.2.9 Pedigree

Pedigree information was obtained from New Zealand Texel Society records. Pedigree diagrams were created using Pedigree Viewer © (Kinghorn pers comm.)

4.2.10 Single Peak QTL Analysis

The method used to detect the existence of QTL was to test for associations between the genotype probabilities of inheriting a given grand-paternal allele and the associated phenotypes at intervals along the chromosomal region of interest (Knott et al., 1996). In order to do this the phase inherited by the progeny from the grand-sire was determined and used to calculate conditional genotype probabilities. This information was used in regression analyses (along with other fixed effects and covariates described below) to determine whether or not there were statistically significant associations between genotype and phenotype.

Phase determination

The grand-parental phase was determined using the chrompic function of CRI-MAP

(Green et al., 1990) using progeny and paternal sire information. This function begins by finding the maximum estimates of the recombination fractions, which are used to find the phase with the highest likelihood for that grand-sire. Groups of marker alleles for any given phase will be inherited together unless recombination between the markers occurs. The two grand parental phases were arbitrarily assigned as Phase 1 and Phase 2.

For each progeny, the allele phase inherited from the sire was determined as Phase 1 or Phase 2. For example, a '0' was given when the phase could not be determined when the sire and individual were both heterozygous for the same alleles and no information on the dams was available. An individual could have a combination of alleles inherited from Phase 1 and Phase 2 if crossing over of the DNA (recombination) occurred in the production of the gamete. An example of phase determination in the grand-parents and then in the progeny is outlined in Table 4.8.

Table 4.8 An example of phase determination in a sire and one of his progeny

	FCB128	BM81124	BULGE20	INRA40	TEXAN2	ILST30	FCB20	RM356
Sire Genotype	AA	CF	HI	EG	AN	BF	BI	EE
Grand-sire Phase 1	[1]	C	I	E	A	B	I	[1]
Grand-mother Phase 2	[1]	F	H	G	N	F	B	[1]
Progeny Genotype	AD	FH	EH	GB	LN	BF	HI	EG
Phase Inherited	0[2]	2	2	2	2	0[3]	[1]	0[2]

[1] Sire phase at this marker was not determined as the sire was homozygous at this marker
[2] Sire phase inherited at this marker could not be determined as the sire phase was not determined
[3] Sire phase inherited at this marker could not be determined as the individual was heterozygous for the same alleles as the sire

Genotype probability

Having determined the marker inherited by each individual from its sire, the probability of inheriting a given putative QTL allele, for given recombination/non-recombination fractions were determined for positions in the region of interest. The genotype of interest is a QTL allele, Q assumed to be inherited with Phase 1, with the alternative q allele inherited with sire Phase 2. Because the QTL location is unknown, information from the flanking markers (M and N) and its estimated position relative to them needs to be used to determine the probability of inheriting Q as shown in Figure 4.1. The probability of inheriting Q was estimated at 2 cM intervals along the region of interest.

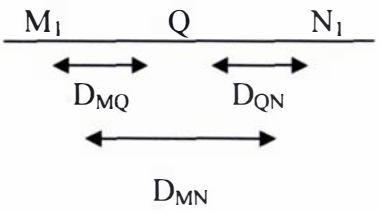


Figure 4.1 An example of a QTL (allele Q) lying between markers M, and N, on sire phase 1 ($_1$). The distances D_{MQ}, D_{QN} and D_{MN} represent the distances in centiMorgan (cM) between the two markers and the QTL and the total distance between the two markers.

The positions of the markers and the distances between them were estimated using the Haldane mapping function, expressed in centiMorgan (cM), which assumes no interference. However, to work out the genotype probabilities these were converted to recombination fractions (r) using the following formula:

$$r_{MN} = 1/2\left(1 - e^{2D_{MN}}\right)$$

where D is the map units (in Morgans).

In a half-sib design where only the sire genotypes and phases are known (that is, no information on the dams), as for this study, there are eight possible marker genotype combinations for any two markers, four relating to Phase 1 (Q) and four relating to Phase 2 (q). In this analysis only information on Phase 1 was used, the four combinations relating to Phase 1 are:-

1. When no recombination (or double recombination) takes place between the QTL and the phase one markers, a haplotype that is M_1QN_1 results.
2. If there was recombination between the first marker and the QTL, but not between the QTL and second marker the haplotype would be M_2QN_1.
3. If there was no recombination between the first marker and the QTL, but there was between the second marker and the QTL the haplotype would be M_1QN_2.
4. If there was recombination between both markers and the QTL the haplotype would be M_2QN_2.

In practice 5 other situations also need to be considered: when a QTL allele is flanked by a marker on one side with no information on the other side.

The conditional genotype probabilities associated with these four different haplotypes

are based on whether recombination (r) has taken place between the marker and the QTL or not (1-r), these formulas are given in Table 4.9.

Table 4.9 Conditional probability formulas for half-sib family design given recombination (r)

Haplotype inherited from Sire[1]	Conditional Probability of Q at QTL Locus[2]
M_1N_1	$(1-r_{MQ})(1-r_{QN})/(1-r_{MN})$
M_1N_2	$(1-r_{MQ})r_{QN}/r_{MN}$
M_2N_1	$r_{MQ}(1-r_{QN})/r_{MN}$
M_2N_2	$r_{MQ}r_{QN}/(1-r_{MN})$

[1] M_1, M_2, N_1 and N_2 correspond to the alternative phases for the first and second markers respectively

[2] This column relates to the recombination fraction between different marker/QTL combinations; the allele Q at the QTL locus is assumed to be associated with phase one of the markers; values that are r_{xy} are where recombination has taken place and $(1-r_{MN})$ being when no recombination has taken place between locus M and N

An example of the use of these values is provided for a putative QTL positioned at 60 cM along the region of interest, where the flanking markers are INRA40 (56.5cM) and TEXAN2 (65.8cM) the relative distances are presented in Table 4.10, and Table 4.11 shows the phase information and the conditional probability values.

Table 4.10 An example of converting distances to recombination fractions for a QTL position relative to two markers using Haldane formula

	Distance (cM)	Recombination Fraction (r)
INRA40-TEXAN2 (M-N)	9.3	0.174 (r_{MN})
INRA40-QTL (M-Q)	3.5	0.074 (r_{MQ})
QTL-TEXAN2 (Q-N)	5.8	0.117 (r_{QN})

Table 4.11 Examples of estimating conditional probability of inheriting favourable allele (Q) at 60cM for four progeny of Sire 1190/00 that inherited varying haplotypes from their sire, given formula and values in Tables 4.9 and 4.10 respectively

Progeny	Haplotype Inherited from Sire	Conditional Probability of Q at QTL Locus
102/01	M_1N_1	0.990
106/01	M_1N_2	0.623
43/01	M_2N_1	0.376
100/01	M_2N_2	0.010

The above rules and formulas do not hold true when the marker is uninformative either in the sire or in the individual (sire is homozygous or individual is heterozygous for the same alleles as the sire). In such instances the next informative flanking marker is used to determine the conditional probabilities. If these are not available then the probability is calculated from single marker information.

Conditional probability values range from zero to one. A conditional probability value of zero indicates that there is no possibility of inheriting the Q allele at the QTL locus, and that the allele inherited is the q allele. A conditional probability value of one indicates that the Q allele has been inherited. In Table 4.11 it can be seen that the Q

allele with the M_2N_2 haplotype would require two events of recombination to take place (between each marker and QTL locus) and given the small distances between the marker and the QTL locus, the probability of this occurring is low. In the case of the M_1N_1 haplotype where no recombination is required the probability of the Q allele being inherited with this haplotype is high. For the two combination haplotypes, the probability is related to the span within which recombination must occur, and is always low if recombination must occur within a small distance.

QTL analysis

All initial QTL analyses were undertaken within sire groups. Analysis of data took place at 2cM intervals along the 122cM region of interest, giving 61 analyses for each sire/trait combination. The basis of the analysis was the Haley Knott Regression method (Haley & Knott, 1992), further adapted for use with outbred populations (Knott et al., 1996). This approach identifies whether or not residual phenotypic differences (after adjustment for fixed effects and covariates) in the mean values across lambs, for a given trait for the different QTL alleles existed by regressing the phenotypes (after adjustment for fixed effects and covariates) on the probabilities that the Q allele as calculated above (Table 4.11). In the example presented in Figure 4.2 the slope of this line is 2.5, with those carrying the Q allele having a higher mean residual value for muscle percent than those carrying the q allele. If there had between no differences between the two alleles (Q and q) the slope would not have been significantly different from zero, depending on the threshold chosen.

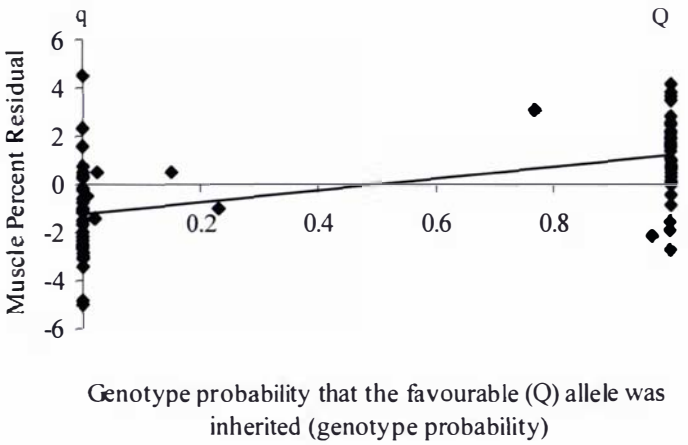


Figure 4.2 An example of a regression of phenotype (muscle percent residuals) against the genotype probability that the Q allele was inherited at 60cM for sire 1199/00

The analyses were carried out in SAS using the General Linear Models (GLM) Procedure (SAS, 1991). The fixed effects and covariates identified for each trait (described in 4.2.16) were fitted in a model along with the genotype probabilities (calculated above) nested within sire number.

The $-\log_{10}$ of the nominal probability values (probability that the difference between the phenotypic values for the trait associated with Q and q alleles are significantly different from zero) were plotted for individual traits and groups of traits, as this statistic is independent of degrees of freedom, which varied between traits.

Details of the models fitted are provided in Section 4.2.16.

Estimation of the size of the QTL effect

The size of the QTL effect was estimated from the covariate effect using SAS (1991) by regressing phenotype on the conditional QTL allele probabilities. The size of the effect was compared against the adjusted phenotypic standard deviation for the trait (created by fitting the model including all fixed effects and adding back the sire sums of squares to the error sums of squares, thus removing non-genetic effects, but ensuring no confounding effects between sire and other fixed effects).

Significance thresholds

There are two types of errors that can occur when searching for a major QTL (Doerge & Churchill, 1996). A type I error occus when a QTL does not exist, but significant results are shown. A type II error occurs when there are QTL effects present, but they are not detected. In order to minimise both of these, suitable significance thresholds should be used. Significance thresholds were estimated in two ways, one using the formulae of Lander and Kruglyak (1995) and the other using the permutation test or "shuffle test" described by Doerge and Churchill (1996). The permutation test requires less assumptions and provides threshold values for each sire, however, when all sires were plotted on the same graph individual thresholds could not be shown, whereas the threshold values described by Lander and Kruglyak (1995) were suitable for any set of data, given its genome length and the desired genome-wide threshold.

The phenotypic data were randomly assigned to the genotypes for the permutation tests

and the analysis rerun 10,000 times, the recommended number of re-runs to test significance at the 1% level. The test statistic was recomputed for each new sample, generating an empirical distribution under a null hypothesis of no phenotype-genotype associations. The threshold values at the 1% and 5% levels were set from the probability of obtaining by chance an estimate with a value as extreme as that found within the orginal data set.

Lander and Kruglyak (1995) proposed specific standards for interpreting thresholds to minimise the chance of Type I and Type II errors occurring. These formulae only take account of the chromosomal distance over which the QTL is being sought and the desired level of significance, and do not take into account the distribution of the data. However, because the formulae are universal they can be used to identify suggestive and significant thresholds across different analyses. For this work nominal p values of 0.00157 and 0.0000505 and the corresponding $-\log_{10}$ transformed values (3.8 and 4.2, respectively) were used for genome wide suggestive and significant thresholds, respectively based on the ovine genome length and chromosome number (Lander and Kruglyak (1995)).

Confidence intervals

Confidence intervals for the position of the QTL peak were generated based on the "bootstrapping" method described by (Visscher et al., 1996). Bootstrap samples were created by sampling with replacement individuals from the data-set for that sire, the number sampled being equal to the number of individuals in the original data-set for that sire. By sampling with replacement it means that some individuals may occur more than once in any one bootstrap set. Boot strapping was repeated 500 times from which the empirical central 90 and 95% confidence intervals for the QTL position were obtained by ordering the estimates and taking the 5^{th} and 95^{th} percentiles or the 2.5^{th} and 97.5^{th} percentiles, respectively.

4.2.11 Information Content

The Information Content (IC) is a measure of how informative the sires are. For each position, if the true allele descent (inherited from sire or dam) could be determined, the conditional probabilities at that position would be either 0 or 1 (Spelman et al., 1996). If this were the case the mean conditional probability would be 0.5 with a variance of

0.25. Given that not all progeny are informative, the mean and variances of the conditional probabilities vary across the region of study. The information content is calculated as the ratio of the variance in conditional probability at any position, to the variance when true descent is known (Spelman et al., 1996). For example for Sire 1199/00, at position zero cM the variance of the conditional probabilities is 0.17, which as a proportion of 0.25 is 0.68; but at 50cM the variance of conditional probabilities is 0.24, which as a proportion of 0.25 is 0.96.

4.2.12 Multi-trait Analysis

Multivariate analysis

For leg muscle, leg fat, loin muscle and loin fat traits multivariate analyses were carried out (a description of the traits within each groups is provided in 4.2.16). Multivariate analysis was carried out by specifying the multiple analysis of variance (MANOVA) function within the GLM procedure in SAS. To do this all traits within a specified group were fitted to the left hand side of the model equation. Canonical coefficients were also specified to provide an indication of the relative rankings of the different traits within a group. The Wilks' λ results were used to calculate the significance of the analysis at each position along the region of interest. The QTL analysis was carried out as previously described, with the inclusion of the MANOVA function.

Principal component analysis

For leg muscle, leg fat, loin muscle and loin fat traits principal component analyses were carried out (a description of the traits within each groups is provided in 4.2.16). Each trait within the groupings was analysed within flock using the GLM Procedure in SAS, using the models previously described and the residuals were stored. These residuals were then analysed using the principal components procedure in SAS, with the principal components stored. The previously described method of QTL analysis was applied to the first and second principal components, except the models only included the sire genotype probabilities, and not the fixed effects already taken into account in the prior GLM procedure.

4.2.13 Two-Peak Analysis

For sire/trait combinations where there was graphical evidence for two peaks reaching the suggestive thresholds based on the single trait analysis, two-peak analyses were

carried out to determine if more than one QTL mapped to the region studied. The model previously described for the single-peak analysis was used, with the fixed effects and covariates identified for each trait (described in 4.2.16) fitted in a model. Additionally in this analysis, the genotype probability at position one nested within sire and the genotype probability at position two nested within sire along with the interaction between the two positions was fitted in the model (the interaction provides a test for epistasis: an effect at one QTL having an effect on the other). All combinations of position one and position two were tested. Results were first interpreted by considering whether or not there was an epistatic interaction between the two positions (by observing the significance of the three terms (two peaks seperately, plus interaction)). If there was no evidence for epistasis, the model without the interaction fitted was considered further. A two vs one peak analysis was then carried out using the best two peak model (from above) to determine if fitting a two peak model provided a better explanation of the data.

4.2.14 Sex-QTL Interaction Analysis

The purpose of this analysis was to determine whether the effect of the QTL was different for each sex as shown by different regression slopes. In addition to the sire genotype probability term in the model equation an interaction with sex was also fitted to give a sex by sire-genotype-probability. Two results were considered. Firstly, whether or not fitting the sex by sire-genotype-probability interaction (Q+I) provided evidence for significant QTL peaks, this could, however, mean that there was a significant QTL in both sexes. The second result considered therefore, was given a peak for Q+I, whether or not the actual sex by sire-genotype-probability interaction was significant (I), that is that the two sexes were different. Estimates of the size of the effect for each sex were calculated by adding the sire genotype probability estimate to the sex by sire genotype probability estimate. The standard error was calculated using the following formula:

$$Var(A+B) = Var(A) + Var(B) + 2Cov(A,B)$$

where A = sire genotype probability estimate, B = sex by sire conditional probability estimate.

4.2.15 Linkage Disequilibrium Analysis

Linkage disequilibrium (LD) analysis can be used to identify markers closely associated

with a given trait at the population level (Baret & Hill, 1997). In this study, it was used to search for allele-phenotype associations within the dam population for each property. The progeny of all dams were considered together (within property), as for any single dam there was at most three progeny. As a result, the conditional probability approach could not be used as across the dam populations there were often more than two alleles for any markers (could not have Q and q). Analysis was carried out at each marker. This is a less informative approach as it does not include information from flanking markers, if no information is available at a particular marker. However, if there was a dam allele association it would appear in such an analysis, although the exact location and relative size of the effect may not be well estimated. All steps previously described were carried out to the point of fitting the model in SAS which now also included dam allele.

4.2.16 Analysis of Data

Trait groups

Traits were assigned into 11 groupings as follows:-

Liveweight traits: BT, W, SC, SLG, ADGBW, ADGWSC

Ultrasound traits: UA, UB, UC, UEMA

Dressing-out percentage: DO

Carcass linear traits: CLNG, WF, WTH, G

Leg muscle traits: LG, SM, ST, BF, AD, QD, GM, MST, LGMS%, LGMUSC, MTB

Leg fat traits: SCF, IMF, TLFT

Leg bone traits: PVBN, FMBN, TLBN, FMLNG

Loin muscle traits: A, B, EMA, LL

Loin fat traits: C, GR, LLSCF

M. semimembranosus meat quality traits: WBPKSM, WBYFSM, CKSM, SLSM, CLSM, CASM, CBSM, PHSM

M. longissimus meat quality traits: WBPKLL, WBYFLL, CKLL, SLLL, CLLL, CALL, CBLL, PHLL

Models fitted

Although each sire was analysed independently, models were separately fitted for each property level providing common error terms. The only fixed effect which remained in all models irrespective of significance level was sire. Other fixed effects fitted were the

time at which measurements were made, sex, age of dam, birth/rearing rank, slaughter group and dissector. All liveweight traits (except birth-weight) were adjusted for birth date, all ultrasound traits were adjusted for liveweight on the day, and all post-slaughter composition traits were adjusted for carcass weight. In the final models only fixed effects significant at the 5% level and interactions significant at the 1% level when using Type III sums of squares for at least three of the traits in a group were included. A list of the models fitted is in Appendix 8.2.

It was most appropriate to adjust the post-slaughter data to a constant carcass weight because the trial design specified that lambs slaughtered at an approximately constant live-weight. Adjusting for carcass weight considers whether or not there were composition differences over and above those associated with differences in carcass weight. However, also of interest was whether or not growth rates of the different components were affected by the QTL. This was answered by adjusting the data for age at slaughter (as opposed to weight).

4.3 Results

4.3.1 Pedigrees

The pedigree diagrams for sires 150/96 and 429/98 are presented in Figures 4.3 and 4.4 and the pedigree diagrams for sires 535/98, 15/98 and 122/99 are presented in Appendix 8.3. The pedigrees for sires 1170/00 and 1199/00 are not included, as their sire pedigree is that of 150/96, and although they are not full-sibs, their dams were both Coopworth ewes, which although fully recorded are not relevant to this study. The pedigree of sire 150/96 is presented in the results, as it is the pedigree that all sires share in common, and is where the alleles inherited by each sire have been traced from. Sire 429/98 is presented in the results, as unlike all other sires none of its alleles were most likely inherited from sire 150/96 (its grandsire), despite sharing alleles in common with the other sires.

For either of these pedigrees (150/96 or 429/98), there are no loops within approximately 3 generations, however, over the whole pedigree there are four inbreeding loops that link sire 150/96 to the dam of 429/98 (animals 33/85; 271/86; 632/82 and 92/82). Within the pedigree of sire 150/96, sire 364/85 is the great great grandsire on the dam line of 150/96 and the great grandsire on the sire line.

Figure 4.3 Pedigree diagram for Sire 150/96. Dam lines are represented in red and sire lines in blue

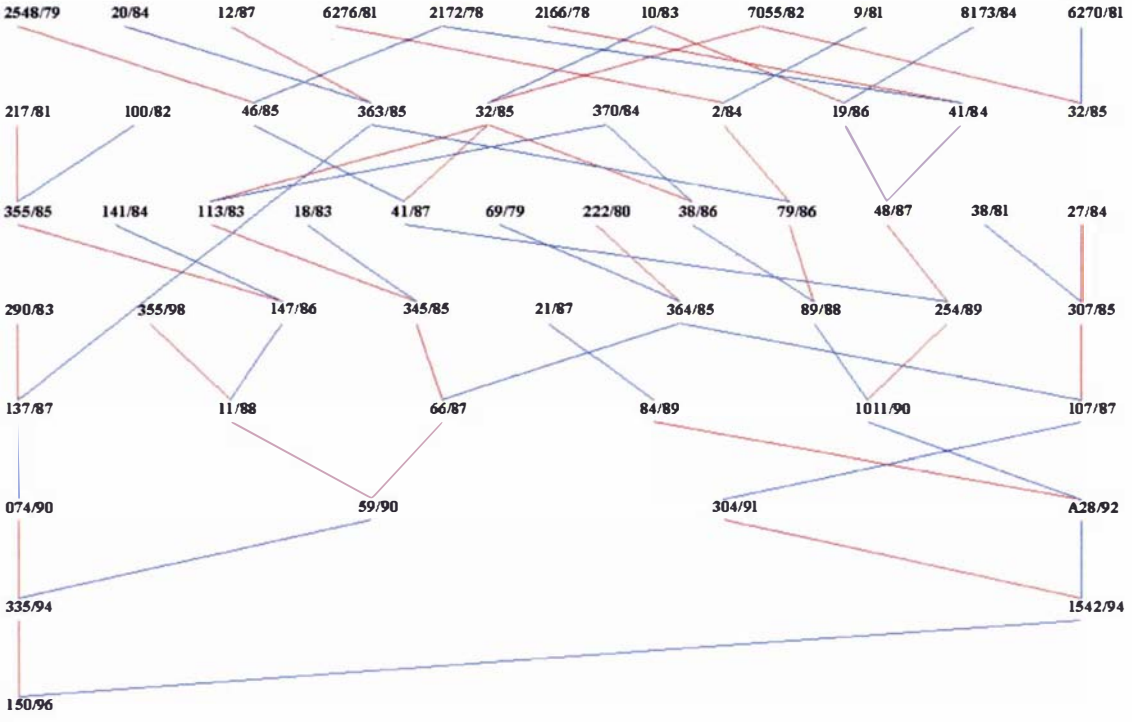
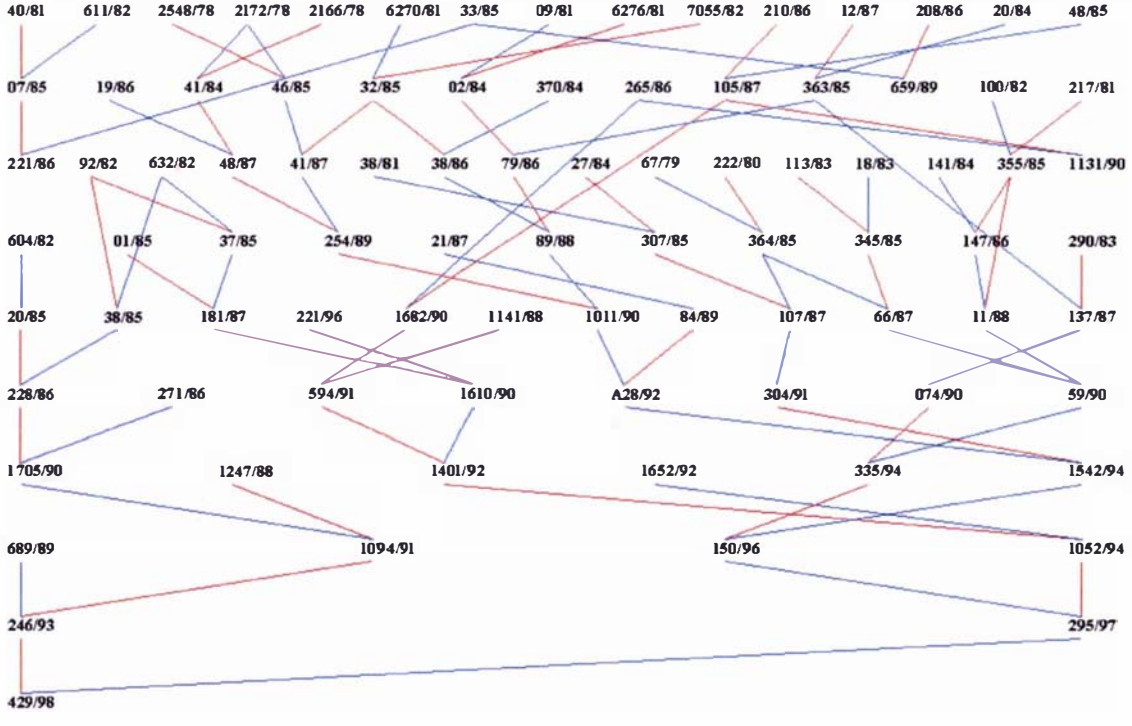


Figure 4.4 Pedigree diagram for Sire 429/98. Dam lines are represented in red and sire lines in blue



4.3.2 Inherited Phases and Haplotypes

Tables 4.12 and Table 4.13 show the phases and haplotypes for the seven sires used in the trial and sire 295/97 (son of 150/96 and sire of 429/98 and 535/98). In the case of 150/96 the two phases were arbitrarily assigned as either having been inherited from its sire (Phase 1) or its dam (Phase 2) and for all his sons the phase inherited from the sire is coded as to whether it was inherited from the paternal-granddam or paternal-grandsire.

Because sire 150/96 was homozygous for markers FCB128, BM81124 and BULGE20 the phase inherited could not be determined, however, for Table 4.12, it was assumed that no recombination had taken place in this region. Sires 1170/00 and 15/98 both inherited the granddam phase (Phase 2) in the region studied (markers FCB128 through RM356), whilst sire 122/99 inherited the grandsire phase (Phase 1). Sire 1199/00 inherited the granddam phase (Phase 2) from markers FCB128 through TEXAN2, however, recombination occurred somewhere between TEXAN2 and FCB20 (could not be fully determined as 150/96 was homozygous at ILST30) with the grandsire phase (Phase 1) inherited at FCB20 and RM356. Sire 295/97 (the sire of 429/98 and 535/98) inherited the granddam phase between markers FCB128 and FCB20. However, recombination had occurred between FCB20 and RM356 with the grandsire phase inherited at marker RM356. Sire 535/98 inherited the great-granddam phase (Phase2) between markers FCB128 and INRA40, recombination occurs after this marker and thus neither phase of 150/96 were inherited. Sire 429/98 inherited the alternative phase from sire 295/97 (from that which was inherited from 150/96) and thus neither phase of 150/96 was inherited by this sire.

Table 4.12 Phase inherited for markers in region of interest on ovine chromosome two for the rams used as sires[1]

	Phase Inherited From Sire 150/96 at Marker								
	FCB128	**TGLA10[2]**	**BM81124**	**BULGE20**	**INRA40**	**TEXAN2**	**ILST30**	**FCB20**	**RM356**
1170/00	2		2	2	2	2	2	2	2
1199/00	2		2	2	2	2	0	1	1
15/98	2	2	2	2	2	2	2	2	2
122/99	1		1	1	1	1	1	1	1
295/97	2		2	2	2	2	2	2	1
429/98	-		-	-	-	-	-	-	-
535/98	2		2	2	0	-	-	-	-

[1] 1 indicates that the sire phase of 150/96 was inherited; 2 indicates that the dam phase of 150/96 was inherited; 0 indicates that the phase inherited from 150/96 could not be determined, whilst – indicates that neither phase of 150/96 was inherited

[2] This marker was only used for sire 15/98

Table 4.13 Sire haplotypes for markers in region of interest on ovine chromosome two[123]

		FCB128	BM81124	BULGE20	INRA40	TEXAN2	ILST30	FCB20	RM356
150/96	Sire	A	C	I	A	B	B	B	G
	Dam	A	C	I	E	A	B	I	E
1170/00	Sire	A	C	I	E	A	B	I	E
	Dam	M	I	L	F	M	G	D	C
1199/00	Sire	A	C	I	E	A	B	B	G
	Dam	F	Z	K	D	C	F	H	B
15/98	Sire	A	C	I	E	A	B	I	E
	Dam	A	F	H	G	N	F	B	E
122/99	Sire	A	C	I	A	B	B	B	G
	Dam	A	C	I	A	O	G	B	N
295/97	Sire	A	C	I	E	A	B	I	G
	Dam	C	B	L	E	E	G	H	B
535/98	Sire	A	C	I	E	E	G	H	B
	Dam	A	C	I	A	B	F	D	M
429/98	Sire	C	B	L	E	E	G	H	B
	Dam	D	C	I	C	C	B	G	A

[1]Cells highlighted in blue and pink indicate that they were inherited from the sire and dam of 150/96 respectively
[2]The underscores represent instances where the sire inherited allele was not determined as the sire was homozygous at that maker
[3]∩ indicates that crossing over has occurred somewhere in the region highlighted

4.3.3 Information Content

The information content (IC) is based on the variance of conditional probabilities at a given position and is related to the number of informative sires and the frequency of the alleles in the dam population. Only sires 1170/00, 1199/00 and 429/98 were heterozygous at all markers (Table 4.13). Sire 15/98 was heterozygous at seven of the nine markers it was scored for, whilst sires 535/98, 150/96 and 122/99 were heterozygous at five, four and three respectively of the eight markers they were scored for. All sire alleles were present within the dam population except allele "G" for marker INRA40. The IC shown in Figure 4.5 of all sires was high (>0.7) from marker BM81124 through ILSTS30, and remained above 0.6 through to marker RM356 with the exception of Sire 15/98 (15/98 was homozygous at marker RM356 and its IC drops from marker FCB20). Sires 535/98 and 150/96 had low IC at marker FCB128 as the sires were homozygous at this marker, but rose through to INRA40, the first marker at which these sires were heterozygous. Sire 15/98 had a low IC at marker FCB128 (as it was homozygous), however, this was the sire for which an additional marker (TGLA10) was run, as TGLA10 was a highly informative marker the IC rose to 0.8 at this marker and continued to rise to marker BM81124.

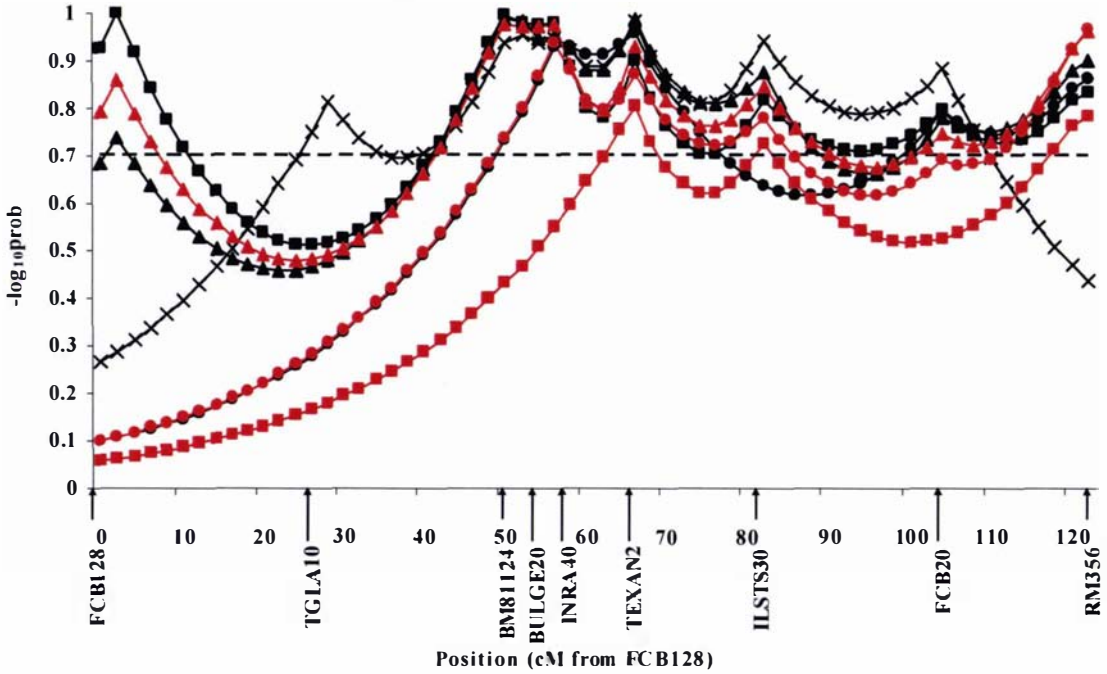


Figure 4.5 Information content along the region of interest of ovine chromosome two for half-sib Texel cross lambs within the sire groups
1170/00 ▪▪▪ ; 1199/00 ▲▲▲; 150/96 ◆◆◆; 15/98 ✱✱✱; 122/99 ▪▪▪ ; 429//98 ▲▲▲; 535/98 ◆◆◆; ---- value above which information content considered to be high

4.3.4 Allele Frequencies

The allele frequencies inherited from the sires for each marker are presented in Table 4.14. With the exception of TGLA10 (which was only genotyped for sire 15/98) all other markers had at least four alleles represented between the sires, the most common alleles being those that sire 150/96 possessed. All other sires had at least one allele in common with this sire. As previously noted, sire 429/98 did not inherit any alleles from 150/96, but it still has an allele in common.

The allele frequencies inherited from the dams for each marker are presented in Table 4.15. There were a number of alleles across the dam populations for each marker, some with higher frequencies than others (e.g. allele F for marker FCB128). Overall, however, there were no alleles which were fixed for any one dam population. The majority of sire alleles were represented in the dam population at low frequencies.

In some cases it could not be determined whether an allele was inherited from the sire or dam, because the progeny were heterozygous for the same alleles as the sire. These are denoted "?" in Tables 4.14 and 4.15.

Table 4.14 Sire-inherited allele frequencies (%) for markers used in Texel cross half-sib lambs within sire groups

		Sire Group						
Marker	**Allele**	**1170/00**	**1199/00**	**122/99**	**15/98**	**150/96**	**429/98**	**535/98**
FCB128	?[1]	1	26	-	-	-	17	-
	A	50	26	100	100	100	-	100
	C	-	-	-	-	-	40	-
	D	-	-	-	-	-	43	-
	F	-	47	-	-	-	-	-
	M	49	-	-	-	-	-	-
TGLA10	?	-	-	-	28	-	-	-
	H	-	-	-	29	-	-	-
	I	-	-	-	43	-	-	-
BM81124	?	6	5	-	28	2	12	-
	C	46	46	100	37	98	48	100
	D	-	-	-	-	-	40	-
	F	-	-	-	35	-	-	-
	I	48	-	-	-	-	-	-
	Z	-	49	-	-	-	-	-
BULGE20	?	25	19	-	6	3	18	1
	H	-	-	-	37	-	-	-
	I	28	39	100	57	97	37	99
	K	-	42	-	-	-	-	-
	L	46	-	-	-	-	46	-
INRA40	?	23	22	-	3	14	5	10
	A	-	-	100	-	41	-	43
	C	-	-	-	-	-	47	-
	D	-	38	-	-	-	-	-
	E	39	41	-	59	44	47	47
	F	38	-	-	-	-	-	-
	G	-	-	-	38	-	-	-
TEXAN2	?	18	2	21	2	9	21	32
	A	28	51	-	56	41	-	-
	B	-	-	32	-	50	-	39
	C	-	47	-	-	-	39	-
	E	-	-	-	-	-	40	30
	M	54	-	-	-	-	-	-
	N	-	-	-	42	-	-	-
	O	-	-	47	-	-	-	-
FCB20	?	42	54	12	19	44	67	70
	B	-	21	88	37	25	-	-
	D	37	-	-	-	-	-	12
	G	-	-	-	-	-	14	-
	H	-	25	-	-	-	19	19
	I	21	-	-	44	31	-	-
RM356	?	21	8	25	-	17	3	3
	A	-	-	-	-	-	47	-
	B	-	37	-	-	-	50	49
	C	47	-	-	-	-	-	-
	E	32	-	-	100	33	-	-
	G	-	55	42	-	50	-	-
	M	-	-	-	-	-	-	48
	N	-	-	34	-	-	-	-

[1] "?" means that the allele inherited from the sire could not be determined due to the individual being heterozygous for the same alleles as the sire at that locus

Table 4.15 Dam-inherited allele frequencies (%) for markers used in Texel cross half-sib lambs within sire groups

Marker	Allele	1199/00	1170/00	150/96	15/98	122/99	429/98	535/98
FCB128	?[2]	26	1	-	-	-	17	-
	A	1	-	3	3	2	1	1
	B	1	2	10	15		12	16
	C	9	17	14	17	9	2	7
	D	25	30	40	23	27	12	21
	E	14	8	10	15	10	4	2
	F	22	42	22	26	51	48	49
	M	2	-	-	1	1	3	3
TGLA10	?	-	-	-	28	-	-	-
	A	-	-	-	1	-	-	-
	C	-	-	-	5	-	-	-
	D	-	-	-	9	-	-	-
	E	-	-	-	5	-	-	-
	F	-	-	-	12	-	-	-
	G	-	-	-	4	-	-	-
	H	-	-	-	8	-	-	-
	I	-	-	-	12	-	-	-
	L	-	-	-	15	-	-	-
	X	-	-	-	1	-	-	-
BM81124	?	5	6	-	28	-	10	-
	A	1	-	-	-	-	2	-
	B	1	2	3	6	5	2	2
	C	3	6	7	4	5	9	18
	D	8	12	6	18	10	4	11
	E	-	-	-	5	-	1	-
	F	68	53	60	12	43	26	29
	G	-	2	2		-	-	-
	H	-	-	-	1	-	-	-
	I	2	4	11	1	4	19	18
	J	1	-	-	1	4	1	3
	L	2	5	-	-	1	-	-
	N	-	3		12	12	4	8
	O	4	4	5	-	3	9	4
	R	-	-	-	1	-	-	-
	S	-	-	-	1	3	-	-
	V	-	1	-	-	-	-	-
	Z	5	3	5	11	10	13	6
BULGE20	?	19	25	3	6	-	18	1
	B	-	-	-	-	-	-	-
	C	1	-	-	1	4	1	1
	D	-	-	-	1	3	-	-
	E	28	32	41	21	23	42	38
	F	3	1	1	-	1	14	16
	G	-	1	-	10	1	-	-
	H	-	2	1	-	4	-	-
	I	2	2	9	4	1	-	2
	J	2	-	-	1	1	-	-
	K	5	7	5	13	13	10	4
	L	25	11	25	41	40	8	29
	M	16	19	15	2	8	6	8

		Sire Group						
Marker	**Allele**	**1170/00**	**1199/00**	**122/99**	**15/98**	**150/96**	**429/98**	**535/98**
INRA40	?	22	23	14	3	-	5	10
	A	1	1	-	1	1	1	-
	B	-	-	-	7	4	1	1
	C	18	15	12	6	3	1	1
	D	8	25	17	43	36	50	49
	E	9	8	9	3	17	3	3
	F	17	11	22	22	30	8	11
	H	23	16	23	9	8	27	17
	M	-	2	1	1	-	-	-
	N	3	-	2	5	1	5	7
TEXAN2	?	1	17	9	2	21	21	32
	A	-	-	1	-	-	-	-
	B	28	26	11	33	6	26	15
	C	1	1	3	4	6	11	18
	D	-	-	-	-	-	1	3
	E	13	18	14	13	14	12	8
	F	-	3	5	6	3	2	3
	L	22	14	18	18	26	2	3
	M	29	13	35	21	18	17	10
	N	-	7	2	-	1	5	6
	O	-	1	-	2	-	4	1
	P	4	1	3	3	1	-	-
	R	1	-	1	-	-	-	-
	S	-	-	-	-	1	-	-
	T	-	-	-	-	1	-	-
ILST30	?	33	38	-	19	34	30	48
	A	1	-	-	2	-	-	-
	B	21	20	35	13	8	6	12
	C	3	11	5	-	-	3	2
	D	1	1	7	-	-	1	4
	E	3	7	5	7	12	4	2
	F	15	9	19	5	19	38	13
	G	23	15	30	55	27	19	18
FCB20	?	47	42	44	19	12	68	70
	A	-	-	-	6	5	-	-
	B	3	-	-	1	-	1	-
	D	13	9	10	12	16	13	7
	E	14	7	5	10	12	4	3
	F	3	14	7	8	3	1	2
	G	10	18	18	12	13	1	6
	H	8	8	11	19	32	4	8
	I	3	3	4	1	1	8	4
	Y	-	-	-	7	5	-	-
	Z	-	-	2	5	1	-	-
RM356	?	6	20	17		25	2	3
	A	-	-	-	2	-	1	-
	B	1	2	-	-	1	1	-
	C	47	20	46	34	36	69	60
	D	4	3	5	16	16	5	1
	E	6	3	-	7	5	2	2
	F	9	8	8	2	4	2	6
	G	17	35	13	26	9	16	23
	M	3	2	-	1	1	-	-
	N	3	5	5	8	3	1	3
	P	4	3	7	5	-	-	-

[1]Sire alleles indicated by shading

[2] "?" means that the allele inherited from the sire could not be determined due to the individual being heterozygous for the same alleles as the sire at that locus

4.3.5 Results of QTL Analysis

In the following sections the results for individual traits are initially presented for each sire group separately (4.3.6), but for traits groups with no significant QTL the results are given in Appendices 8.4 through 8.10. Following the within-sire results for individual traits are the results of multi-trait analysis for groups of related traits (4.3.7). These analyses were also carried out within sire groups, but are presented together. Finally an across-sire, multi-trait analysis was carried out. Results of the linkage disequilibrium analysis and the QTL-sex-interaction analysis are presented in Sections 4.3.10 and 4.3.11. Although it is standard procedure to address interaction analyses first, initial analyses showed there to be no consistent evidence for these interactions. Hence to aid development of the results, the interaction results are presented after the analyses which exclude the interactions. Overall summary tables are provided in 4.4.

4.3.6 Single Trait Analysis

Analysis of individual traits, grouped into like traits are presented for each sire. A list of the column headings used in the tables and figures is presented in Table 4.16. Results in the tables relate to the position of the maximum QTL peak for that particular sire/trait combination, and hence the position reported in the tables corresponds to the position of the maximum peak in the corresponding figures. Details of the abbreviations used in the text are provided in 4.2.4.

Table 4.16 Descriptions of the column headings used in tables

Column Heading	Description
Trait	Trait (unit of measurement).
Phen Mean ± adj. std	Within-sire raw unadjusted phenotypic mean of the trait ● the adjusted standard deviation (phenotypic standard deviation with non genetic fixed effects and covariates removed).
Estimate ● stderr	Estimate of size of the effect of the Q allele substitution effect relative to the "wild-type" allele (q) for each sire at the peak in units of the trait ± standard error.
SD Units	Magnitude of the QTL peak: (estimate/adj. std).
Permutation	$-\log_{10}$prob thresholds derived by permutation tests with 10,000 replicates.
95% 99%	Determined for 95% and 99% confidence levels.
max $-\log_{10}$prob	The significance of the QTL peak in terms of $-\log_{10}$ of the nominal probability. Significance Level *, ** - the result reached the permutation test 95%, and 99% thresholds, respectively. $^{sug.\ sig}$ _ the result reached the alternative recommended suggestive (sug) and significance (sig) thresholds (2.8 and 4.3 respectively) of Lander and Kruglyak (L&K) (1996). These values are represented as dashed (sug) and solid (sig) lines on the figures.
Pos (cM) [CI]	Position of the QTL peak relative to marker FCB128 in cM. Confidence Intervals were derived for the position in cM by bootstrapping with 500 replicates.

Sire 1199/00

No peaks reached the suggestive thresholds for sire 1199/00 for weight, ultrasound muscle, linear or meat quality traits, or dressing-out percentage. The results for the non-significant traits are presented in Appendix 8.4.

Leg muscle traits

There was evidence of significant peaks for SM, ST, BF, GM, MST and TLMUS weight (Table 4.17; Figure 4.6). The position of the peaks lay between 38 and 72cM. All peaks reached the 99% significance threshold, with SM, ST, MST and TLMST also reaching the L&K significance threshold, whilst GM reached the L&K suggestive threshold. The effects ranged between 0.7 and 1.0 SD units, for GM and ST respectively.

Peaks existed for all other muscle related traits (Table 4.18; Figure 4.7). The position of the peaks lay between 42 and 72cM. All peaks reached the 99% significance and L&K significance threshold. The effects explained between 1.0 and 1.4 SD units for MTB and LGMUSC, respectively.

Table 4.17 Details of peaks for sire 1199/00 Texel cross half-sib QTL analysis for a region of ovine chromosome two – leg muscle weight traits[1]

Trait	Phen mean ± adj. std	Estimate ± stderr	SD units	Permutation max 95%	99%	-log$_{10}$ prob	pos (cM) [CI]
Trimmed leg (g)	2,541.0 ± 136.3	29.5 ± 18.0	0.2	1.8	2.4	1.0 ^{ns}	56
M. semimembranosus (g)	232.8 ± 18.8	16.0 ± 3.2	0.9	2.2	3.0	5.9** ^{sig}	52 [28 - 82]
M. semitendinosus (g)	80.9 ± 8.1	7.8 ± 1.9	1.0	1.8	2.4	4.3 ** ^{sig}	44 [28 - 66]
M. biceps femoris (g)	224.1 ± 18.0	12.4 ± 4.4	0.7	1.6	2.2	2.3 **	38 [15 - 122]
M. quadriceps (g)	342.6 ± 24.1	10.5 ± 4.9	0.4	1.8	2.3	1.5 ^{ns}	70
M. adductor (g)	106.4● 9.6	3.9 ± 1.9	0.4	1.9	2.5	1.4 ^{ns}	52
M. gluteus medius (g)	155.8 ± 15.1	10.9 ± 3.1	0.7	1.8	2.4	3.3 ** ^{sug}	54 [39 - 82]
Muscle trim (g)	620.0 ± 46.0	38.4 ± 8.7	0.8	2.0	2.8	4.7 ** ^{sig}	62 [15 -74]
Total leg muscle (g)	1,762.7 ± 115.1	94.4 ± 18.8	0.8	1.9	2.6	6.0** ^{sig}	64 [28 - 74]

[1] Descriptions of column headings are given in Table 4.16

Table 4.18 Details of peaks for sire 1199/00 Texel cross half-sib QTL analysis for a region of ovine chromosome two – other muscle related traits[1]

Trait	Phen mean ± adj. std	Estimate ± stderr	SD units	Permutation 95%	99%	max -log$_{10}$ prob	pos (cM) [CI]
Leg muscularity	0.4 ± 0.02	0.02 ± 0.004	1.1	2.1	2.9	5.0 ** ^{sig}	62 [28 - 104]
Leg muscle percent	69.3 ± 2.2	3.1 ± 0.4	1.4	2.6	3.5	11.5 ** ^{sig}	68 [39 - 74]
Femur muscle to bone	4.2● 0.4	0.4 ± 0.1	1.0	2.4	3.3	4.7 ** ^{sig}	42 [29 - 74]

[1] Descriptions of column headings are given in Table 4.16

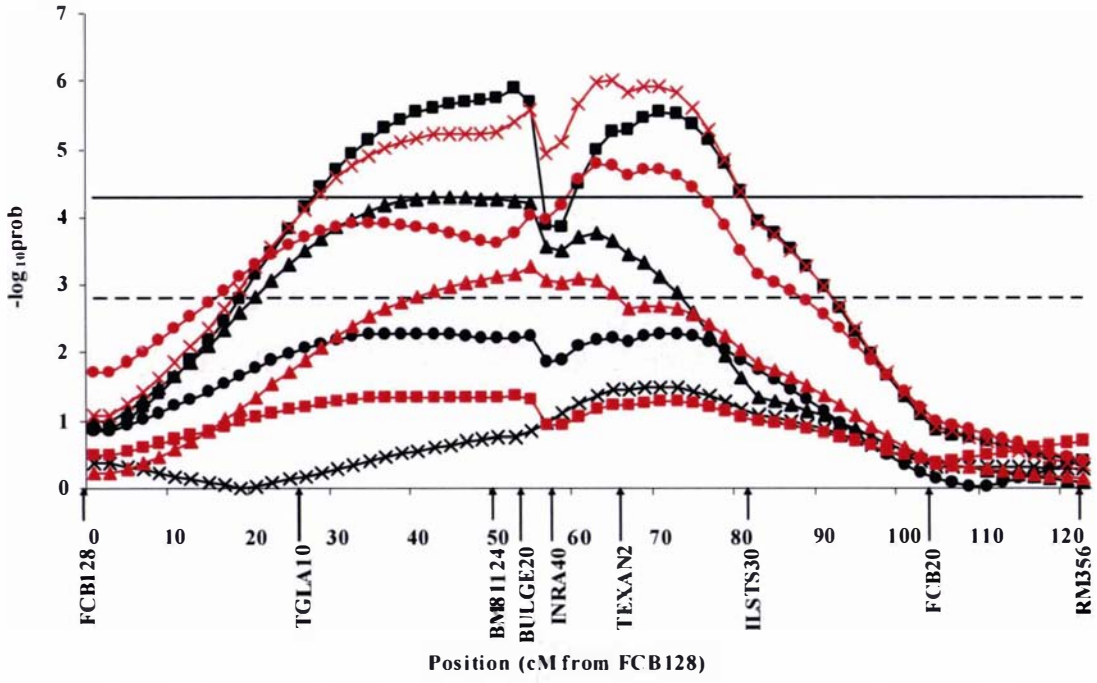


Figure 4.6 –log$_{10}$prob curves for sire 1199/00 half-sib QTL analysis for a region of ovine chromosome two – weights of leg muscles

M. semimembranosus weight ■■■; *M. semitendinosus* weight ▲▲▲; *M. biceps femoris* weight ◆◆◆ ; *M. quadriceps* weight ✱✱✱; *M. adductor* weight ●●● ; *M. gluteus medius* weight ▲▲▲ ; muscle trim weight ◆◆◆total muscle in the leg weight ✱✱✱; suggestive threshold - - - -; significance threshold ——

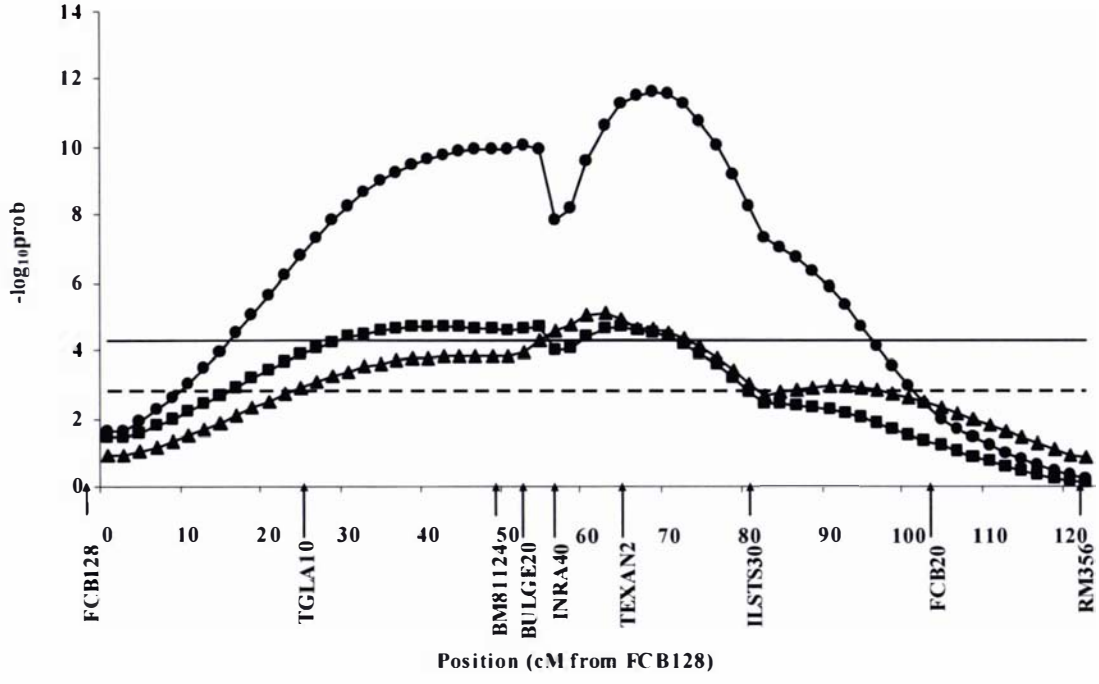


Figure 4.7 –log$_{10}$prob curves for sire 1199/00 half-sib QTL analysis for a region of ovine chromosome two – other muscle related traits

Femur muscle to bone ■■■; leg muscularity ▲▲▲ ; leg muscle percent ◆◆◆ ; suggestive threshold - - -; significance threshold ——

Loin muscle traits

Loin muscle weight was the only loin muscle trait to show evidence of a peak. The peak at 52cM reached the 95% significance threshold and explained approximately 0.5 of a SD unit or 5% of the mean. Further details are in Appendix 8.4.

Ultrasound fat traits

There was evidence for a peak for UC. This peak at 20cM reached the 95% significance threshold and explained approximately 0.7 of a SD unit or 19% of the mean. Further details are in Appendix 8.4.

Leg fat traits

Significant peaks existed for all leg fat weight traits (Table 4.19; Figure 4.8). The position of the peaks lay between 68 and 78 cM. All peaks reached the 99% significance threshold and the SCF, LGFT% and TLFT peaks reached the L&K significance threshold and IMF reach the L&K suggestive threshold. The effects explained between 1.0 and 1.3 of a SD unit for IMF and SCF respectively.

Table 4.19 Details of peaks for sire 1199/00 Texel cross half-sib QTL analysis for a region of ovine chromosome two – leg fat weight traits[1]

Trait	Phen mean ± adj. std	Estimate ± stderr	SD units	Permutation 95%	Permutation 99%	max $-\log_{10}$ prob	pos (cM) [CI]
Subcutaneous fat (g)	201.1 ± 35.3	-43.2 ± 7.6	1.2	2.3	3.1	7.4 ** sig	68 [39 - 74]
Intermuscular fat (g)	141.0 ± 23.4	-24.0 ± 5.9	1.0	2.0	2.6	4.2 ** sug	78 [39 - 93]
Total leg fat (g)	342.1 ± 49.2	-66.2 ± 10.3	1.3	2.4	3.3	9.1 ** sig	70 [50 - 74]
Leg fat %	13.5 ± 1.9	-2.3 ± 0.4	1.2	2.3	3.0	6.2 ** sig	70 [39 – 74]

[1] Descriptions of column headings are given in Table 4.16

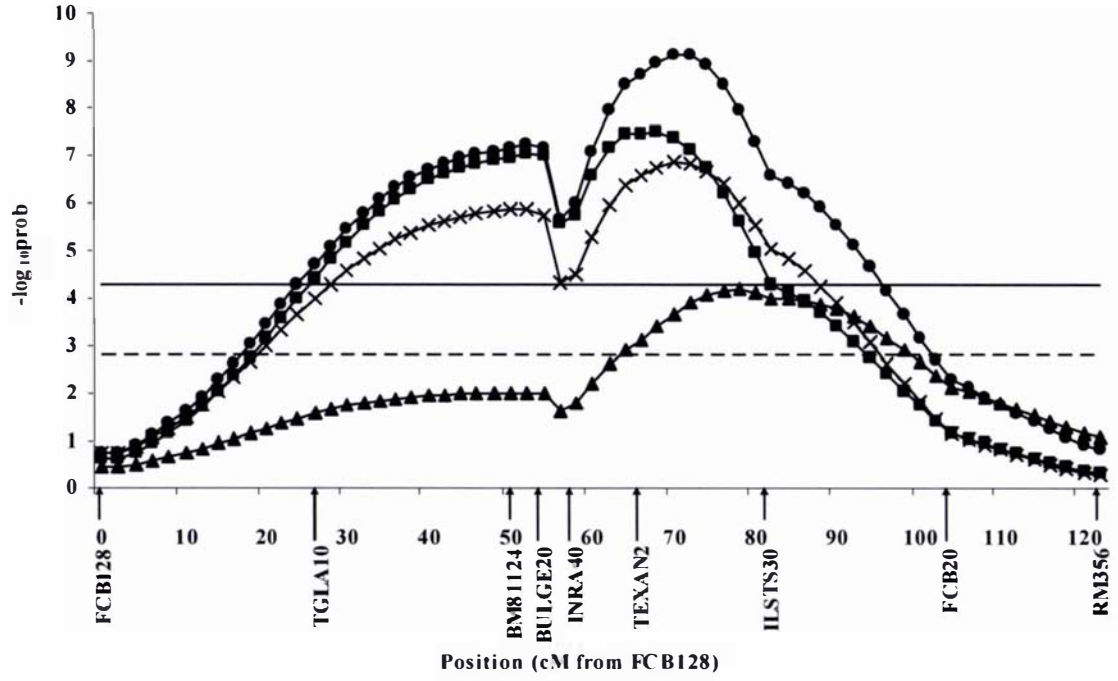


Figure 4.8 –$\log_{10}$prob curves for sire 1199/00 half-sib QTL analysis for a region of ovine chromosome two - leg fat weight traits

Subcutaneous fat in the leg weight ■■■; intermuscular fat in the leg weight ▲▲▲; total fat in the leg weight ◆◆◆; leg fat % ✕✕✕; suggestive threshold - - - -; significance threshold ——

Loin fat traits

Significant peaks existed for soft-tissue depth (GR) and loin subcutaneous fat weight (Table 4.20; Figure 4.9). The position of the peaks lay between 54 and 66cM. Both peaks reached the 95% significance threshold and the effects explained between 0.6 and 0.7 of a SD unit for GR and LLFT, respectively.

Table 4.20 Details of peaks for sire 1199/00 Texel cross half-sib QTL analysis for a region of ovine chromosome two – loin fat traits[1]

Trait	Phen mean ± adj. std	Estimate ± stderr	SD units	Permutation 95%	Permutation 99%	max $-\log_{10}$ prob	pos (cM) [CI]
GR (mm)	4.6 ± 1.8	-1.0 ± 0.3	0.6	2.2	2.9	2.4 *	66 [2 -104]
Fat depth C (mm)	2.6 ± 1.0	-0.3 ± 0.2	0.3	2.2	3.1	0.9 ns	52
Loin subcutaneous fat (g)	71.8 ± 19.4	-13.4 ± 6.0	0.7	1.3	1.7	1.6 *	54 [39 -104]

[1] Descriptions of column headings are given in Table 4.16

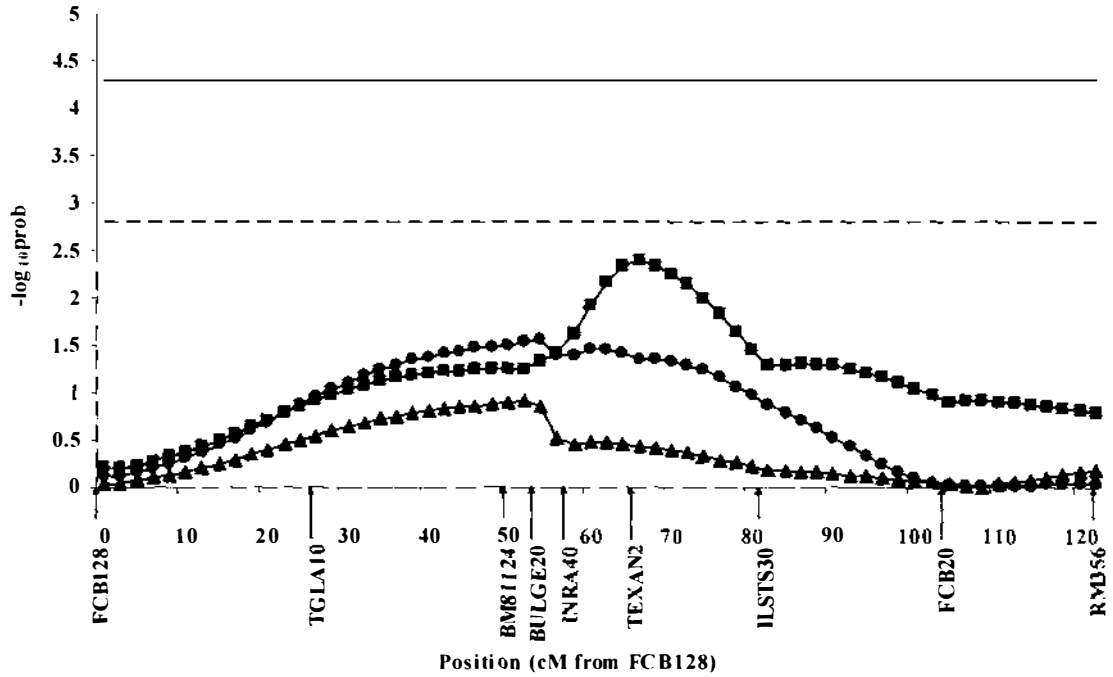


Figure 4.9 –$\log_{10}$prob curves for sire 1199/00 half-sib QTL analysis for a region of ovine chromosome two - loin fat traits

Carcass fat depth (GR) ■■■; works measured *M. longissimus* fat depth (C) ▲▲▲; *M. longissimus* fat weight ◆◆◆; suggestive threshold - - - -; significance threshold ——

Sire 1170/00

No peaks reached the suggestive thresholds for Sire 1170/00 for weight, ultrasound fat, linear or loin fat traits or dressing-out percentage. These results are presented in Appendix 8.5.

Ultrasound muscle traits

Peaks existed for UB and UEMA, at approximately 122cM (Table 4.21; Figure 4.10). Both peaks reached the 95% significance threshold and the peaks explained approximately 0.5 of a SD unit.

Table 4.21 Details of peaks for sire 1170/00 Texel cross half-sib QTL analysis for a region of ovine chromosome two – ultrasound muscle traits[1]

Trait	Phen mean ± adj. std	Estimate ± stderr	SD units	Permutation max 95%	99%	-$\log_{10}$ prob	pos (cM) [CI]
M. longissimus width (mm)	57.3 ± 4.0	1.8 ± 0.7	0.4	1.9	2.7	1.7 ns	122
M. longissimus depth (mm)	20.5 ± 1.9	0.9 ± 0.4	0.5	1.8	2.6	1.8 *	122 [2 - 122]
M. longissimus area (mm^2)	913.7 ± 140.9	69.5 ± 26.9	0.5	1.8	2.5	2.0 *	122 [15 - 122]

[1] Descriptions of column headings are given in Table 4.16

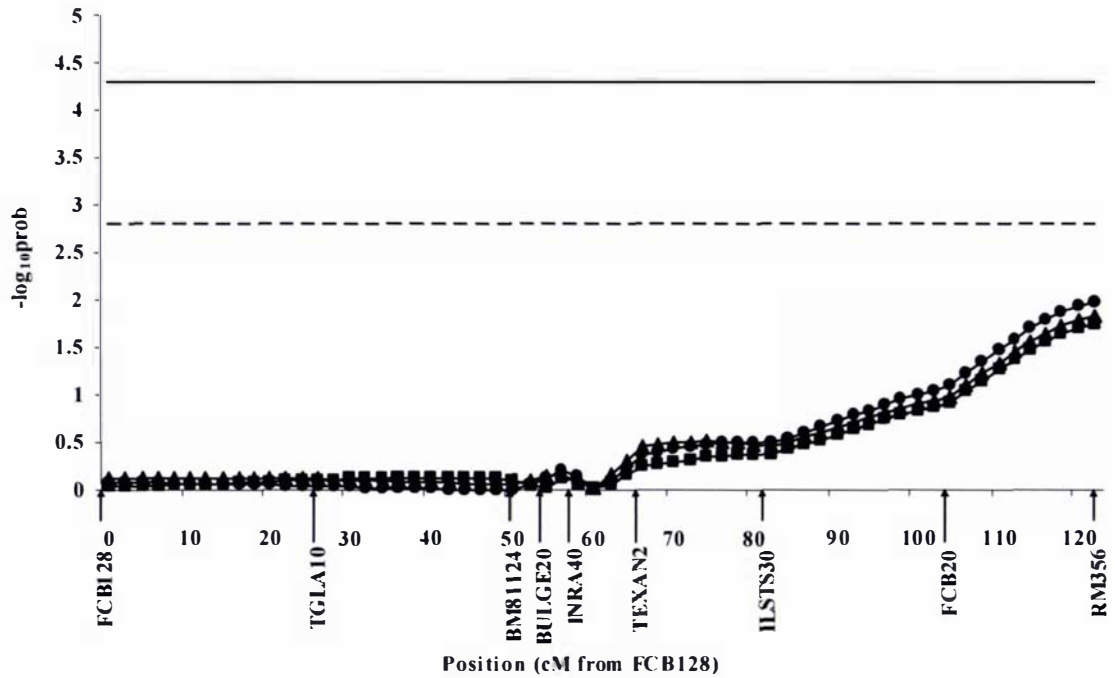


Figure 4.10 –log$_{10}$prob curves for sire 1170/00 half-sib QTL analysis for a region of ovine chromosome two – ultrasound muscle traits

Ultrasound *M. longissimus* width (A) ▪▪▪ ; ultrasound *M. longissimus* depth (B) ▲▲▲; ultrasound *M. longissimus* area ◆◆◆; suggestive threshold - - - -; significance threshold ——

Leg muscle traits

Significant peaks existed for TLG SM BF AD MST and total TLMUS weight (Table 4.22; Figure 4.11). The position of the peaks lay between 52 and 60cM. Both the SM and TLMUS peaks reached the 99% significance threshold, the former also reaching the L&K significance threshold, with the latter reaching their suggestive threshold. Other peaks reached the 95% significance threshold. The effects explained between 0.4 and 0.8 of a SD unit for TLG and SM, respectively.

Significant peaks existed for the other muscle related traits – LGMUSC and LGMS% (Table 4.23; Figure 4.12). The position of the peaks lay between 50 and 60cM. Both peaks reached the 99% significance threshold and reached the L&K suggestive threshold. The effects explained between 0.7 and 0.9 of a SD unit for the LGMS% and LGMUSC respectively.

Table 4.22 Details of peaks for sire 1170/00 Texel cross half-sib QTL analysis for a region of ovine chromosome two – leg muscle weight traits[1]

Trait	Phen mean ● adj. std	Estimate ● stderr	SD units	Permutation 95%	99%	max $-\log_{10}$ prob	pos (cM) [CI]
Trimmed leg (g)	2,561.3 ± 135.0	58.9 ± 19.1	0.4	2.0	2.8	2.6 *	60 [28 - 122]
M. semimembranosus (g)	238.1 ± 17.6	13.7 ± 3.2	0.8	1.9	2.5	4.7 ** sig	54 [28 - 82]
M. semitendinosus (g)	88.9 ± 8.5	2.6 ± 1.7	0.3	1.8	2.6	0.9 ns	54
M. biceps femoris (g)	234.2 ± 18.6	9.7 ± 3.4	0.5	2.1	3.0	2.3 *	52 [15 - 122]
M. quadriceps (g)	336.1 ± 22.2	8.7 ± 4.9	0.4	1.6	2.2	1.1 ns	62
M. adductor (g)	110.7 ± 9.2	5.7 ± 1.9	0.6	1.7	2.4	2.5 **	52 [28 -104]
M. gluteus medius (g)	154.2 ± 15.8	7.3 ± 3.1	0.5	1.8	2.6	1.7 ns	58
Muscle trim (g)	634.5 ± 44.0	23.2 ± 8.0	0.5	1.8	2.5	2.4 *	56 [28 - 122]
Total leg muscle (g)	1,796.8 ± 111.4	67.2 ± 17.5	0.6	2.0	2.8	3.8 ** sug	54 [28 - 122]

[1] Descriptions of column headings are given in Table 4.16

Table 4.23 Details of peaks for sire 1170/00 Texel cross half-sib QTL analysis for a region of ovine chromosome two – other muscle related traits[1]

Trait	Phen mean ● adj. std	Estimate ± stderr	SD units	Permutation 95%	99%	max $-\log_{10}$ prob	pos (cM) [CI]
Leg muscularity	0.4 ± 0.02	0.02 ± 0.004	0.9	2.0	2.9	3.9 ** sug	60 [30 - 122]
Leg muscle percent	70.1 ± 1.8	1.3 ± 0.4	0.7	1.7	2.3	3.0 ** sug	50 [28 - 82]
Femur muscle to bone	4.5 ± 0.3	0.1 ± 0.1	0.4	1.7	2.3	1.3 ns	54

[1] Descriptions of column headings are given in Table 4.16

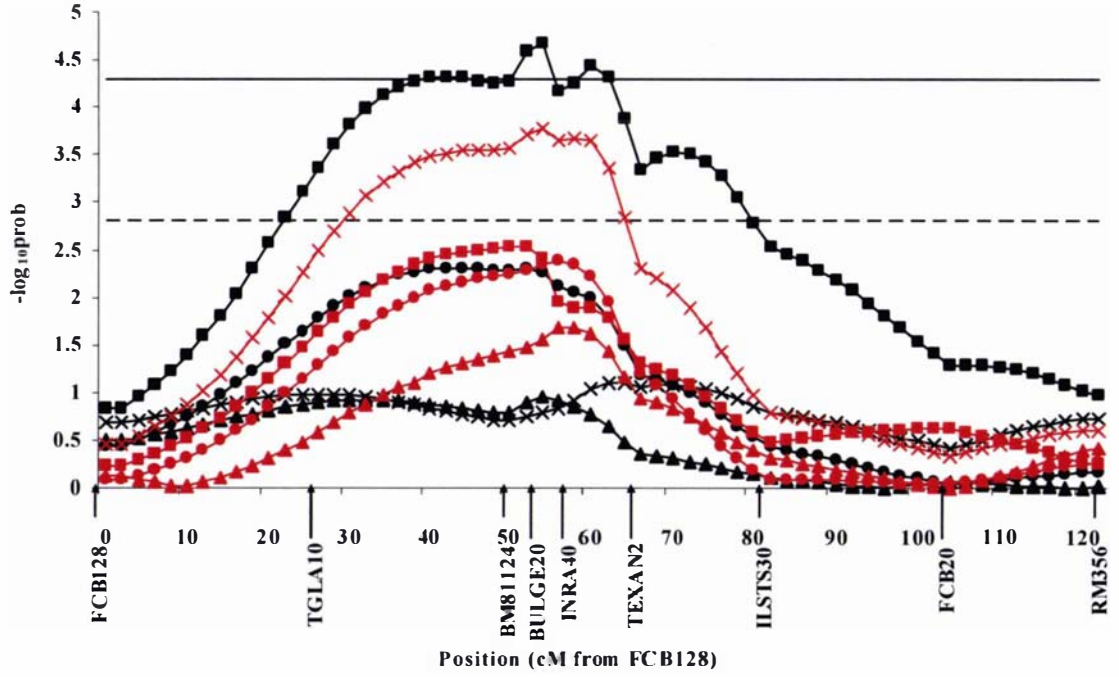


Figure 4.11–$\log_{10}$prob curves for sire 1170/00 half-sib QTL analysis for a region of ovine chromosome two – weights of leg muscles

M. semimembranosus weight ■■■; *M. semitendinosus* weight ▲▲▲; *M. biceps femoris* weight ◆◆◆ ; *M. quadriceps* weight ✱✱✱; *M. adductor* weight ●●● ; *M. gluteus medius* weight ▲▲▲ ; muscle trim weight ◆◆◆total muscle in the leg weight ✱✱✱; suggestive threshold - - - -; significance threshold ——

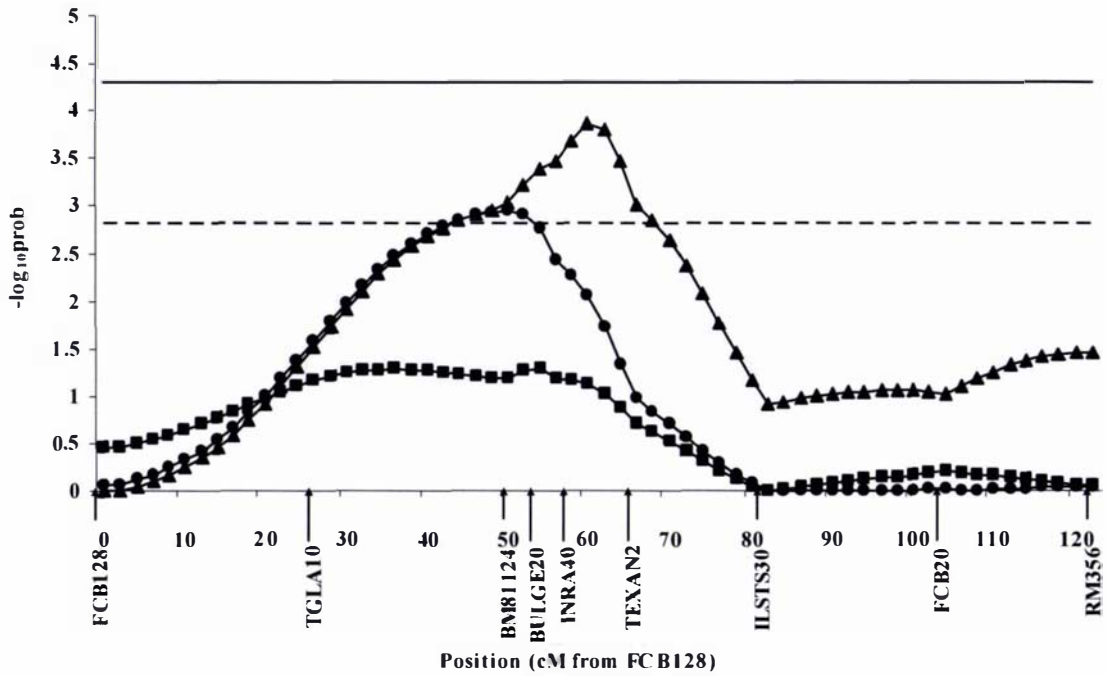


Figure 4.12 –log$_{10}$prob curves for sire 1170/00 half-sib QTL analysis for a region of ovine chromosome two – other muscle related traits

Femur muscle to bone ■■■; leg muscularity ▲▲▲; leg muscle percent ◆◆◆; suggestive threshold - - -; significance threshold ——

Loin muscle traits

A significant peak existed only for A at 38cM. The peak reached the 95% significance threshold and explained approximately 0.6 of a SD unit. Further details of this peak are in Appendix 8.5.

Leg fat traits

There were significant peaks for subcutaneous and total fat weight at approximately 50cM (Table 4.24; Figure 4.13). Both of these peaks reached the 99% significance threshold, but do not reached the L&K suggestive threshold. The effect explained approximately 0.6 of a SD unit.

Table 4.24 Details of peaks for sire 1170/00 Texel cross half-sib QTL analysis for a region of ovine chromosome two – leg fat weight traits[1]

Trait	Phen mean ± adj. std	Estimate ± stderr	SD units	Permutation 95%	Permutation 99%	max -log$_{10}$ prob	pos (cM) [CI]
Subcutaneous fat (g)	208.1 ● 36.6	-21.2 ● 7.1	0.6	1.5	2.1	2.5 **	48 [28 - 104]
Intermuscular fat (g)	134.0 ± 24.6	-5.6 ± 5.1	0.2	1.8	2.5	0.6 ^{ns}	50
Total leg fat (g)	342.1 ± 48.6	-26.0 ± 9.1	0.5	1.5	2.2	2.3 **	50 [28 -104]
Leg fat %	13.4 ± 1.9	-1.1 ● 0.4	0.6	2.3	3.0	2.2 ^{ns}	50 [2 – 122]

[1] Descriptions of column headings are given in Table 4.16

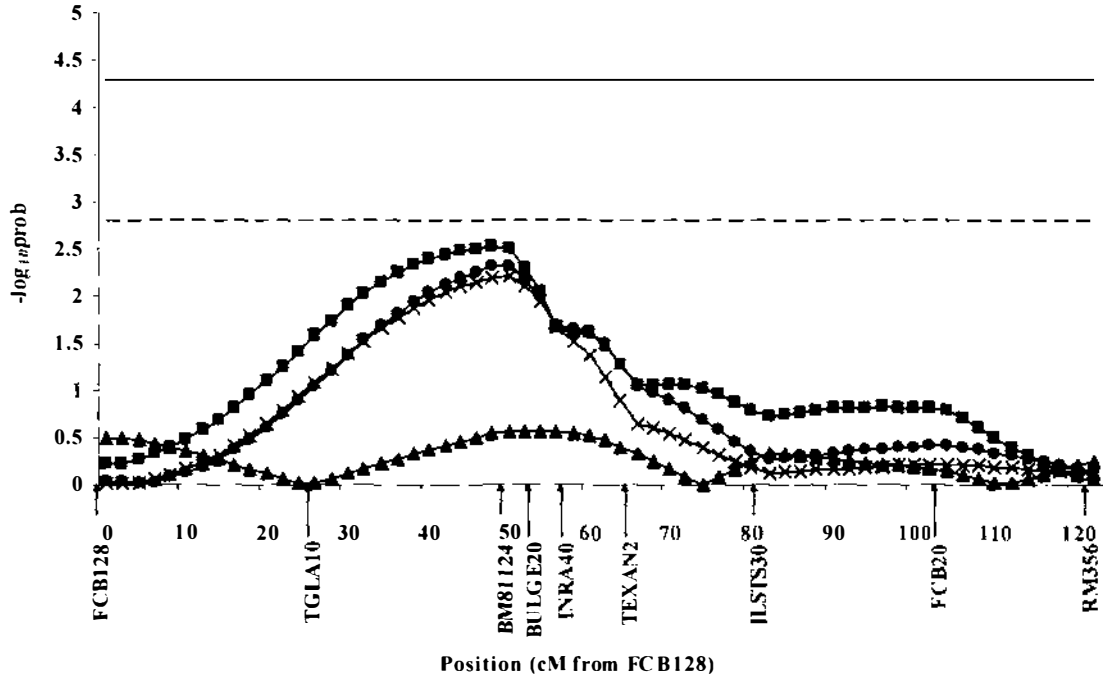


Figure 4.13 $-\log_{10}$prob curves for sire 1170/00 half-sib QTL analysis for a region of ovine chromosome two - leg fat weight traits

Subcutaneous fat in the leg weight ■■■; intermuscular fat in the leg weight ▲▲▲; total fat in the leg weight ◆◆◆; leg fat % ✕✕✕; suggestive threshold - - - -; significance threshold ——

Quality traits

A significant peak for the quality trait WBYFSM existed at 14cM. The peak reached the 95% significance threshold and the effect explained approximately 0.8 of a SD unit. Further details of this peak are in Appendix 8.5.

Sire 150/96

Only one peak existed for sire 150/96, a peak for AD at 96cM. The peak reached the 95% significance level and the effect explained approximately 0.7 of a SD unit. Further details of this peak and peaks for all other traits for sire 150/96 are in Appendix 8.6.

Sire 15/98

No significant peaks were detected for Sire 15/98 for weight, ultrasound fat or meat quality traits. These results are presented in Appendix 8.7.

Carcass linear traits

Of the carcass linear traits a peak only existed for G at approximately 34cM. The peak reached the 95% significance threshold and the effect explained 0.5 of a SD unit. Further details of the peak are given in Appendix 8.7.

Dressing-out percentage

A significant peak for dressing-out percentage existed at 72cM. The peak reached the 99% significance threshold and explained approximately 0.3 of a SD unit. Further details of the peak are given in Appendix 8.7.

Ultrasound muscle traits

All muscle-related ultrasound traits showed evidence of peaks (Table 4.25 ; Figure 4.14) between 54 and 66cM. All peaks reached the 99% significance threshold and the effects explained approximately 0.5 of a SD unit.

Table 4.25 Details of peaks for sire 15/98 Texel cross half-sib QTL analysis for a region of ovine chromosome two – ultrasound muscle traits[1]

Trait	Phen mean ± adj. std	Estimate ± stderr	SD units	Permutation 95%	Permutation 99%	max $-\log_{10}$ prob	pos (cM) [CI]
M. longissimus width (mm)	58.9 ± 5.1	2.3 ● 0.7	0.5	2.0	2.6	2.8 **	66 [52 - 104]
M. longissimus depth (mm)	23.0 ± 2.3	1.1 ● 0.4	0.5	1.9	2.6	2.8 **	54 [39 - 104]
M. longissimus area (mm^2)	1,058.1 ± 170.2	92.5 ● 25.7	0.5	2.0	2.6	3.4 ** sug	62 [50 - 104]

[1] Descriptions of column headings are given in Table 4.16

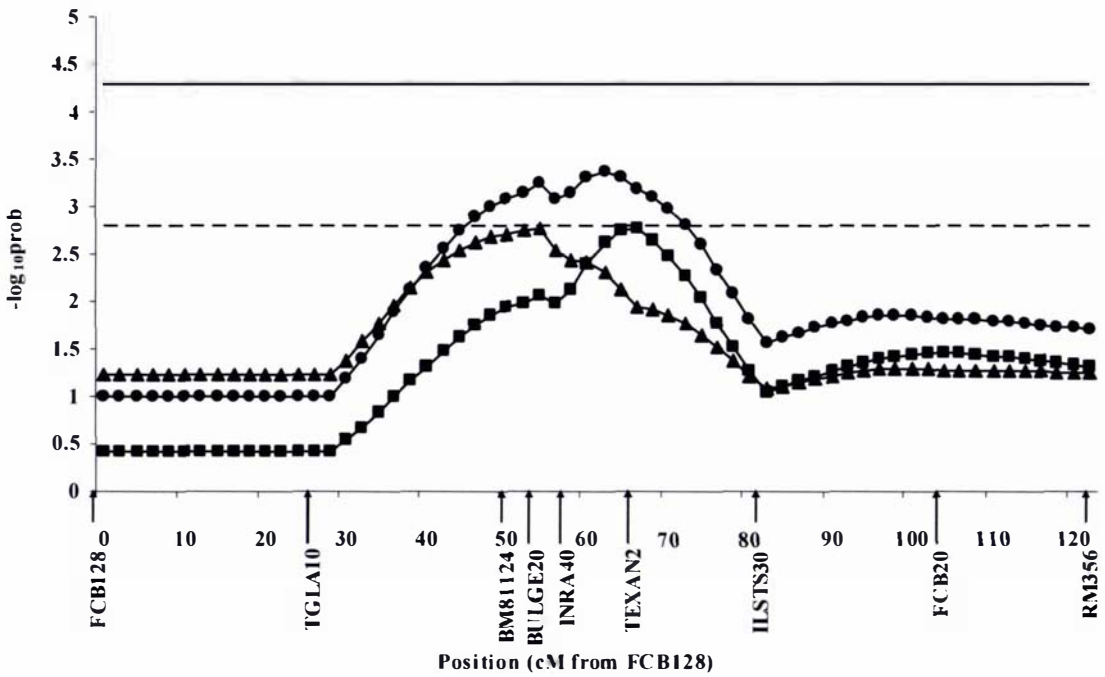


Figure 4.14 –$\log_{10}$prob curves for sire 15/98 half-sib QTL analysis for a region of ovine chromosome two – ultrasound traits

Ultrasound *M. longissimus* width (A) ■■■ ; ultrasound *M. longissimus* depth (B) ▲▲▲; ultrasound *M. longissimus* area ◆◆◆; suggestive threshold - - - -; significance threshold ——

Leg muscle traits

Peaks existed for all leg muscle weights except GM weight (Table 4.26; Figure 4.15). The position of the peaks lay between 34 and 56cM. The peaks for TLG, SM, ST, QD,

MST, and TLMS reached the 99% significance threshold, although only QD and TLMS reached the L&K significance threshold, with the remainder reaching the L&K suggestive threshold. The peaks for BF and AD reached the 95% significance threshold. The effects ranged between 0.4 and 0.7 SD units, for TLG and QD respectively.

Peaks existed for all other muscle-related traits (Table 4.27; Figure 4.16). The peaks lay between 52 and 62cM. The peaks for LGMUSC and LGMS% reached the 99% significance threshold and the L&K suggestive threshold, whilst MTB reached the 95% significance threshold. The effects were around 0.8 SD unit.

Table 4.26 Details of peaks for sire 15/98 Texel cross half-sib QTL analysis for a region of ovine chromosome two – leg muscle weight traits[1]

Trait	Phen mean ± adj. std	Estimate ± stderr	SD units	Permutation 95%	Permutation 99%	max $-\log_{10}$ prob	pos (cM) [CI]
Trimmed leg (g)	2,691.6 ± 170.5	73.5 ± 21.3	0.4	1.9	2.7	3.1 ** sug	40 [20 -104]
M. semimembranosus (g)	249.7 ± 19.8	11.5 ± 3.3	0.6	1.9	2.7	3.2 ** sug	56 [40 -122]
M. semitendinosus (g)	92.9 ± 9.2	6.0 ± 1.7	0.7	1.9	2.7	3.1 ** sug	46 [29 - 93]
M. biceps femoris (g)	243.2 ± 22.9	12.1 ± 4.4	0.5	1.9	2.7	2.2 *	34 [2 - 104]
M. quadriceps (g)	376.7 ± 29.4	21.8 ± 4.9	0.7	1.9	2.7	4.5 ** sig	46 [39 - 104]
M. adductor (g)	123.4 ± 12.2	7.9 ± 2.7	0.7	1.8	2.4	2.4 *	42 [2 - 82]
M. gluteus medius (g)	161.8 ± 15.9	4.7 ± 2.9	0.3	1.9	2.7	0.9 ns	56
Muscle trim (g)	706.1 ± 55.2	32.5 ● 8.7	0.6	1.9	2.7	3.5 ** sug	52 [39 - 93]
Total leg muscle (g)	1,953.7 ± 138.7	90.1 ± 18.5	0.7	1.9	2.7	5.0 ** sig	52 [39 - 104]

[1] Descriptions of column headings are given in Table 4.16

Table 4.27 Details of peaks for sire 15/98 Texel cross half-sib QTL analysis for a region of ovine chromosome two – other muscle related traits[1]

Trait	Phen mean ● adj. std	Estimate ± stderr	SD units	Permutation 95%	Permutation 99%	max $-\log_{10}$ prob	pos (cM) [CI]
Leg muscularity	0.5 ● 0.02	0.01 ± 0.003	0.8	1.9	2.7	3.6 ** sig	62 [2 -82]
Leg muscle percent	72.6 ± 1.8	1.6 ± 0.4	0.9	1.9	2.7	4.2 ** sug	52 [39 - 82]
Femur muscle to bone	4.9 ± 0.4	0.3 ± 0.1	0.7	1.9	2.7	2.6 *	52 [39 - 82]

[1] Descriptions of column headings are given in Table 4.16

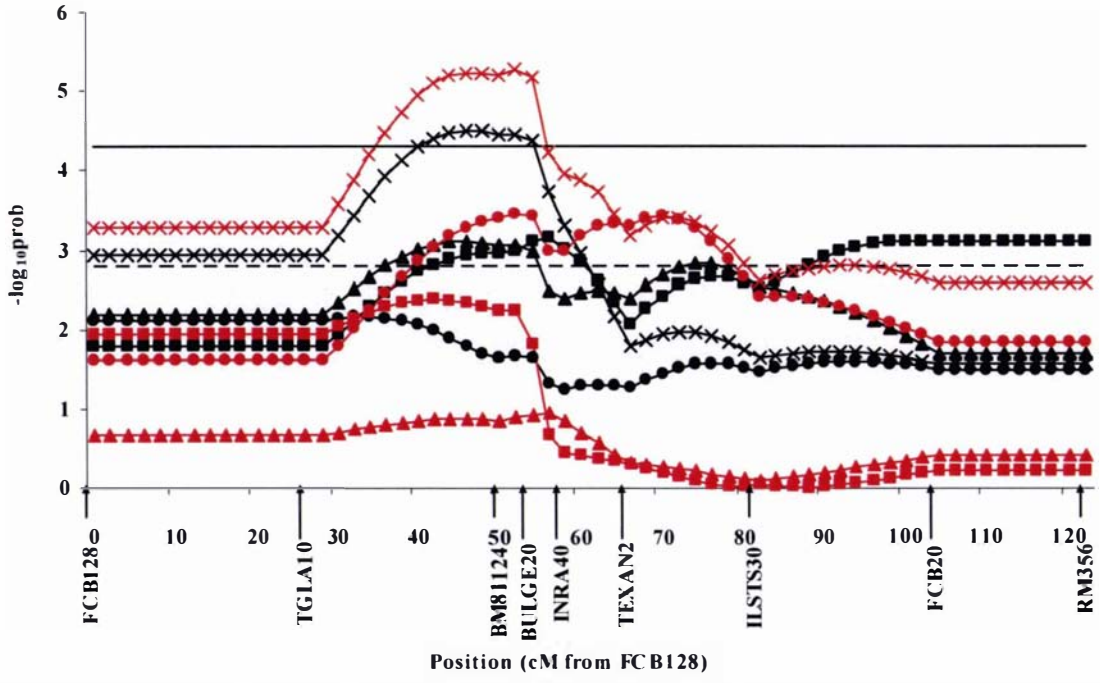


Figure 4.15 –log$_{10}$prob curves for sire 15/98 half-sib QTL analysis for a region of ovine chromosome two – weights of leg muscles

M. semimembranosus weight ■■■; *M. semitendinosus* weight ▲▲▲; *M. biceps femoris* weight ◆◆◆ ; *M. quadriceps* weight ✱✱✱; *M. adductor* weight ●●● ; *M. gluteus medius* weight ▲▲▲ ; muscle trim weight ◆◆total muscle in the leg weight ✱✱✱; suggestive threshold - - - -; significance threshold ——

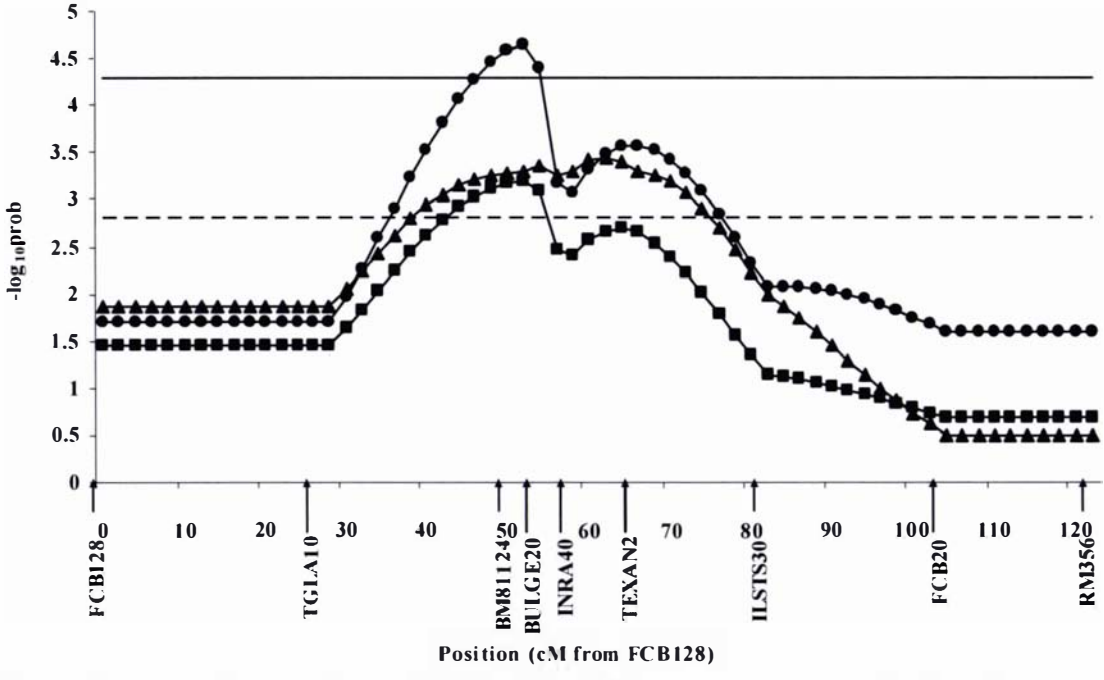


Figure 4.16 –log$_{10}$prob curves for sire 15/98 half-sib QTL analysis for a region of ovine chromosome two – other muscle related traits

Femur muscle to bone ■■■; leg muscularity ▲▲▲; leg muscle percent ◆◆◆ ; suggestive threshold - - -; significance threshold ——

Loin muscle traits

For the loin muscle traits, a significant peak only existed for LL weight at

approximately 66cM. The peak reached the 99% significance threshold and L&K suggestive threshold. The effect explained approximately 0.6 of a SD unit. Further details of the peak are in Appendix 8.7.

Leg fat traits

Peaks existed for SCF and TLFT (Table 4.28; Figure 4.17). The position of the peaks lay between 68 and 74cM. The peak for TLFT reached the 99% significance threshold and the L&K suggestive threshold, whist the peak for SCF reached the 95% significance threshold. The effects were approximately 0.7 of a SD unit.

Table 4.28 Details of peaks for sire 15/98 Texel cross half-sib QTL analysis for a region of ovine chromosome two – leg fat weight traits[1]

Trait	Phen mean ± adj. std	Estimate ± stderr	SD units	Permutation max 95%	99%	$-\log_{10}$ prob	pos (cM) [CI]
Subcutaneous fat (g)	194.9 ± 37.1	-24.5 ± 7.6	0.7	2.0	2.8	2.7 *	74
Intermuscular fat (g)	122.5 ± 24.7	-12.4 ± 5.7	0.5	1.9	2.7	1.5 ^{ns}	66
Total leg fat (g)	317.4 ± 50.3	-34.9 ± 10.2	0.7	2.0	2.7	3.0 ** ^{sug}	68 [50 - 93]
Leg fat %	11.8 ± 1.7	-1.0 ± 0.4	0.6	2.4	3.2	1.8 ^{ns}	70 [39 – 104]

[1] Descriptions of column headings are given in Table 4.16

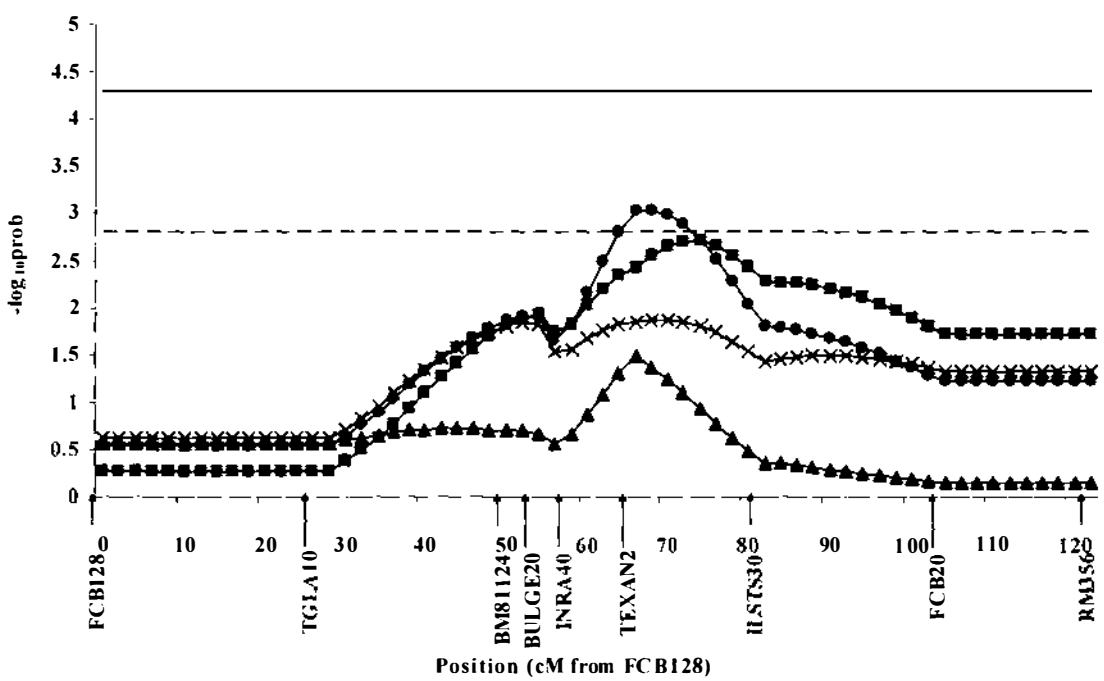


Figure 4.17 $-\log_{10}$prob curves for sire 15/98 half-sib QTL analysis for a region of ovine chromosome two - leg fat weight traits

Subcutaneous fat in the leg weight ■■■; intermuscular fat in the leg weight ▲▲▲; total fat in the leg weight ◆◆◆; leg fat % ××××; suggestive threshold - - - -; significance threshold ——

Longissimus fat traits

Of the loin fat traits, a peak only existed for C at approximately 62cM. The peak reached the 95% significance threshold. The effect explained approximately 0.5 of a

SD unit. Further details of the peak are in Appendix 8.7.

Sire 122/99

Only two peaks existed for sire 122/99, a peak for A at 96cM and a peak for DO at 76cM. The peak for A reached the 95% significance threshold and the effect explained approximately 0.8 of a SD unit. The peak for DO reached the 95% significance threshold and the L&K suggestive threshold, the effect explained 1.0 of a SD unit. Further details of this peak and peaks for all other traits for sire 122/99 are in Appendix 8.8.

It must be remembered that this sire was not evaluated for any of the dissection or meat quality traits.

Sire 429/98

No significant QTL were detected for Sire 429/98 for ultrasound muscle or loin muscle traits, or dressing-out percentage. These results are presented in Appendix 8.9.

Live-weight traits

A peak existed for W at approximately 48cM. The peak reached the 95% significance threshold and the effect explained 0.4 of a SD unit. Further details of the peak are in Appendix 8.9.

Carcass linear traits

A peak existed for WF at approximately 42cM. The peak reached the 95% significance threshold and the effect explained 0.5 of a SD unit. Further details of the peak are in Appendix 8.9.

Dressing-out percentage

A significant peak for dressing-out percentage existed at 96cM. The peak reached the 95% significance threshold and explained approximately 0.3 of a SD unit. Further details of the peak are given in Appendix 8.9.

Leg muscle traits

Peaks existed for SM, ST, AD and TLMUS (Table 4.29; Figure 4.18). The peaks lay

between 50 and 54cM. The peaks for AD and TLMS reached the 99% significance threshold and the L& K suggestive threshold, whilst the others only reached the 95% significance threshold. The effects were 0.5 and 0.7 SD units for SMWT and ADWT, respectively. Note that the Q allele for this sire is associated with negative estimates, that is the Qq genotype is poorer than the qq genotype.

Significant peaks existed for all other muscle related traits (Table 4.30; Figure 4.19). The peaks lay between 36 and 52cM. The peaks for LGMS% and MTB reached the 99% significance threshold and L&K significance threshold. The peak for LGMSC reached the 95% significance threshold. The effects were 0.7 and 1.2 SD units for LGMSC and LGMS%, respectively. Note that the Q allele for this sire is again associated with negative estimates.

Table 4.29 Details of peaks for sire 429/98 Texel cross half-sib QTL analysis for a region of ovine chromosome two – leg muscle traits[1]

Trait	Phen mean ● adj. std	Estimate ± stderr	SD units	Permutation 95%	Permutation 99%	max $-\log_{10}$ prob	pos (cM) [CI]
Trimmed leg (g)	2,693.1 ± 154.0	-27.6 ± 23.7	0.2	1.4	1.9	0.6 ^{ns}	54
M. semimembranosus (g)	262.9 ± 19.9	-9.9 ± 3.7	0.5	1.8	2.5	2.1 *	54 [29 -122]
M. semitendinosus (g)	93.5 ± 9.6	-5.2 ± 1.8	0.6	2.1	2.8	2.3 *	54 [34 -104]
M. biceps femoris (g)	248.3 ± 18.8	-7.8 ± 3.6	0.4	1.8	2.4	1.5 ^{ns}	54
M. quadriceps (g)	372.6 ± 24.9	-10.4 ± 4.4	0.4	1.8	2.4	1.7 ^{ns}	54
M. adductor (g)	124.1 ± 11.3	-7.7 ± 2.2	0.7	2.0	2.8	3.3 ** ^{sug}	50 [39 - 122]
M. gluteus medius (g)	165.3 ± 16.6	-4.5 ± 4.5	0.3	1.9	2.5	0.5 ^{ns}	26
Muscle trim (g)	670.0 ± 49.9	-22.8 ● 9.1	0.5	1.9	2.5	1.9 ^{ns}	54
Total leg muscle (g)	1,936.1 ± 119.1	-66.6 ± 19.1	0.6	1.8	2.4	3.2 ** ^{sug}	54 [34 - 58]

[1] Descriptions of column headings are given in Table 4.16

Table 4.30 Details of peaks for sire 429/98 Texel cross half-sib QTL analysis for a region of ovine chromosome two – other muscle related traits[1]

Trait	Phen mean ± adj. std	Estimate ± stderr	SD units	Permutation 95%	Permutation 99%	max $-\log_{10}$ prob	pos (cM) [CI]
Leg muscularity	0.5 ± 0.02	-0.01 ± 0.004	0.7	2.2	3.0	2.6 *	52 [29 - 104]
Leg muscle percent	71.9 ± 1.8	-2.1 ± 0.4	1.2	2.2	3.0	5.3 ^{**sig}	40 [15 - 57]
Femur muscle to bone	4.8 ± 0.3	-0.3 ± 0.1	1.2	2.1	2.8	4.5 ** ^{sig}	36 [15 - 74]

[1] Descriptions of column headings are given in Table 4.16

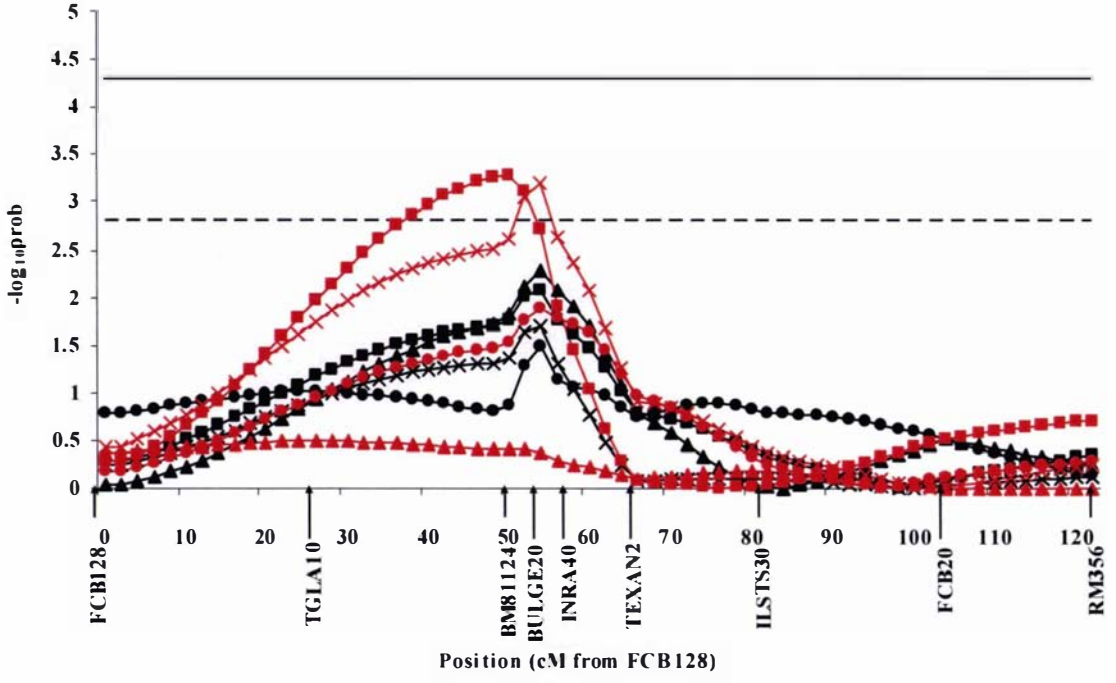


Figure 4.18 –log$_{10}$prob curves for sire 429/98 half-sib QTL analysis for a region of ovine chromosome two – weights of leg muscles

M. semimembranosus weight ■■■; *M. semitendinosus* weight ▲▲▲; *M. biceps femoris* weight ◆◆◆ ; *M. quadriceps* weight ✱✱✱; *M. adductor* weight ●●● ; *M. gluteus medius* weight ▲▲▲ ; muscle trim weight ◆◆◆total muscle in the leg weight ✱✱✱; suggestive threshold - - - -; significance threshold ——

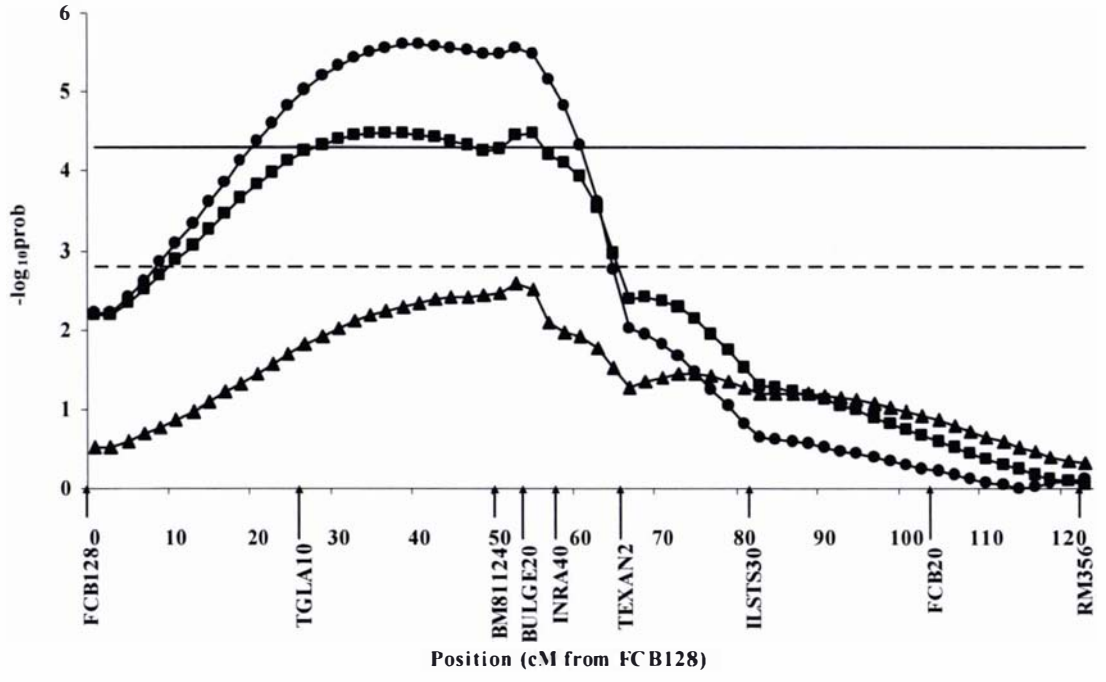


Figure 4.19 –log$_{10}$prob curves for sire 429/98 half-sib QTL analysis for a region of ovine chromosome two – other muscle related traits

Femur muscle to bone ■■■; leg muscularity ▲▲▲ ; leg muscle percent ◆◆◆ ; suggestive threshold - - -; significance threshold ——

Ultrasound fat traits

A peak for UC existed at approximately 64cM. The peak reached the 95% significance threshold and the effect explained 0.5 of the adjusted standard deviation. Further details of the peak are in Appendix 8.9.

Loin fat traits

A significant peak only existed for LLFT weight at 28cM. The peak reached the 95% significance level and the L&K suggestive threshold. The effect explained 0.9 of a SD unit. Further details of the peak are given in Appendix 8.9. Note that the Q allele for this sire is associated with positive estimates.

Leg fat traits

Significant peaks existed for all leg fat weight traits with the exception of IMF weight (Table 4.31; Figure 4.20). The peaks lay between 38 and 46cM and reached the 99% significance threshold and the L&K suggestive thresholds. The effects were between 0.7 and 0.9 SD units for TLFT and SCF/LGFT%, respectively. Note that the Q allele for this sire is associated with positive estimates, that is more fat.

Table 4.31 Details of peaks for sire 429/98 Texel cross half-sib QTL analysis for a region of ovine chromosome two – leg fat traits[1]

Trait	Phen mean ● adj. std	Estimate ± stderr	SD units	Permutation 95%	Permutation 99%	max $-\log_{10}$ prob	pos (cM) [CI]
Subcutaneous fat (g)	203.4 ± 41.2	36.0 ± 9.6	0.9	2.0	2.8	3.6 ** sug	38 [15 - 70]
Intermuscular fat (g)	125.8 ± 23.1	8.5 ± 4.6	0.4	2.1	2.9	1.2 ns	50
Total leg fat (g)	329.2 ± 53.6	39.7 ± 10.6	0.7	2.1	2.8	3.6 ** sug	46 [29 - 62]
Leg fat %	12.2 ± 1.8	1.7 ± 0.4	0.9	2.1	2.5	4.1 ** sug	40 [15 – 82]

[1] Descriptions of column headings are given in Table 4.16

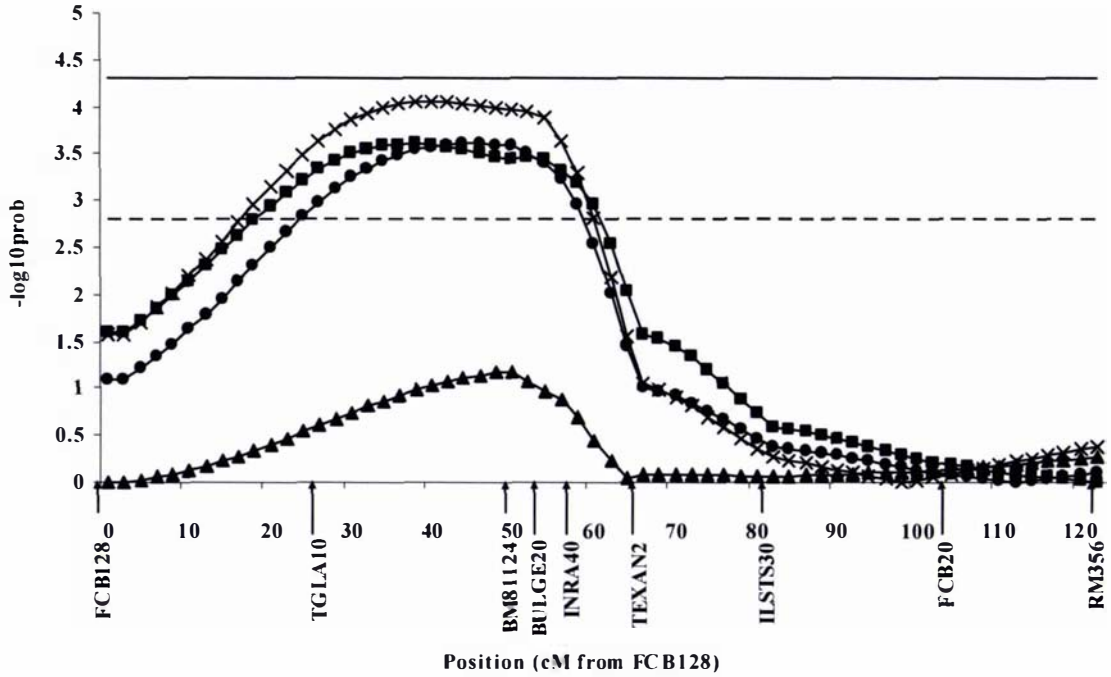


Figure 4.20 $-\log_{10}$prob curves for sire 429/98 half-sib QTL analysis for a region of ovine chromosome two - leg fat weight traits

Subcutaneous fat in the leg weight ■■■; intermuscular fat in the leg weight ▲▲▲; total fat in the leg weight ◆◆◆; leg fat % ✱✱✱; suggestive threshold - - - -; significance threshold ——

Quality traits

Significant peaks existed for pHSM, WBPKSM, WBYFSM, CLSM, CASM, CBSM, CLLL, CALL and CBLL (Table 4.32; Figures 4.21 and 4.22). The position of the peaks lay between 56 and 122cM. All peaks reached the 95% significance threshold. The effects explained approximately 0.6 of a SD unit.

Table 4.32 Details of peaks for sire 429/98 Texel cross half-sib QTL analysis for a region of ovine chromosome two – *M. semimembranosus* and *M. longissimus* meat quality traits[1]

Trait	Phen mean ± adj. std	Estimate ± stderr	SD units	Permutation 95%	Permutation 99%	max $-\log_{10}$ prob	pos (cM) [CI]
M. semimembranosus							
pH	5.6 ± 0.1	0.1 ± 0.0	0.6	2.1	2.8	2.0 *	120 [2 – 122]
Colour "L*"	31.8 ± 1.4	0.8 ± 0.3	0.6	2.1	2.8	2.0 *	56 [29 - 122]
Colour "a*"	14.5 ± 1.1	-0.7 ± 0.3	0.7	2.0	2.7	2.3 *	110 [2 - 122]
Colour "b*"	6.2 ± 0.6	-0.4 ± 0.2	0.6	2.1	2.8	2.0 *	108 [15 -122]
Sarcomere lng. (µm)	1.5 ± 0.6	0.2 ± 0.2	0.4	2.0	2.8	1.0 ns	82
Cooking loss (%)	37.0 ± 1.3	-0.7 ± 0.3	0.5	2.1	2.8	1.5 ns	62
W-B peak force	12.0 ± 2.6	1.6 ± 0.7	0.6	1.6	2.1	1.7 *	122 [28 - 122]
W-B yield force	9.2 ± 2.5	1.4 ± 0.6	0.6	1.6	2.2	1.7 *	122 [28 - 122]
M. longissimus							
pH	5.6 ± 0.1	0.1 ± 0.0	0.5	1.9	2.5	1.7 ns	122
Colour "L*"	34.1 ± 1.7	1.0 ± 0.4	0.6	1.8	2.4	2.0 *	56 [9 - 104]
Colour "a*"	14.5 ± 1.6	-0.9 ± 0.3	0.6	2.3	3.2	2.6 *	122 [15 -122]
Colour "b*"	6.5 ± 0.9	-0.5 ± 0.2	0.6	2.0	2.8	2.0 *	122 [15 -122]
Sarcomere lng. (µm)	1.8 ± 0.1	0.0 ± 0.0	0.4	1.9	2.5	0.7 ns	104
Cooking loss (%)	30.6 ± 1.5	-0.6 ± 0.4	0.4	2.1	2.9	0.9 ns	96
W-B peak force	6.9 ± 1.6	0.8 ± 0.4	0.5	1.7	2.3	1.5 ns	122
W-B yield force	5.6 ± 1.2	0.4 ± 0.3	0.4	1.8	2.5	0.9 ns	122

[1] Descriptions of column headings are given in Table 4.16

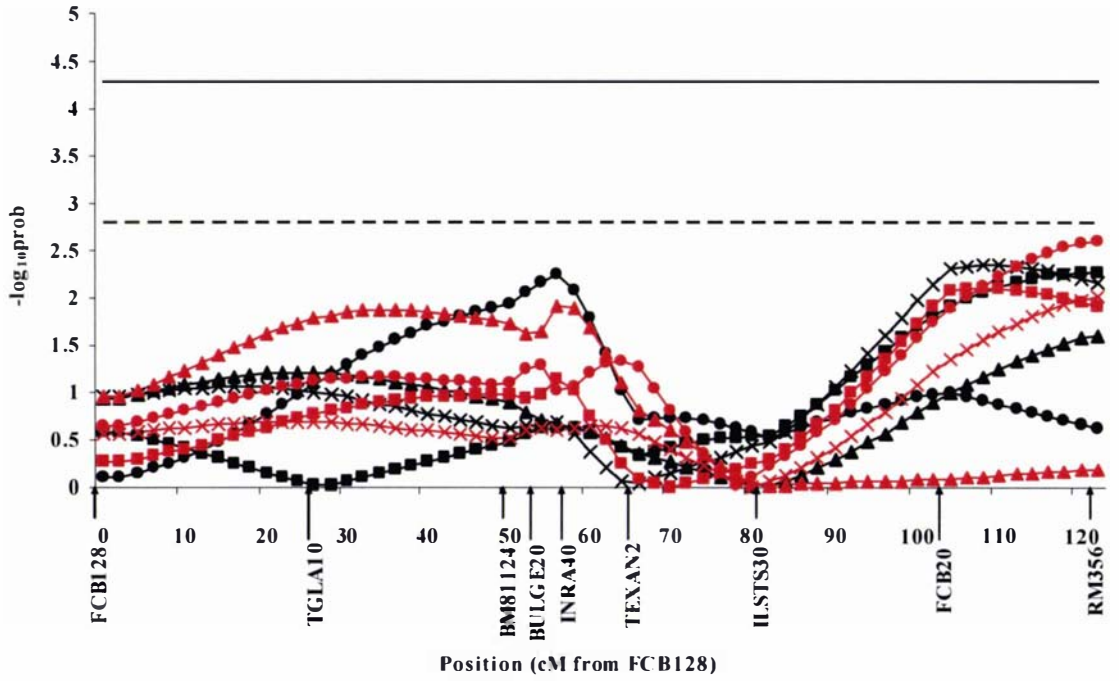


Figure 4.21 –log$_{10}$prob curves for sire 429/98 half-sib QTL analysis for a region of ovine chromosome two - quality traits

pH *M. semimembranosus* ■■■; pH *M. longissimus* ▲▲▲; colour "L*" *M. semimembranosus* ◆◆◆ ; colour "a*" *M. semimembranosus* ✱✱✱; colour "b*" *M. semimembranosus* ●●●; colour "L*" *M. longissimus* ▲▲▲; colour "a*" *M. longissimus* ◆◆◆ ; colour "b*" *M. longissimus* ✱✱✱; suggestive threshold ----; significance threshold ——

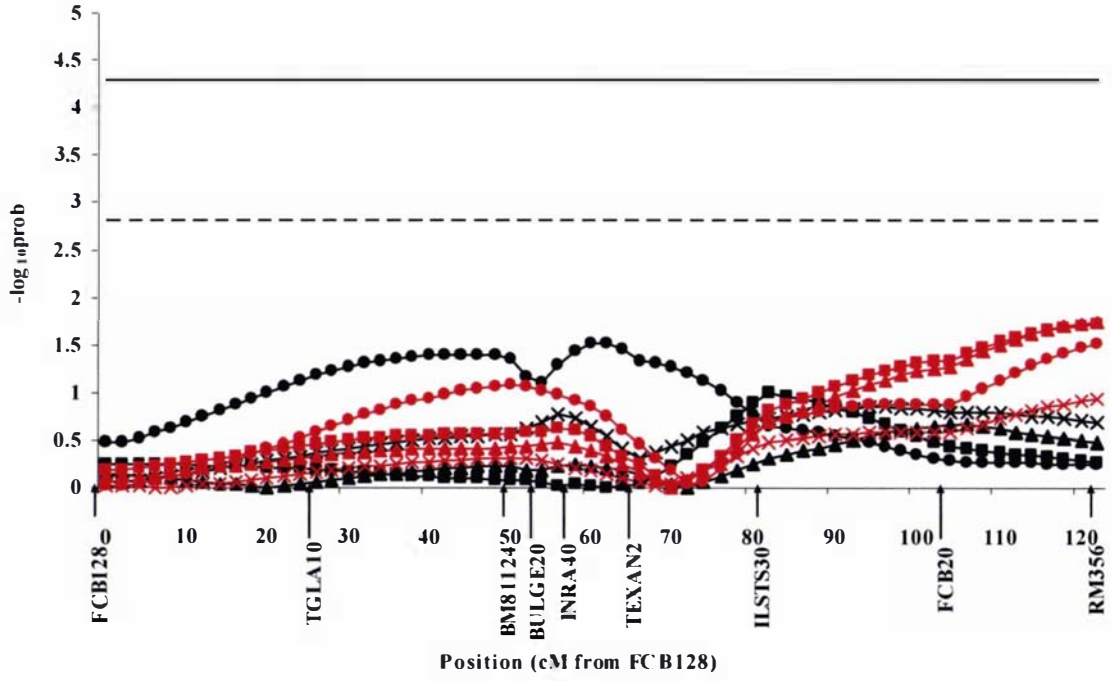


Figure 4.22 –log$_{10}$prob curves for sire 429/98 half-sib QTL analysis for a region of ovine chromosome two - quality traits

Sarcomere length *M. semimembranosus* ■■■; sarcomere length *M. longissimus* ▲▲▲; cooking loss (%) *M. semi.* ◆◆◆ ; cooking loss (%) *M. long.* ✱✱✱; Warner-Bratzler peak force *M. semi.* ●●●; Warner-Bratzler yield force *M. semi..* ▲▲▲; Warner-Bratzler peak force *M. long.* ◆◆◆ ; Warner-Bratzler yield force *M. long.* ✱✱✱; suggestive threshold ----; significance threshold ——

Sire 535/98

No significant peaks were detected for sire 535/98. Details of the peaks for this sire are presented in Appendix 8.10.

4.3.7 Multi-Trait Analysis

Multi-trait analysis was carried out for trait groups. For the principal component analysis, only results from the analysis of the first principal component in each analysis are presented below, as analysis of the second and third components produced non-significant results. Only the graphs derived from the multivariate analysis are presented, as the graphs derived from the principal component analysis were similar. Weighting factors for each trait within a group are presented in Appendix 8.11.

Muscle weights in the leg

The following traits were included in the multi-trait analysis of leg muscle weight traits: SM, ST, BF, QD, AD, GM and MST.

Using this approach, significant peaks existed for muscle traits in the leg for sires 429/98, 1170/00, 1199/00 and 15/98 using principal component analysis, but only for the latter three when multivariate analysis was used (Table 4.33). The positions of the peaks were between 52-54cM. In the graphical representation there appears to be a second significant peak for sire 1199/00, at around 70cM (Figure 4.23).

Table 4.33 Details of QTL peaks for half-sib Texel cross population for a region of ovine chromosome two - muscle in leg (as determined by multi-trait analysis of weight of *M. semimembranosus*, *M. semitendinosus*, *M. biceps femoris*, *M. quadriceps*, *M. adductor* and *M. gluteus medius*)[1]

	Principal Component		Multivariate	
Sire Number	**-log$_{10}$prob**	**Position (cM)**	**-log$_{10}$prob**	**Position (cM)**
1170/00	3.9^{sug}	54	2.5	52
1199/00	5.0^{sig}	54	5.5^{sig}	54
150/96	0.8	104	0.8	114
15/98	5.9^{sig}	52	4.5^{sig}	52
429/98	3.2^{sug}	54	1.8	52
535/98	0.5	116	0.8	80

[1] Descriptions of column headings are given in Table 4.16; note permutation tests were not carried out for these traits

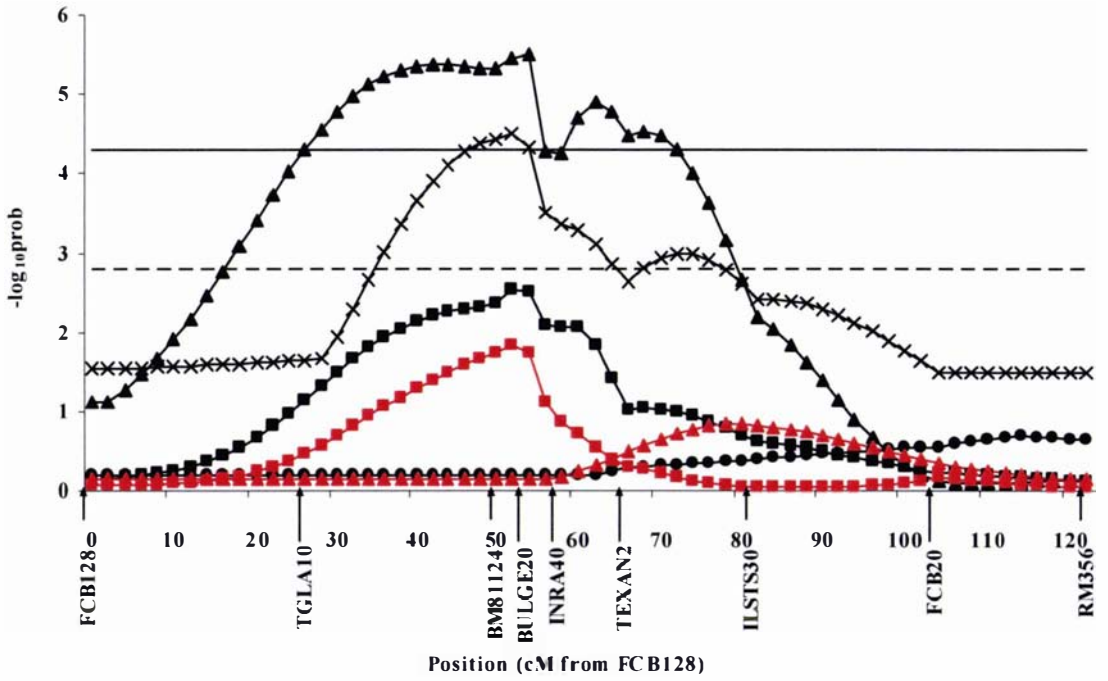


Figure 4.23 –log$_{10}$prob curves for half-sib Texel cross population for a region of ovine chromosome two - muscle in leg (as determined by multivariate analysis of weight of *M. semimembranosus*, *M. semitendinosus*, *M. biceps femoris*, *M. quadriceps*, *M. adductor* and *M. gluteus medius*)

1170/00 ■■■ ; 1199/00 ▲▲▲; 150/96 ◆◆◆; 15/98 ✱✱✱; 429/98 ●●● ; 535/98 ▲▲▲; suggestive threshold - - -; significance threshold —

Muscle measurements in the loin

The following traits were included in the multi-trait analysis of loin muscle traits: EMA, A, B and LL.

Significant peaks existed for muscle measurements on the loin for sires 1199/00 and 15/98 using principal component analysis, but fail to reach the threshold when multivariate analysis was used (Table 4.34). The position of the peak was at 52-54cM. In the graphical representation (Figure 4.24) there appears to be a second significant peak for sire 15/98, at around 65cM.

Table 4.34 Details of QTL peaks for half-sib Texel cross population for a region of ovine chromosome two - muscle traits in the loin (as determined by multi-trait analysis of weight of *M. longissimus* linear dimensions of A and B and *M. longissimus* area)[1]

	Principal Component		Multivariate	
Sire Number	**-log$_{10}$prob**	**Position (cM)**	**-log$_{10}$prob**	**Position (cM)**
1170/00	1.1	82	0.1	122
1199/00	3.6^{sug}	54	2.3	54
150/96	0.4	104	0.7	48
15/98	2.5^{sug}	52	1.8	66
429/98	1.0	50	1.3	48
535/98	0.5	50	0.1	66

[1] Descriptions of column headings are given in Table 4.16

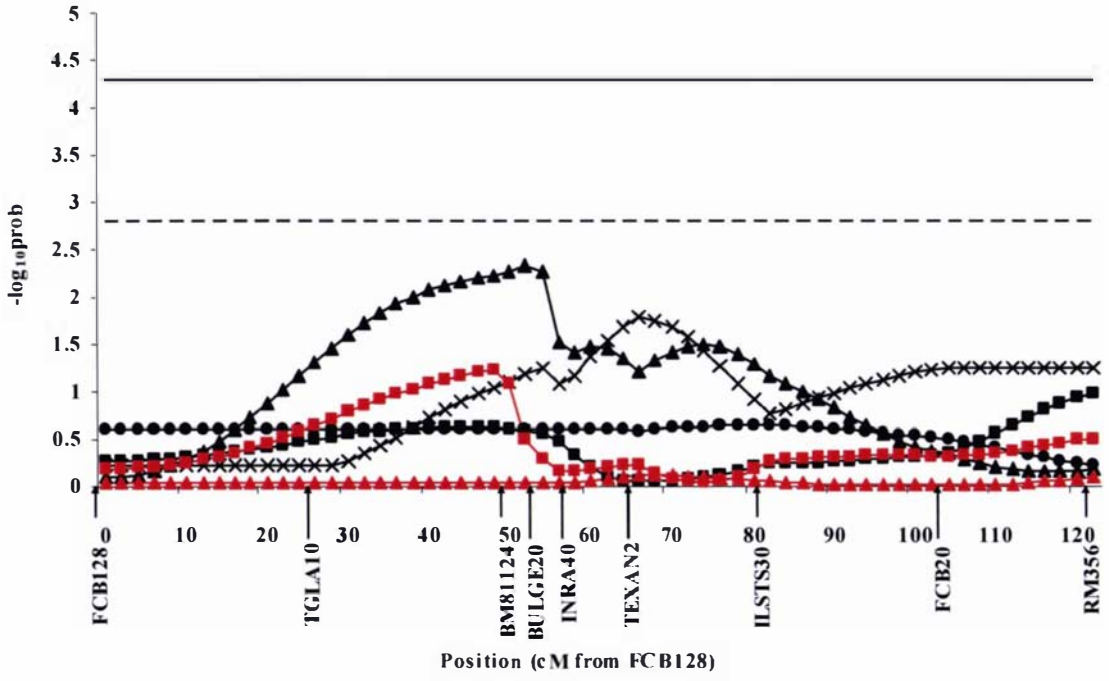


Figure 4.24 –log$_{10}$prob curves for half-sib Texel cross population for a region of ovine chromosome two - muscle traits in the loin (as determined by multivariate analysis of weight of *M. longissimus*, linear dimensions of A and B and *M. longissimus* area)

1170/00 ■■■ ; 1199/00 ▲▲▲; 150/96 ◆◆◆; 15/98 ✱✱✱; 429/98 ■■■ ; 535/98 ▲▲▲; suggestive threshold - - -; significance threshold —

Fat weights in the leg

The SCF and IMF traits were included in the multi-trait analysis of leg fat weight traits.

Significant peaks exited for traits in the leg for sires 429/98 and 1199/00 when using both multi-trait analysis methods, there is a peak for 15/98 which approaches the threshold (Table 4.35; Figure 4.25). Graphically there appears to be two groups of peaks at 48-54cM and 68-72cM.

Table 4.35 Details of QTL peaks for half-sib Texel cross population for a region of ovine chromosome two - fat percentage in leg (as determined by multi-trait analysis of weight of leg subcutaneous and intermuscular fat)

	Principal Component		Multivariate	
Sire Number	**-log$_{10}$prob**	**Position (cM)**	**-log$_{10}$prob**	**Position (cM)**
1170/00	2.0	50	1.7	48
1199/00	8.0^{sig}	72	6.7^{sig}	72
15/98	2.7	68	2.4	68
150/96	0.7	106	0.3	112
429/98	3.2^{sug}	48	3.8^{sug}	52
535/98	1.0	66	1.9	64

[1] Descriptions of column headings are given in Table 4.16

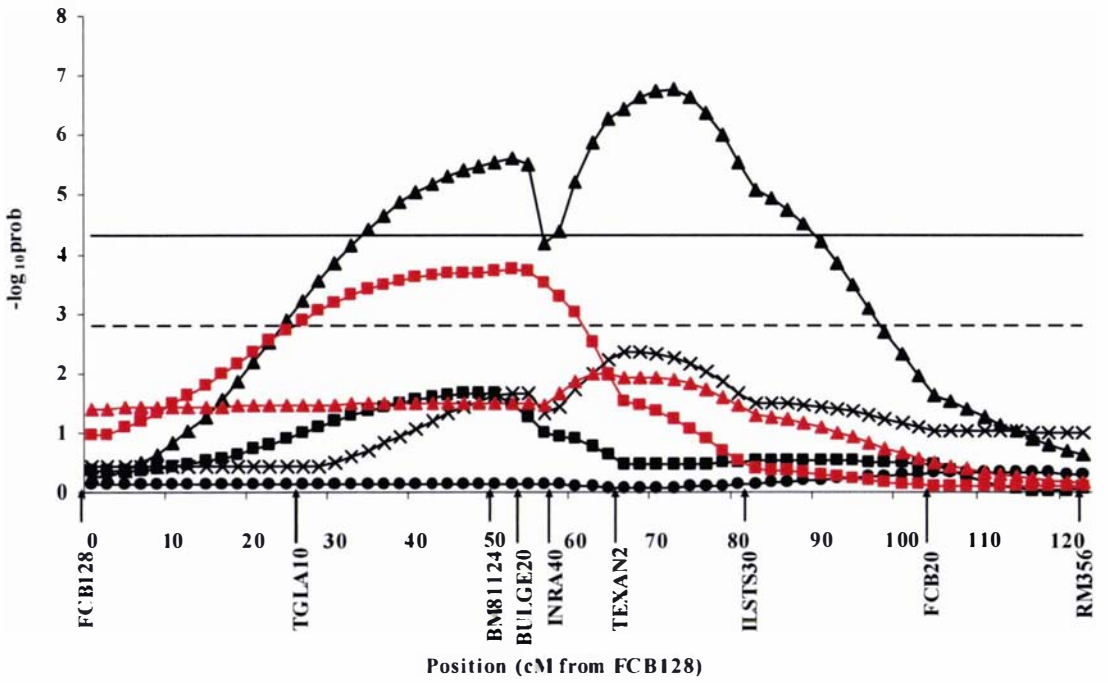


Figure 4.25 –$\log_{10}$prob curves for half-sib Texel cross population for a region of ovine chromosome two - fat percentage in leg (as determined by multivariate analysis of weight of leg subcutaneous and intermuscular fat)

1170/00 ■■■ ; 1199/00 ▲▲▲; 150/96 ◆◆◆; 15/98 ✱✱✱; 429/98 ■■■ ; 535/98 ▲▲▲; suggestive threshold - - -; significance threshold —

Fat measurements in the loin

The following traits were included in the multi-trait analysis of loin fat traits: LLFT, GR and C.

Significant peaks existed for loin fat traits for sires 1199/00 and 429/98 using principal component analysis, but fail to reach the threshold when multivariate analysis was used (Table 4.36).

Table 4.36 Details of QTL peaks for half-sib Texel cross population for a region of ovine chromosome two - fat traits in loin (as determined by multi-trait analysis of weight of loin subcutaneous fat weight, the linear dimensions of fat depth over loin and GR)[1]

	Principal Component		Multivariate	
Sire Number	**-$\log_{10}$prob**	**Position (cM)**	**-$\log_{10}$prob**	**Position (cM)**
1170/00	2.0	44	1.2	42
1199/00	3.8^{sug}	64	2.3	70
150/96	0.4	122	1.0	54
15/98	1.3	74	1.2	60
429/98	3.7^{sug}	32	2.0	56
535/98	0.5	112	1.7	94

[1] Descriptions of column headings are given in Table 4.16

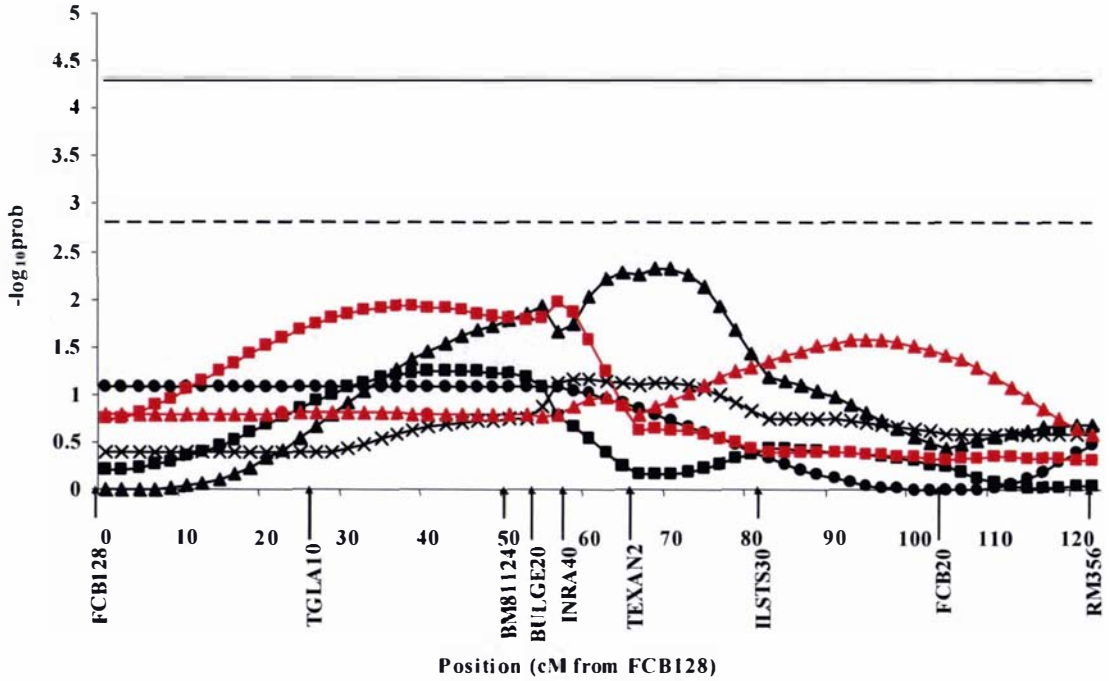


Figure 4.26 –log$_{10}$prob curves for half-sib Texel cross population for a region of ovine chromosome two - fat traits in loin (as determined by multivariate analysis of weight of loin subcutaneous fat weight, the linear dimensions of fat depth over *M. longissimus* and GR)

1170/00 ■■■ ; 1199/00 ▲▲▲; 150/96 ◆◆◆; 15/98 ✱✱✱; 429/98 ■■■ ; 535/98 ▲▲▲; suggestive threshold - - -; significance threshold —

Combined leg muscle and fat analysis by sire

Multivariate analysis was used to analyse muscle and fat traits within the leg. Analyses were carried out using the sum of leg muscle and fat weight traits (weight traits); and the percentage muscle and fat in the leg traits (percentage traits).

Significant combined muscle/fat peaks existed for sires 1170/00, 1199/00, 15/98 and 429/98 (Table 4.37). For these sires, the position of the peak lay at 50-52cM, although for sire 1199/00 the most significant peak lay at 68-70cM (Figure 4.27 and Figure 4.28).

Table 4.37 Details of QTL peaks for half-sib Texel cross population for a region of ovine chromosome two - combined leg (determined by multi-trait analysis of the sum of leg muscle and fat weight traits (weight traits); and percentage muscle and fat in the leg traits (percentage traits))[1]

	Weight Traits		Percentage Traits	
Sire Number	**-log$_{10}$prob**	**Position (cM)**	**-log$_{10}$prob**	**Position (cM)**
1170/00	3.0^{sug}	52	2.5	50
1199/00	8.5^{sig}	70	11.6^{sig}	68
150/96	1.2	104	0.3	108
15/98	4.0^{sug}	52	4.4^{sig}	52
429/98	4.0^{sug}	52	4.4^{sig}	52
535/98	1.3	62	0.6	70

[1] Descriptions of column headings are given in Table 4.16

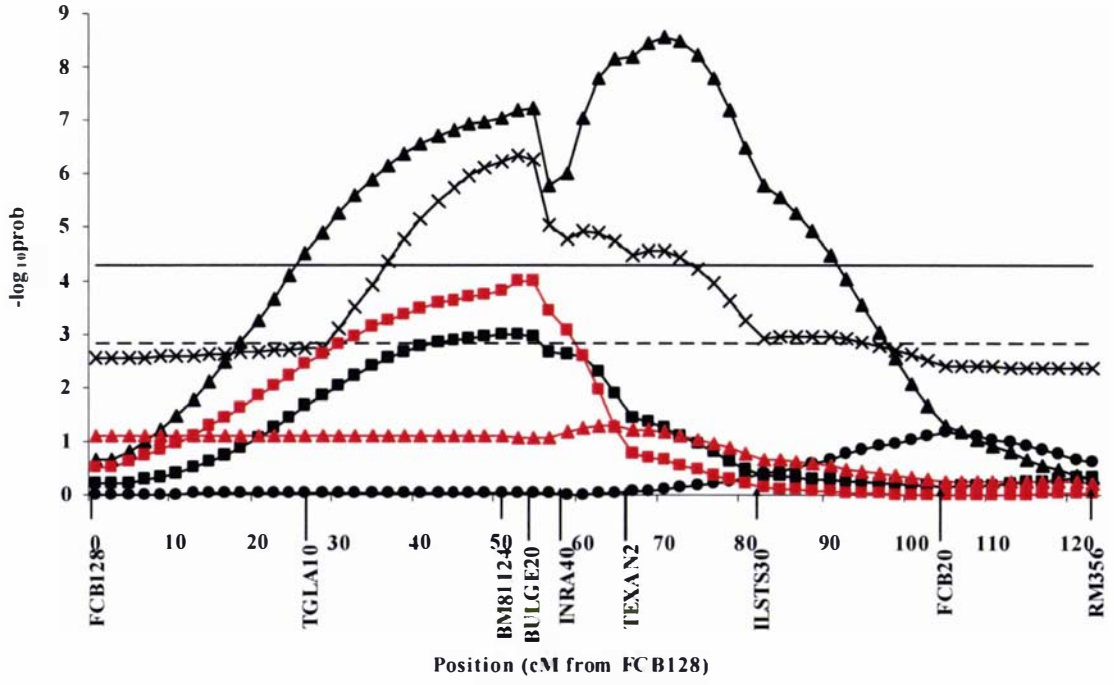


Figure 4.27 –$\log_{10}$prob curves for half-sib Texel cross population for a region of ovine chromosome two - combined leg (as determined by multivariate analysis of the sum of leg muscle weights and the sum of leg fat weight traits)

1170/00 ■■■ ; 1199/00 ▲▲▲; 150/96 ◆◆◆; 15/98 ✱✱✱; 429/98 ●●● ; 535/98 ▲▲▲; suggestive threshold - - -; significance threshold ––

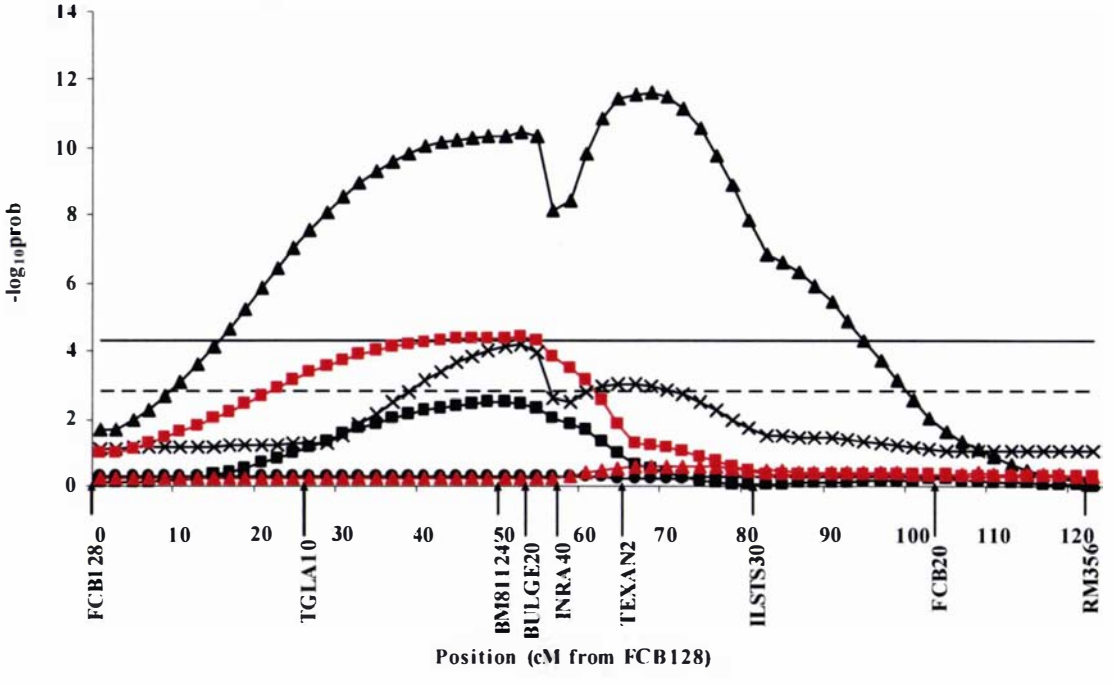


Figure 4.28 –$\log_{10}$prob curves for half-sib Texel cross population for a region of ovine chromosome two - combined leg (as determined by multivariate analysis of the percentage muscle in the leg and the percentage fat in the leg traits)

1170/00 ■■■ ; 1199/00 ▲▲▲; 150/96 ◆◆◆; 15/98 ✱✱✱; 429/98 ●●● ; 535/98 ▲▲▲; suggestive threshold - - -; significance threshold —

Combined leg muscle and fat analysis across sire

The final analysis was an across-sire multi-trait analysis. The same two analyses that were carried out in the last section were used, but the data were combined across sires with a flock effect fitted. The analyses resulted in $-\log_{10}$prob values of 14.0 and 14.5 for the weight and percentage traits respectively, and both mapped to 52cM. As can be seen from the graphical representation (Figure 4.29), even when all sires are analysed together there remains evidence for a second peak

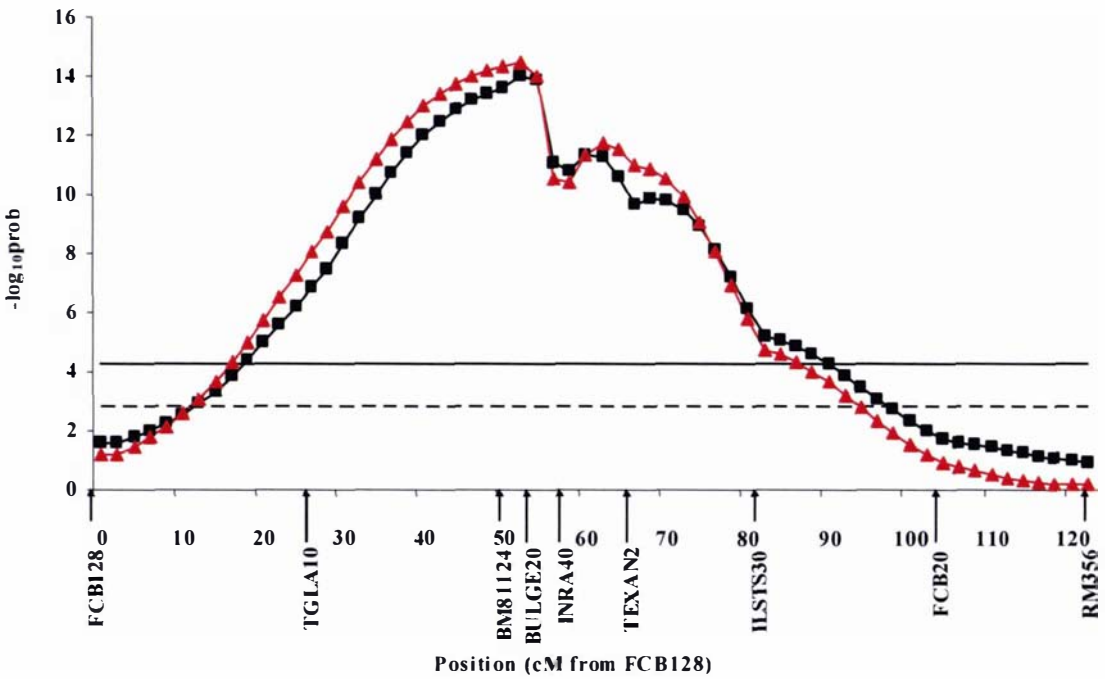


Figure 4.29 $-\log_{10}$prob curves for half-sib Texel cross population for a region of ovine chromosome two - combined leg (as determined by multivariate analysis of the percentage muscle in the leg and the percentage fat in the leg traits; and as determined by multivariate analysis of the sum of leg muscle weights and the sum of fat weight traits)

Weight traits ■■■; Percentage traits ▲▲▲; suggestive threshold - - -; significance threshold —

4.3.8 Two-Peak Analysis

Given strong graphical evidence for two-peaks for sire 1199/00, and some evidence for sire 15/98, two-peak analyses were carried out for these sires for a selection of traits that had shown graphical evidence for two peaks. The traits analysed were SM, TLMS, LGMS%, SCF, TLFT and LGFT%.

For sire 1199/00 there is evidence ($P<0.05$) for two independent peaks within the region of interest (no epistasis) (Table 4.38). The first peaks mapping to 50-52cM, and the second to 70-74cM, positions which are consistent with the graphical evidence.

For sire 15/98, although there is evidence for two peaks, with an epistatic interaction within the region (Table 4.38), they are not conclusive. For SM, the first peak maps to 52cM, whilst the second maps to 122cM which is at the boundary of the marked region of the genome used this study. For LGMS% and LGFT%, the positions of the two peaks lie within eight and 14cM respectively of each other.

Table 4.38 Details of two peak QTL analysis for half-sib Texel cross population for a region of ovine chromosome two

			Position 1		Position 2			
Sire	Model[1]	Trait	(cM)	Estimate ± Stderr[2]	(cM)	Estimate ± Stderr[2]	Sig.[3]	Epi. Effect[4]
1199/00	E + D	*M. semimembranosus* (g)	52	12.7 ± 3.7	78	6.7 ± 4.3	ns	
	Dir	Total leg muscle (g)	40	64.2 ± 23.6	72	54.5 ± 22.8	*	
	Dir	Leg muscle percent	50	1.4 ± 0.5	70	2.0 ± 0.6	*	
	Dir	Subcutaneous fat (g)	52	-22.4 ± 9.3	66	-22.7 ± 10.3	*	
	Dir	Total leg fat (g)	52	-27.1 ± 11.3	74	-44.9 ± 13.5	*	
	Dir	Leg fat percent	50	-1.1 ± 0.5	74	-1.4 ± 0.7	*	
15/98	E + D	*M. semimembranosus* (g)	52	22.2 ± 5.8	122	29.4 ± 8.3	**	-27.3 ± 10.3
	Dir	Total leg muscle (g)	52	87.1 ± 19.6	112	33.0 ± 22.6	ns	
	E + D	Leg muscle percent	46	9.4 ± 4.0	54	6.6 ± 2.7	*	-14.3 ± 5.1
	E + D	Subcutaneous fat (g)	56	22.7 ± 14.9	70	33.0 ± 29.0	ns	
	E + D	Total leg fat (g)	56	38.7 ± 20.5	70	27.8 ± 39.9	ns	
	E + D	Leg fat percent	32	-2.4 ± 1.4	46	-3.6 ● 1.0	*	5.0 ± 1.6

[1] Model fitted included: E + D – test for two peaks with epistasis; Dir – test for two unique peaks
[2] Estimate of the size of the QTL effect at each peak ± the standard error
[3] Significance of the two versus one QTL peak test; * is P<0.05; ** is P<0.01
[4] When an E + D two peak test was significant, the size of the epistatic effect is reported

4.3.9 Age at Slaughter as a Covariate

All post-slaughter data were re-analysed using age at slaughter as a covariate, in place of carcass weight. The results gained were very similar to those obtained when carcass weight was used. These data are not presented, as given the slaughter criterion of fixed weight, it was most appropriate to have adjusted the data for carcass weight.

4.3.10 Linkage Disequilibrium Analysis

The first linkage disequilibrium (LD) analysis involved fitting a model solely considering dam LD effects. When this model was fitted there was no evidence of QTL. This implies that the QTL are not segregating within the dam populations used.

In a separate analysis, analysis of sire alleles included adjustment for the effects of LD from the dams. Fitting the LD effect increased the significance threshold achieved in only five flock/trait/marker combinations (Table 4.39). Non-significant results are

presented in Appendix 8.12. For Tanbar the increase in $-\log_{10}p$ values for SM and BF, raised the significance to the L&K suggestive threshold, whilst for MST the increase in $-\log_{10}p$ values raised it from reaching the suggestive to the significant L&K threshold. For Skye, the increase in $-\log_{10}p$ values for LGMUSC raised it from the suggestive to the significant L&K threshold. For Woodlands, the increase in $-\log_{10}p$ value for AD raised the significance to the L&K suggestive threshold.

Table 4.39 Details of QTL results where adjustment for linkage disequilibrium (LD) increased the significance threshold of the peaks. Analysed by marker, by flock for a region of ovine chromosome two

		QTL Analysis			QTL Analysis Adjusted for LD		
		Marker	$-\log_{10}P$	sug/sig[1]	Marker	$-\log_{10}P$	sug/sig[1]
Tanbar	*M. semimembranosus* weight (g)	BULGE20	2.6		BULGE20	3.1	sug
	M. biceps femoris weight (g)	BULGE20	2.6		BM81124	3.1	sug
	Muscle trim weight (g)	BULGE20	3.2	sug	INRA40	4.2	sig
Skye	Leg muscularity	TEXAN2	3.4	sug	TEXAN2	4.7	sig
Woodlands	*M. adductor* weight (g)	BM81124	2.3		BULGE20	3.2	sug

[1]The significance threshold defined in Table 4.16

4.3.11 Sex-QTL Interaction Analysis

There were 41 instances where fitting a sex-interaction in the QTL analysis (Q+I) resulted in significant peaks (L & K suggestive threshold). However, the actual sex-interaction (I) was only significant for seven of these (Table 4.40). Non-significant results are presented in Appendix 8.13. For sire 15/98 peaks were detected in ewe lambs but not ram lambs for UB and UEMA. Peaks were detected in ram lambs but not ewe lambs for WC, SCF and ST. A graphical example of sex differences in QTL peaks for Sire 15/98 is in Figure 4.30.

Table 4.40 Details of QTL peaks for half-sib Texel cross population for a region of ovine chromosome two analysed by fitting a QTL by sex interaction[1].

			Ewe		Ram			
		Pos. (cM)	$-\log_{10}$ prob	estimate ± stderr	$-\log_{10}$ prob	estimate ± stderr	Sex[2]	Sig Int.[3]
15/98	US *M. longissimus* width (mm)	54	3.2^{sug}	2.1 ± 0.6	0.5	0.6 ± 0.6	E	P<0.05
	US *M. longissimus* area (mm^2)	60	4.0^{sug}	175.2 ± 42.1	0.4	36.3 ± 39.3	E	P<0.05
	Loin subcutaneous fat depth (mm)	54	0.2	0.1 ± 0.3	4.0^{sug}	-1.0 ± 0.24	R	P<0.01
	Leg subcutaneous fat weight (g)	54	0.1	3.6 ± 10.4	4.0^{sug}	-37.8 ± 9.4	R	P<0.01
	M. semitendinosus weight (g)	72	0.7	1.1 ± 2.4	4.0^{sug}	9.8 ± 2.3	R	P<0.01
429/98	*M. adductor* weight (g)	50	3.5^{sug}	-11.56 ± 3.1	0.5	-3.0 ± 3.0	E	P<0.001
1199/00	*M. biceps femoris* weight (g)	72	0.2	22.7 ± 4.4	4.0^{sug}	2.2 ± 4.3	R	P<0.05

[1] Descriptions of column headings are given in Table 4.16
[2] Sex in which the QTL peak was significant
[3] Significance of the sex interaction at the position

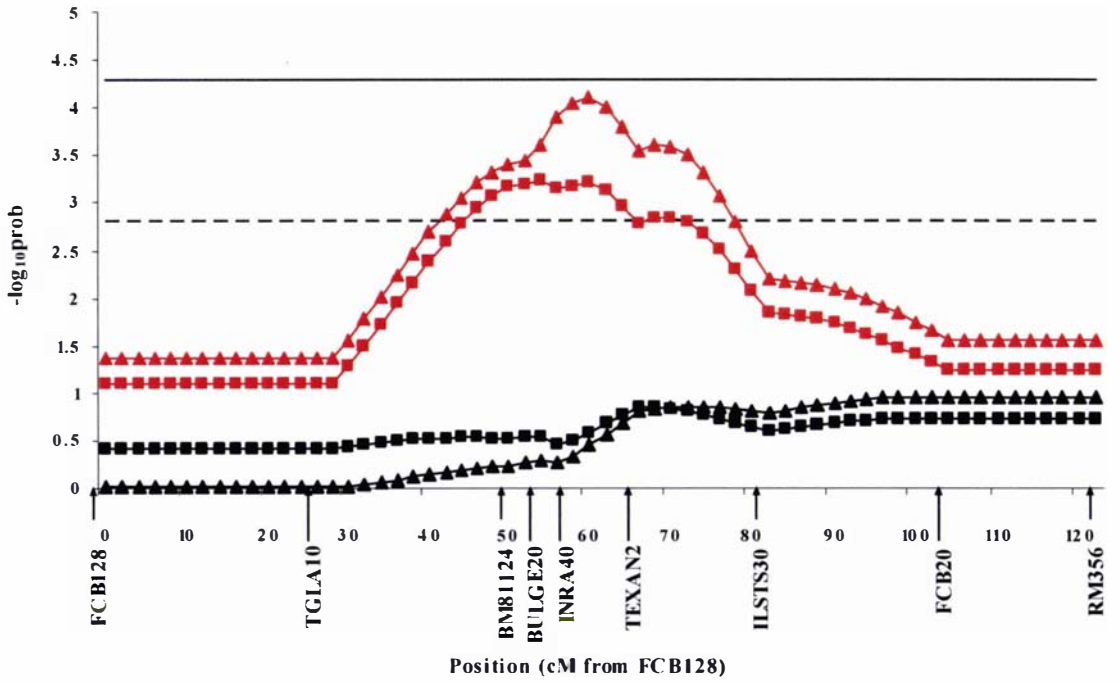


Figure 4.30 –$\log_{10}$prob curves for sire 15/98 half-sib QTL analysis for a region of ovine chromosome two – sex interaction analysis for *M. longissimus* dimensions
Ram *M. longissimus* width (A) ■■■ ; Ewe *M. longissimus* width (A) ●●● ; Ram *M. longissimus* area ▲▲▲; Ewe *M. longissimus* area ▲▲▲; suggestive threshold –; significance threshold - - -

4.4 Summary

The following three tables summarise the results presented above regarding the existence of QTLs (Table 4.41), the likely positions of the QTLs (Table 4.42) and the size of the QTL effects (Table 4.43).

From Table 4.41 it can be seen that no significant QTL were detected for sire 535/98, and significant QTL were detected for only one trait for sire 150/96 and two traits for sire 122/99. However, for sire 122/99 because of low progeny numbers it was not measured for many of the traits, and for those it was measured for it was likely to have reduced power (due to the low numbers). The greatest number (23) of QTL were detected for sire 429/98, with the only trait groups for which QTL were not detected being ultrasound muscle and loin muscle traits. The trait group for which the most number of QTL were detected were the leg muscle (25), fat traits (11), and the ratio traits (11), with QTL detected in four of the seven sires for these traits.

Table 4.41 A summary of the number of significant QTL detected, by trait group for each sire.

	Sire Number[1,2]						
Trait Group (total no. measured)	**1199/00**	**1170/00**	**150/96**	**15/98**	**122/99**	**429/98**	**535/98**
Weight (6)	-	-	-	-	-	1	-
Ultrasound muscle (3)	-	2	-	3	-	-	-
Ultrasound fat (1)	1	-	-	-	-	1	-
Dressing-out percentage (1)	-	-	-	1	1	1	-
Linear (4)	-	-	-	1	-	1	-
Leg muscle (9)	6	6	1	8	n/a	4	-
Other muscle related (3)	3	2	-	3	n/a	3	-
Loin muscle (4)	1	1	-	1	1	-	-
Leg fat (4)	4	2	-	2	n/a	1	-
Loin fat (3)	2	-	-	1	-	3	-
Meat quality (16)	-	1	-	-	n/a	9	-

[1] - = no evidence for a QTL
[2] n/a traits not measured for progeny from this sire

There were two chromosomal regions (Table 4.42) where QTL affecting muscle and fat traits were detected, one at around 52cM, and a second around 64-70cM. There is additionally a region around 122cM where a QTL for meat quality traits appears to map. Data relating to the confidence intervals surrounding these peaks are not presented in these tables, but can be found in the relevant section for each sire. In general, confidence intervals for each peak tended to be large, spanning between 40 and 100cM.

The size of the QTL effects (Table 4.43) varied for a trait group between sires. An additional measure that is presented in these tables is the percentage of the QTL estimate relative to the phenotypic mean. On average, the size of the effects relating to fat traits was larger than those affecting muscle traits. The muscle effects were around 4-8% of the phenotypic mean or 0.5–1.3 of the adjusted SD, whilst those for the fat traits, were 9-24% of the phenotypic mean or 0.5-1.2 of the adjusted SD.

Table 4.42 Summary of results by trait group, showing the likely position (cM) of significant QTL.

	Sire Number[1,2]						
Trait Group	**1199/00**	**1170/00**	**150/96**	**15/98**	**122/99**	**429/98**	**535/98**
Weight	-	-	-	-	-	48	-
Ultrasound muscle	-	122	-	54 & 66[3]	-	-	-
Ultrasound fat	20	-	-	-	-	64	-
Dressing-out percentage	-	-	-	72	76	96	-
Linear	-	-	-	34	-	64	-
Leg muscle	54 & 68[3]	54	96	54 & 72[3]	n/a	54	-
Other muscle related	54 & 64[3]	54 & 62[3]	-	54 & 62[3]	n/a	54	-
Loin muscle	52	56	-	66	96	-	-
Leg fat	54 & 70[3]	50	-	58 & 70[3]	n/a	54	-
Loin fat	54 & 66	38	-	62	-	28	-
Meat quality	-	14	-	-	n/a	122	-

[1] - = no evidence for a QTL
[2] n/a traits not measured for progeny from this sire
[3] Graphical evidence suggested two peaks within the region studied

Table 4.43 Summary of results by trait group, showing the size of significant QTL effects both in terms of standard deviation units and as a percentage of the phenotypic mean for the trait

	Sire Number[1,2]						
Trait	**1199/00**	**1170/00**	**150/96**	**15/98**	**122/99**	**429/98[3]**	**535/98**
	Expressed in Terms of Standard Deviation Units						
Weight	-	-	-	-	-	0.4	-
Ultrasound muscle	-	0.5	-	0.5	-	-	-
Ultrasound fat	-0.7	-	-	-	-	-	-
Dressing-out percentage	-	-	-	0.3	1.0	0.3	-
Linear	-	-	-	0.5	-	0.5	-
Leg muscle	0.9	0.6	0.7	0.6	n/a	0.6	-
Other muscle related	1.3	0.8	-	0.8	n/a	1	-
Loin muscle	0.5	0.6	-	0.6	0.8	-	-
Leg fat	-1.2	-	-	-0.6	n/a	-0.8	-
Loin fat	-0.7	-0.5	-	-0.5	-	-0.9	-
Meat quality - pH	-	-	-	-	n/a	0.6	-
Meat quality - colour	-	-	-	-	n/a	0.6	-
Meat quality - WB	-	0.8	-	-	n/a	0.6	-
	Expressed as a Percentage of the Phenotypic Mean						
Weight	-	-	-	-	-	5	-
Ultrasound muscle	-	5	-	5	-	-	-
Ultrasound fat	-19	-	-	-	-	-18	-
Dressing-out percentage	-	-	-	2	5	1	-
Linear	-	-	-	2	-	2	-
Leg muscle	7	4	5	5	n/a	5	-
Other muscle related	8	4	-	4	n/a	4	-
Loin muscle	5	3	-	6	4	-	-
Leg fat	-20	-	-	-11	n/a	-12	-
Loin fat	-20	-9	-	-20	-	-24	-
Meat quality - pH	-	-	-	-	n/a	2	-
Meat quality - colour	-	-	-	-	n/a	5	-
Meat quality - WB	-	14	-	-	n/a	14	-

[1] - = no evidence for a QTL

[2] n/a traits not measured for progeny from this sire

[3] Note that for Sire 429/98, Q is associated with negative and positive values for muscle and fat traits, respectively

4.5 Discussion

4.5.1 Trial Design

A half-sib design as first described by Neimann-Sorensen and Robertson (1961) was used. This approach has been used to identify a number of QTL in cattle (Beever et al., 1990; Napolitano et al., 1996; Casas et al., 2000). An alternative design would have been an F2 backcross design, which is also commonly used for QTL detection (Walling et al., 1998; Perez-Enciso et al., 2000). The F2 backcross approach was not chosen, as the time required to generate progeny is approximately double that required to carry out a half-sib design.

4.5.2 Pedigree of Sires

Pedigrees dating back to the original imported animals and some of their ancestors were achieved for most animals in the pedigrees of the experimental sires, however, there were also a number of animals where pedigree information did not exist, despite the animals having been born in New Zealand. From the results of the QTL analysis it would appear that there are at least 3 sources of alleles, two from the parents of sire 150/96 and another from the dam of 429/98. Although there are no immediate inbreeding loops within the pedigrees, a number exist in previous generations, which do link 150/96 and the dam of 429/98. These inbreeding loops are in part due to the small genetic base that was introduced into New Zealand (approximately 200 animals formed the base imports). Once a firm haplotype is confirmed, the opportunity exists to use the iterative allelic peeling procedure described by Thallman et al. (2001) to explore in detail the likely transmission of the alleles of interest through this pedigree.

4.5.3 Informativeness of Sires

One of the keys to obtaining significant results in a half-sib QTL study, is to use sires that are heterozygous for markers in the region of interest, thus enabling the comparison of progeny that have inherited alternate alleles from the sire. Broad et al. (2000) showed that of the 36 rams genotyped over five markers in their trial, only 12 were heterozygous for one or more of the markers, with the remaining 24 homozygous at all markers suggesting a low level of polymorphism around the GDF8 locus within the purebred Texel population, possibly a consequence of selection at the locus. In the current study all purebred Texel sires were heterozygous for at least one marker, although only one was heterozygous at all loci (Table 4.13). The IC of the sires presented in Figure 4.5 reflects a low level of polymorphism for some sires and markers, however, it is improved on the work of Broad et al. (2000). This low IC meant from the outset that the ability to detect QTL would be compromised for sires 150/96, 122/99 and 535/98 if the QTL was between markers FCB128 and INRA40. The most likely reason that the IC content in this study was on average higher than that seen by Broad et al. (2000) is that the purebred dams used in the original study had similar alleles to the sires, thereby resulting in a higher proportion of progeny heterozygous for the same alleles as their sire. In the current study commercial "non-Texel" dams were used and although the same alleles were present in the dam population, their lower frequency allowed the sire inherited allele to be determined more often.

To eliminate the occurrence of marker alleles that cannot be traced to the sire, a program such as Arlequin (Schneider et al., 1997) could be used. This program infers alleles based on the number of times a certain allele combination has been inherited by other siblings. However, it was felt that it was unlikely to improve the results achieved in this study.

4.5.4 Single Trait Analysis of Growth Rates

Differences in growth rates, indicate that animals are on different growth paths, and will likely have different mature weight. Studies involving the Callipyge locus have found no differences in growth rates between animals exhibiting the increased muscling phenotype and those not (Jackson and Green, 1993).

No QTL were detected that affected growth rate in the region studied.

4.5.5 Single Trait Analysis of Dressing-Out Percentage

An increased DO indicates that there was more carcass relative to non-carcass components. Studies involving the Callipyge locus have found differing results for DO. The work of Abdulkhaliq et al. (2002) found no differences between callipyge and "normal" lambs, yet Jackson et al. (1997a) found significant differences, with Callipyge lambs having a significantly higher DO. Studies with the muscular-hypertrophy phenotype in cattle show these animals to have a higher DO than their "normal" counterparts (Dumont, 1982). The QTL reported by Marcq et al. (2002) also appears to affect DO, and maps to the region of markers BM81124-BULGE20.

There was evidence for a QTL effect on DO for three sires 15/98, 122/99 and 429/98 however, the position of the peaks varied, although the peaks for sires 15/98 and 122/99 was at 72-76cM, which places it between markers TEXAN2 and ILST30 and thus in the location of the proposed second QTL peak (4.5.11). Unlike the work of Marcq et al. (2002) there was no evidence for a peak in the region where the other muscling/fat peak has been detected.

Overall, from this study there is limited evidence for a QTL affecting DO in the region of Chr2 studied.

4.5.6 Single Trait Analysis of Linear Measurements

Differences in carcass shape have been previously detected in animals known to have different carcass composition. Kadim et al. (1989) showed differences in carcass shape between lines of Southdown animals that had been divergently selected for back fat, with the fatter animals wider, but shorter (in both height and length). Thus if a QTL was affecting muscling or fat traits, it is possible that differences in carcass linear measurement might be detected between the genotypes. Marcq et al. (2002) measured carcass length, carcass width and leg length, but only found evidence for a significant QTL affecting carcass width at approximately marker BM81124, which was in close proximity to the peaks for the muscle trait QTL they detected.

Only two instances of QTL affecting linear traits were identified in this study, both were for gigot width but in different positions, with peaks recorded at 34cM (sire 15/98) and 64cM (sire 429/98). The peak for sire 429/98 corresponded to the position where a QTL for ultrasound fat depth C was detected, however, no other significant traits for 15/98 had peaks near 34cM in the region of TGLA10.

Overall, from this study there is no consistent evidence for QTL affecting linear measurements in the region of Chr2 studied.

4.5.7 Single Trait Analysis of Whole Leg Measurement

Differences in the weight of carcass cuts have previously been seen with the Callipyge locus in which the leg makes up a larger proportion of carcass weight relative to other cuts (Goodson et al. 2001), and the rib-eye muscling locus in which the loin makes up a larger proportion of carcass weight relative to other cuts (Nicoll et al. 1998). A QTL affecting hind-quarter weight was also detected by Marcq et al. (2002), in the region of markers BM81124 and BULGE20.

Peaks were detected for trimmed leg weight adjusted for carcass weight in sires 1170/00 and 15/98; they mapped to 60 and 40cM respectively. Sires 1170/00, 1199/00 and 15/98 all carry the same sire alleles in the favourable phase in this region, yet there was no peak for this trait for sire 1199/00. One explanation is that the variant was inherited from the dam. The fact that it was not detected by the LD analysis (4.5.13) suggests it is either at low frequency or not in strong LD with the markers used. A significant QTL

for trimmed leg weight adjusted for carcass weight indicates a re-distribution of muscle and therefore cut weight throughout the carcass.

Overall, there is no consistent evidence for a QTL affecting weight of the leg in the region of Chr2 studied.

4.5.8 Single Trait Analysis of Muscle Measurements

Ultrasound measurements

Detection of QTL based on ultrasound data of *M. longissimus* measurements assumes that the QTL affects loin muscle traits. Based on Broad et al. (2000) and similar results by Walling et al. (2001), it appeared likely that the QTL sought in this study would affect the loin. Howevcr, other loci affecting muscling in sheep have been shown to have differing effects across the carcass. The Callipyge locus has a large effect on the leg and the loin with lesser effects on other cuts (Goodson et al., 2001), similarly the rib-eye muscling locus only has a large effect on the loin with little effect on other carcass cuts (Nicoll et al., 1998). The region of DNA in this study was chosen for examination as it contains the GDF8 locus. Mutations in this gene are known to cause the muscle-hypertrophy phenotype in Belgian Blue cattle. In the Belgian Blue enhanced muscling has been shown to be most pronounced in the leg, although there was evidence of enhanced muscling in the loin as well (Dumont, 1982).

The ultrasound measurements collected in this study are most comparable to the work of Broad et al. (2000) and Walling et al. (2001). In this current study there was no firm evidence for an ultrasound measured muscle QTL, was there was only evidence from two sires and they had peaks at different positions. The position of these two peaks was at 122cM (marker RM356) and 54-62cM (markers BULGE20 to TEXAN2) for sires 1170/00 and 15/98 respectively. The second of these peaks encompasses the region identified by Broad et al. (2000) who found a peak for ultrasound muscle traits around INRA40 (57cM). The peak of Walling et al. (2001), however, was around FCB128 (0 cM), although their confidence interval encompassed INRA40. In both of these studies, the evidence was not consistent across sires as to the presence and location of the peak.

The ability to detect a QTL affecting *M. longissimus* traits by ultrasound relies on there having been suitable phenotypic differentiation of the alternate genotypes at an early

age. M. *longissimus* muscle has been shown to be early maturing relative to other muscles (Butterfield, 1988), with a high growth impetus until about 20% mature weight has been achieved and a low growth impetus subsequently. Given that the average weight of lambs across flocks at scanning was 36kg which was more than 20% of their predicted mature weight (70-100kg), it was likely that this muscle was well developed at this time and that if differences between genotypes were to exist, they should be evident by this time.

Overall there is no conclusive evidence for QTL affecting ultrasound measurements of *M. longissimus* dimensions in the region of Chr2 studied.

Leg measurements

The main effect of the Callipyge locus and the muscle-hypertrophy locus is that of increased muscle weight in the carcass relative to bone as determined by carcass dissection (Jackson et al. 1997a and Dumont, 1982). The QTL reported by Marcq et al. (2002) on ovine chromosome two, increases muscle weight in the carcass as determined by dissection and maps to the region of markers BM81124 through BULGE20 (50-54cM). The work of Jackson et al. (1997b) with the Callipyge locus showed that although most muscles within the pelvic limb were larger in Callipyge lambs relative to normal lambs, there was no difference between the phenotypes for the *Peronius tertius* muscle, and a limited difference in the *Rectus femoris* muscle. In their work, the pelvic limb muscles most affected by the callipyge locus were the *semimembranosus*, *superficial gluteal* and *adductor* (Jackson et al., 1997b). In a review of French work looking at the muscle-hypertrophy phenotype in cattle, Dumont (1982), noted that there was considerable variation in the degree of hypertrophy expressed by muscles in the pelvic limb, with M. *biceps femoris* showing the greatest, followed by M. *gluteus medius*, *M. semitendinosus*, *M. quadriceps* femoris and *M. semimembranosus*, whilst the pectineus and *flexor digitorum superficialis* showed little hypertrophy.

Peaks were detected for 4, 5, 6 and 3 out of the seven individual muscle weights for sires 1170/00, 1199/00, 15/98 and 429/98, respectively, and also total leg muscle weight for these same four sires. A QTL effect was seen for all muscles in at least one of these sires, with M. *semimembranosus* the only muscle to show a significant QTL effect across all four sires. The position of the peaks ranged from 34 – 56 cM, although the

majority were in the 50 –56cM region (markers BM81124 - INRA40). The amount of variation explained ranged from 0.5 to 0.9 of a phenotypic standard deviation, or 4 to 7% of the phenotypic mean, with effects largest in sire 1199/00, and least in sire 429/98. For sire 429/98, the Q allele is associated with decreased muscle weights relative to q. Three of the four sires have the same alleles associated with Q through this region, with the fourth sire (sire 429/98) having the same alleles at markers BM81124 and BULGE20 associated with the q allele. This provides good evidence for a QTL affecting muscle within the animals studied associated with particular alleles (to be discussed later). The locations of the peaks were also in general agreement with those found for *M. longissimus* traits, and map to the same approximate location as the peak described by Marcq et al. (2002).

There appears to be evidence for a second peak affecting muscle traits for sire 1199/00 and possibly also for 15/98. These second peaks occurred for the same traits that the first peaks were detected for, and were noticed in the graphical presentation of the results. For sire 1199/00 but not for sire 15/98 the second peaks reached a similar peak value to that of the first peaks. The position of these second peaks appears to be at approximately 65cM. This second peaks suggests that a second QTL may be segregating in the region studied (two peak analysis 4.5.11).

Jackson et al. (1997) and Dumont (1982), have previously shown the impact of QTL varies from one muscle to another. However, if a QTL is detected for any one muscle it should be consistent across sires, which was not the case for the results reported. In observing the results for those muscles within a sire that did not reach the significance thresholds, there did appear to be evidence of peaks in similar positions, some of which were approaching the significance thresholds. Given that the muscles of the leg are some of the first to differentiate and mature to provide mobility for the young, with high impetus up to 20% of mature weight (Butterfield, 1988), differentiation of the muscles should have taken place by the time these lambs were slaughtered and so differences between the two genotypes should have also been evident.

Overall, there is strong evidence across four sires for a QTL affecting leg muscle traits between markers BM81124 - INRA40 (50-56cM). For two sires there was evidence for a second peak around marker TEXAN 2 (65cM).

Muscle percent

Muscle percent is a function of total muscle weight relative to carcass weight, therefore, most studies looking at muscling loci such as Callipyge and the muscle-hypertrophy phenotype have shown an increase in muscling percent with the favourable phenotype (Jackson et al. 1997a; Dumont et al. 1982). The QTL reported by Marcq et al. (2002) also increased muscle percent and mapped to the region of markers BM81124-BULGE20.

In this study a QTL affecting muscling percent in the leg, was detected in all four sires which have previously shown evidence of a QTL based on individual muscles. The most significant peak for sire 1199/00 was at 68-72cM, however, there appeared to be two peaks for this sire with a second peak at approximately 54cM. This peak at 54cM mapped to the same position as the peaks for the other three sires, between markers BM81124 and BULGE20, which was the region where most other peaks have mapped to and was where Marcq et al. (2002) had shown a peak to exist. The second peak detected at 64cM, between TEXAN2 and ILST30, was where the proposed second peak from the individual muscle traits mapped to. The size of the effect is between 0.8 and 1.3 of a standard deviation unit, or 4 to 8% of the phenotypic mean. For sire 429/98, the Q allele is associated with decreased muscle percentage relative to q. The region of the first peak is where three of the four sires (1170/00, 1199/00 and 15/98) have the same alleles associated with the Q allele and sire 429/98 has the same alleles associated with the q allele.

Overall, this supports the findings based on the individual peaks, of strong evidence for at least one (possibly two) QTL affecting leg muscle traits in the region of Chr2 studied.

Muscularity and muscle to bone ratio (MTB)

Muscularity is the depth of muscle relative to a skeletal dimension and is thus related to conformation (Purchas et al., 1991), whilst MTB is based on the ratio of weights of the two components and so the two measures are not identical (Purchas, 1993). The MTB ratio is independent of the effects of fat, and thus provides an indication of whether an increase in muscling percent was due to a decrease in fat percent, or an increase in muscle to bone ratio. Dumont (1982) in his review of French literature, noted that one of the features of muscle-hypertrophied cattle was their increased MTB. Although not

directly reported, it would appear that from the work of Goodson et al. (2001) that Callipyge lambs also have a higher MTB relative to "normal" lambs as the Callipyge lambs had significantly higher weights of lean for no change in bone weight. Jackson et al. (1997) showed that Callipyge lambs had significantly decreased weights of bone relative to "normal" lambs in addition to their increased muscle weight. The QTL reported by Marcq et al. (2002) also appeared to affect bone%, and it mapped to a similar region as that for the muscling peaks. However, the direction of the effect was not reported and so whether there was an effect on MTB was unclear. None of the previously reported studies of muscling loci have considered muscularity, but such QTL may exist based on the work of Wolf et al. (2001) who reported significant differences in muscularity in two genetic lines of Texels.

In this study QTL affecting MTB were detected in all four sires which have previously shown evidence of a QTL based on individual muscle weights (1199/00, 1170/00, 15/98 and 429/98), and for LGMUSC for three of these sires (not 1170/00). There was evidence for two peaks for sires 15/98 and 1199/00 for LGMUSC at approximately 62cM and 54cM, the later of which was the location of the one peak for sire 429/98. The mapping of two peaks for sires 15/98 and 1199/00 is consistent with the individual muscle traits. For MTB the position of the peaks for sires 1170/00 and 1199/00 was at 62cM, with graphical evidence (reached L&K significance threshold) for a second peak at 52cM for sire 1199/00 which was the location of the peaks for sires 15/98 and 429/98. The region around 52cM is where the majority of other peaks in this study have mapped to; whilst the second peak detected at 64cM, which is close to where a number of other "second peaks" have mapped to. For sire 429/98, the Q allele is associated with decreased muscle percentage relative to q. The region of the peak at 52cM is where three of the four sires (1170/00, 1199/00 and 15/98) have the same alleles associated with the Q allele and sire 429/98 has the same alleles associated with the q allele.

Overall, these findings support the existence of at least one (possibly two) QTL affecting leg muscle traits in the region of Chr2 studied.

Loin measurements

Only three peaks were detected in this study for *M. longissimus* traits measured at the processing plant, a peak for A at 38cM (sire 1170/00), and two peaks for LL weight at

52 and 66cM (for sires 1199/00 and 15/98 respectively). This presents inconsistencies in that no QTL were detected in the ultrasound muscle data for sire 1199/00, despite this sire being in similar in phase to sires 1170/00 and 15/98 for up to six of the eight markers. Additionally, the peak for sire 1170/00 detected here lay at 38cM, whereas it was previously detected at 122cM. Also 38cM lies towards the middle of the widest marker spacing in the study, where the information content for this sire was low (0.5).

That the results are not consistent can, to an extent, be explained by the low correlation between the ultrasound and works measurements of A and B, which were 0.25 and 0.41, respectively, despite approximately half of the lambs being slaughtered immediately after scanning when effectively the same measurement was made in the live animal. These correlations are lower than those of McEwan et al. (1989) who showed correlations between works measurements and ultrasound of 0.38 and 0.72 for A and B respectively. However, using either result, it would appear that the repeatabilities of the traits are not high, most likely due the distortion that takes place between the animal standing (alive) to being hung as a carcass. These low correlations, mean that evidence for one trait (e.g. ultrasound) would not necessarily be expected for the other (e.g. processing plant), as has been shown in these results.

Overall, there is no conclusive evidence for QTL affecting loin muscle traits in the region of Chr2 studied, which is in agreement with the findings of the ultrasound data.

4.5.9 Single Trait Analysis of Fat Measurements

Ultrasound measurements

Although a QTL affecting fat depth was detected via ultrasound measurements in two sires, there was no agreement in the position of the peak. For one sire (1199/00) a peak for fat depth was at 20cM, whilst for the other (429/98) it was 64cM. The peak for sire 1199/00 was very broad, and it was surprising that the peak lay at 20cM, as this was in the middle of the widest marker spacing in the study, where the IC of this sire was low (0.5). The peak for 429/98 was close to INRA40/TEXAN2, which places it where the potential eye-muscle QTL was identified, however, 429/98 did not show evidence of an eye-muscle QTL. Neither of these peaks map to the region of ILST30/FCB20, the region identified by both Walling et al. (2001) and the follow up work to Broad et al. (2000).

The ability to detect a QTL affecting fat depots such as C relies on differentiation of the alternate genotypes at an early weight. Of the three major carcass components (muscle, fat and bone) fat is the last tissue depot to mature and thus increases as a proportion of liveweight as an animal ages (Butterfield, 1988). Given that the lambs were only at approximately 50% of their mature liveweight, fat levels would have been expected to be proportionately low, especially given their Texel parentage, as Texels are known for lower fat percentages relative to other breeds (Wolf et al., 1980). This was confirmed by the average UC measurement across all lambs being only 1.7mm, in comparison to 2.4mm at slaughter. When this was compared to measurements from other trials involving Texel-cross lambs slaughtered at approximately 22kg carcass-weight where C measurements of 5.6mm across sexes were reported (Wolf et al., 2001), the values seen in this study are relatively low. This would suggest that fat deposition was only just starting to occur, this could mean that differences between alternate genotypes were lower than would have been expected at higher carcass weights (and greater fat depths) leading to a reduced ability to detect a QTL.

Overall, there is no conclusive evidence for a QTL affecting ultrasound measured fat depth C in the region of Chr2 studied.

Leg measurements

Generally increases in muscling are associated with decreases in fatness. Therefore, given the significant QTLs found for muscle traits, it might be expected that significant fat-related QTL would be found. This is not to say that a QTL affecting fat has been found, which in turn has an effect on muscling (a point to be discussed later). However, the underlying principle remains that if there is an increase in partitioning of nutrients into one body component, there will tend to be less nutrients available for other components. This has been shown to be the case for cattle with muscle-hypertrophy, and the Callipyge phenotype in sheep with animals that exhibit increased muscling also have decreased fat (Dupont, 1982; Goodson et al. 2001). The QTL reported by Marcq et al. (2002) also appears to affect fat weight, and maps to the region of markers BM81124-BULGE20. Jackson et al., (1997a) showed that within the Callipyge phenotype that the effect of fat was more apparent within the subcutaneous fat depot than the intermuscular fat depot.

There was evidence for peaks affecting subcutaneous fat weight and total fat in the leg, for sires 1170/00, 1199/00, 15/98 and 429/98. In addition there was a peak for intermuscular fat weight for sire 1199/00. The positions of the peaks varied between sires and also within sire, however, in general there appears to be two QTL for fatness one at around 54cM, (BULGE20 - INRA40) and the other around 70cM, (TEXAN2 - ILST30). The size of the effect also varied between sires, ranging from 0.6 to 1.2 adjusted phenotypic standard deviations, or 12 to 20% of the phenotypic mean. For sire 429/98, the Q allele is associated with increased fat weights relative to q. The position of the first peaks at around 54cM is in a similar region to that where the QTL for leg muscle and loin muscle traits mapped to, and it mapped to the same location as that described by Marcq et al. (2002). The region of the first peak at 54cM is where three of the four sires (1170/00, 1199/00 and 15/98) have the same alleles associated with the Q allele and sire 429/98 has the same alleles associated with the q allele. The position of the second peak at around 70cM was in the region where a potential second peak for muscle weight was also detected. Thus, there appears to be strong consistent evidence for up to two QTL affecting fat traits in this region.

Overall, there is strong evidence across four sires for a QTL affecting leg fat weight traits between markers BULGE20 - INRA40 (54cM). For two sires there was evidence for a second peak between markers TEXAN 2 and ILST30 (70cM).

Loin measurements

There was evidence for QTL affecting GR and LLFT weight for sire 1199/00 at approximately 60cM, and for C for 15/98 at 62cM, that is in the region of INRA40/TEXAN2. A peak for LLFT weight was also detected for sire 429/98 at 28cM. This presents inconsistencies in that peaks for UC measurements were detected in sires 1199/00 and 429/98. Although the peaks for 429/98 map to a similar position, as previously discussed, this is in the low information content region for that sire. It was the reverse for 1199/00 for whom UC was in the uninformative region, whilst the peaks seen here for GR and LLFT were at approximately 60cM (an informative region). No peaks were detected for sire 15/98 for the UC data. For sire 429/98, the Q allele is associated with decreased muscle percentage relative to q.

The correlation between the ultrasound and works measurements of fat depth C was low

(0.48). This value is lower than that reported by McEwan et al. (1989) of 0.71. The correlation between works measurements of C and GR of 0.6, was moderate, but slightly lower than those reported by Kirton and Johnson (1979) which ranged from 0.71 to 0.78 and that of McEwan et al. (1989) which was 0.73. These results overall, suggest at best only moderate repeatability between these traits. These low/moderate correlations, mean that evidence for one trait (e.g. ultrasound) would not necessarily be expected for other traits (e.g. works) for the same sire, as has been shown in these results. Mapping of a QTL to this region is consistent with both the ultrasound measurements and the works measurements of *M. longissimus* area, albeit in different sires and not consistent across traits.

Overall there is no conclusive evidence for QTL affecting loin measurements of fat in the region of Chr2 studied.

Bone traits

There are conflicting results in the literature as to whether or not loci known to affect muscle also have an effect on the amount of bone. In a review of French literature, Dumont (1982) showed in a trial comparing normal and muscle-hypertrophied bulls and heifers, that there were no significant differences in the amount of bone in the bulls, but that muscle-hypertrophied heifers had lower bone weights compared with "normal" heifers (i.e. sex interaction). Goodson et al. (2001) found no differences in the amount of bone between Callipyge and "normal" lambs; yet Jackson et al. (1997a) found Callipyge lambs to have significantly lower bone weight compared with "normal" lambs. Marcq et al. (2002) showed evidence for a QTL affecting bone traits in the region where QTL for muscling and fat traits were detected.

In this study, despite QTL being detected for muscle and fat traits, no QTL for carcass weight-adjusted bone traits were detected.

4.5.10 Single Trait Analysis of Meat Quality Traits

Whether or not the palatability and appearance of meat changes in animals with increased muscling is an important consideration for the meat industry. Evidence from the studies into the muscle-hypertrophy phenotype in cattle have shown that the meat from muscle-hypertrophied animals is paler and more tender, but not different in terms

of juiciness or flavour relative to that from normal cattle (Bailey et al., 1982). The Callipyge phenotype in sheep, however, has been shown to result in tougher meat as measured by Warner-Bratzler measurements (Koohmaraie et al., 1995; Field et al., 1996), and by sensory panel (Shackelford et al., 1997). This decrease in tenderness was reported to be associated with increased calpastatin activity (Koohmaraie et al., 1995; Duckett et al., 2000). Attempts to improve the tenderness via different aging and conditioning processes have been unsuccessful (Kuber et al., 2003). Jopson et al. (2001) showed that, although the mean shear force values of meat from lambs carrying the favourable form of the rib-eye muscling locus were higher, this difference could be overcome by appropriate aging of the meat under normal conditions.

Differences in pH and sarcomere length have not commonly been assessed in studies observing the Callipyge phenotype. However, the work of Koohmaraie et al. (1995) showed no differences in these traits between M. *longissimus* muscles of Callipyge and "normal" lamb. Kuber et al. (2003) showed a more rapid decline in pH for Callipyge lambs compared with normal lambs over a period of aging. No differences in cooking loss between Callipyge and "normal" lambs were detected by Shackelford et al. (1997). Although several studies have reported a Chr2 QTL causing increased muscling in the Texel breed of sheep, none until this study have investigated whether this QTL, has an effect on meat quality. It is important to reiterate at this point that the QTL affecting muscling and fat traits in this current study has been found in the region of 52 – 64cM or markers BM81124 through ILST30. From the current study there appears to be evidence for a QTL affecting meat quality traits in sire 429/98, that maps to 122cM or at marker RM356 which was at the edge of the region studied, although the confidence intervals covered the majority of the region studied. At this position there was evidence for a QTL affecting pH of M. *semimembranosus*, colour "a" and "b" of M. *semimembranosus* and *M. longissimus*, and Warner-Bratzler measured peak forces of M. *semimembranosus* and *M. longissimus*. Colour was decreased and Warner-Bratzler shear force values were increased, and thus there is a QTL associated with meat tenderness at this location. There were two quality peaks which mapped to the region identified for muscling at 56cM, they were for colour "L" of M. *semimembranosus* and *M. longissimus*, the effect of these peaks was to increase the values. Sire 429/98 had a unique (compared to other sires in the trial) allele combination at markers FCB20 and RM356 (where the majority of the quality QTL were detected). These results suggest

that there is possibly a further QTL downstream from the region of study that has effects on meat quality, and that is independent of muscle and fat, as there were no QTL detected for muscle or fat traits in this region. Whether or not such a QTL exists, can be investigated by further marker genotyping in that region.

Overall there is conclusive evidence for one sire for QTL affecting meat quality traits at marker RM356 (122cM), however, this location is unique compared to the locations reported for the muscle and fat QTL.

4.5.11 Combined Trait Analysis

Single trait analysis, although useful for detection of QTL and their effects given minimal assumptions, does not account for the correlation between traits, a correlation which can increase the power and accuracy of QTL detection (Jiang & Zeng, 1995; Weller et al., 1996; Mangin et al., 1998; Henshall & Goddard, 1999; Knott & Haley, 2000). Gilbert and Le Roy (2003), compared three alternative multi-trait analysis methods via simulation: multivariate function, principal component analysis and discriminant analysis. They discussed advantages and disadvantages of each method and concluded that all methods increased the power of detection by 30-100%. In this study multivariate and principal component analysis, were used.

Multivariate analysis involves the inclusion of many traits within a single QTL analysis (Jiang & Zeng, 1995; Calinski et al., 2000). It has been used to summarise the findings of previously presented single trait analysis of QTL affecting meat quality traits (Szyda et al., 2003). It has limitations in that when the size of the pedigree involved is large, and a large number of traits are involved, computation is difficult which limits the success of this approach (Gilbert & Le Roy, 2003). Interpretation of the size of the QTL effect is difficult, given traits analysed together have different measurements (Weller et al., 1996).

Principal component analysis has been proposed as an alternative type of multi-trait analysis that can used to combine correlated traits to produce non-correlated values which can then be used in QTL analyses (Weller et al., 1996; Mangin et al., 1998). This approach has been used in the search for QTL in studies looking at: non-production traits in dairy cattle (Spelman et al., 1999b); bone measurements and osteochondrosis

scores in pigs (Andersson-Eklund et al., 2000); and carcass data for cattle (Morris et al., 2002). Although the principal component data provides information on the position of the QTL, as discussed by Weller et al. (1996), there were difficulties in estimating the size of the effect with back transformation of the data required.

As single trait analyses had already been carried out in the current study with estimations of the size of the effect reported, multi-trait analyses were only used to gain an additional estimate of the position of the peak. Thus, it was only carried out for groups of traits where QTL for single traits had been identified, including muscle and fat traits of the leg and loin.

The use of multivariate analysis and principal component analysis as means of multi-trait analysis led to similar results being achieved and are therefore considered as overall multi-trait results in the following discussion.

Peaks for individual leg muscles were also detected using multi-trait analysis when all muscles were included together for the four sires with significant peaks for individual leg muscles. The positions of the peaks as determined from this analysis were at 54cM for three of the sires (429/98, 1170/00 and 119/00) and 52cM for the fourth sire (15/98). This provides a more conclusive estimation of the position of the QTL, in the region of BM81124 – INRA40, but closest to marker BULGE20, which corresponds to the position described by Marcq et al. (2002). Graphically there did appear to be a second peak for sire 1199/00, with the peak around 70cM. Again for sire 429/98, the Q allele is associated with decreased muscle values.

Based on the analysis of the single traits there was evidence for *M. longissimus* muscle peaks for three sires, however, the multi-trait analysis only shows evidence for two sires having peaks (1199/00 and 15/98, not 1170/00). The positions of the peaks reported for these sires (54 and 52cM), corresponds with the positions of the peaks for these sires for the multi-trait analysis of leg muscle.

Multi-trait analysis for leg fat weight traits revealed peaks for sires 1199/00, 429/98 and 15/98, with no peak for sire 1170/00, despite a peak in the single trait analysis being detected for this sire. The positions of the peaks (48cM and 68cM for 429/98 and

15/98, respectively; and 56 and 72cM for the two peaks of sire 1199/00) correspond with the regions reported for the individual fat traits, but only correspond approximately with the region reported for the muscle traits. Thus multi-trait analysis has not narrowed down the region of the fat QTL position. Whereas, Marcq et al. (2002) who used only single-trait analysis had peaks for fat that independently mapped to the same position as the muscle peaks. Again for sire 429/98, the Q allele is associated with increased fat values.

Multi-trait analysis of loin fat traits revealed peaks for sires 1199/00 and 429/98, with no peak for sire 15/98, for which a peak in the single trait analysis was detected. The positions of these peaks (32cM and 64cM for 429/98 and 1199/00, respectively) corresponded with the region reported for the individual fat traits, but did not correspond with the region reported for the muscle traits. Again for sire 429/98, the Q allele is associated with increased fat values.

The combined sire analysis of the muscle and fat traits, whether by weight or by percentage again confirmed the position of the major peak to be at 52cM, but again provided further evidence for a second peak. The across sire analysis yielded the highest $-\log_{10}$prob values achieved in any of the analysis, with values of 14.0 and 14.5 for weight and percentage analyses respectively.

Thus having built up single trait by sire, to multi-trait by sire, to combined sire multi-trait analysis, there is strong evidence for the existence of at least one QTL in the region. Whether or not a second peak exists is addressed in the next section. Therefore the results of this study are in strong agreement with the work of Marcq et al. (2002).

4.5.12 Two-peak Analysis

There are likely to be multiple genes on any one chromosome which impact on the same trait. Within the region of Chr2 studied in this trial then, it is possible that there could be more than one gene influencing carcass characteristics. Testing for two-peaks within a region was reported by Walling et al. (1998; 2000) searching for QTL affecting growth and fat traits in pigs, as graphically they had evidence to suggest that there were two QTL segregating in their region of interest. Compared to fitting a single QTL model, however, the two-peak test did not provide a better explanation of the results.

Likewise, in this current study there was graphical evidence for two QTL peaks for two of the sires used. Only these two sires and the traits with which there appeared to be evidence were tested. The first test considered whether or not epistasis was occurring (one QTL impacting on another QTL in the region), with one QTL impacting on the other or vice versa, however, there was no evidence to suggest this to be the case. The second tested for the presence of two versus one QTL peak. Sire 15/98 showed no conclusive evidence for two peaks (independent, or epistatic). Sire 1199/00 showed some evidence for two peaks (significant at the 5% level) for muscle and fat traits. The position of the peaks described in the two-peak analysis was similar to that visually seen at 52cM and 72cM, providing further evidence that the analysis was accurate.

Evidence for a peak in the region of 72cM (region of ILST30 to TEXAN2) is consistent with the work of Stone et al. (1999) who reported suggestive evidence for a QTL affecting retail product yield in the region of TEXAN2. However, in more recent work on the same population, the position of this peak has been mapped to the region of marker RM356 (Casas et al. 2003).

If there is a second QTL peak in the region for this Texel population, it is interesting that only one of the four sires showing evidence for one QTL is showing evidence for it. This can at least in part be explained, as this sire shows evidence of crossing over in the region of the second peak, and so he has a unique haplotype in this region.

Firm conclusions about the presence of a second peak can not be made. However, there is sufficient evidence to suggest that there may be a second QTL in the region, a possibility which needs to be further investigated.

4.5.13 Sex-QTL Interaction Analysis

The search for QTL sex interactions was first carried out by Knott et al. (1998) looking for QTL for growth and fat traits between an outbred wild boar and large white pig cross. In this work, although they found evidence for three traits where there appeared to be a significant sex-interaction, for two of these the results, fitting the sex-interaction did not improve the results. For the third trait, inclusion of the sex-interaction resulted in the peak reaching the significance threshold, whereas when the interaction had not been fitted, the peak only reached the suggestive threshold. De Koning (2001) also

tested for sex interactions in a search for QTL affecting growth and reproductive traits in pigs, however, this approach again failed to find any evidence of an interaction.

The results for this study show evidence for a QTL affecting muscle and fat traits in sires 1170/00, 1199/00 429/98 and 15/98, with the position of the peaks and the size of the effects being similar. If a consistent sex-interaction existed, it would be expected to be observable in each of these sires. There were only seven instances where there were significant sex differences for QTL peaks out of 360 comparisons, with five of these being for sire 15/98. The other two could be considered random chance observations (at α=0.01 one false positive result can be expected per 100 analyses carried out). Given, there were five significant differences for sire 15/98 this could be indicative of a true interaction. However, on closer observation there is limited consistency in these results. There is evidence for two loin ultrasound muscle traits, both of which have a peak in the ewe lambs and not the ram lambs. Although the positions of the peaks differ, they are in agreement with the two positions reported for the single trait analysis for this sire. There are two fat traits where there is a significant QTL in the ram lambs but not in the ewe lambs (SCF and WC). Both map to the same position, however, the position of the peaks is not consistent with those reported for the single trait analysis. There is only evidence for a sex difference in QTL peaks for one of the leg muscle traits (ST), although this peak does map to a region where a single peak was detected for this sire.

Although all of the sex peaks map to the positions where the peaks for the single- and multi-trait QTL were detected, there is only evidence for one leg muscle trait and one of the leg fat weight traits. Although there is some evidence for a sex-interaction for Sire 15/98, if this was a true effect, there should have been more evidence for the leg fat weight traits and leg muscle traits.

If sex-interactions do exist they are likely to be a result of epistatic interactions at another locus impacting on the QTL locus. To date, no sex differences in the detection of QTL have been described (Knott et al., 1998; de Koning, 2001). That sex-interactions do exist is a realistic hypothesis, given the different growth paths and composition of the different sexes. It is likely that the major sex hormones interact with important metabolic pathways via genes/signalling pathways to cause differences in muscle and fat deposition, as are seen in this study. The only documented example

where sex differences for a phenotype have been seen is in work involving the muscle-hypertrophy phenotype in cattle (Dumont, 1982). In Dumont's review of literature there was discussion of work where no differences in the amount of bone were shown between muscle-hypertrophy and normal bulls, but differences between the two groups were seen in heifers, with the muscle-hypertrophied animals having lower bone weight.

In summary, the evidence is inconclusive in the current study for a sex interaction, however, this does not mean that the possibility should be excluded. Because of the trial design, there are at best only 45 animals per sex group for a given sire(assuming all are informative) which means that the ability and accuracy of QTL detection is poor, given the initial power calculations recommended the use of 90 progeny per "group", it is suggested that to test this possibility, more lambs of each sex would have to be generated for any one sire.

4.5.14 Linkage Disequilibrium Analysis

Linkage disequilibrium (LD) analysis has traditionally been used to search for marker-phenotype associations at the population level for discrete traits in human genetic analysis of disease traits, with individuals either affected or normal (Baret & Hill, 1997). However, it can also be used to search for association between any phenotype and marker. If such associations do occur, it suggests that the marker and QTL loci tend to be inherited together and are therefore said to be in LD, that is, they occur together more often than would be expected by chance. In this situation LD analysis was used to determine if there was an association between any of the dam alleles and the phenotypes observed. Although the dams were not genotyped, for sire-informative progeny the allele inherited from the dam could be implied.

There were no clear associations between any of the dam alleles and the phenotypes. There were some instances where adjusting for the dam allele did improve the significance of the peaks detected in the QTL analysis. However, these tended to only be for muscle traits, with increases seen in "one off" muscle traits for all properties. Although in a number of instances adjusting for LD increased the likely significance level of the traits, it did not appear that the LD effects were consistent. Thus, the conclusion is that there was not strong evidence for an LD effect from the dam contributing toward the observed QTL. This suggests that the mutation seen in the

Texel population causing the phenotypes, is likely to be either unique or rare in other breeds. This observation was expected, given there was no known Texel parentage for any of the dams, and there is no known increased muscling phenotype for any of the dam breeds. Detection of LD was, however, somewhat limited in that there were only very low frequencies for some of the dam alleles within the populations and the marker spacings were rather large compared to the likely level of LD expected in the dam breeds (McRae et al., 2002).

Overall, there is no evidence for a LD effect influencing any of the traits collected, within the region of Chr2 studied.

4.5.15 Haplotype

From the results discussed above it appears that at least for one QTL the bounding markers of interest are BM81124 and BULGE20. The C and I alleles form the favourable haplotype at those markers (Table 4.13). Sires 150/96, 122/99 and 535/98 are homozygous at these markers which explains why no QTL were detected in this region for these sires. However, it must be acknowledged that the lamb numbers for 122/99 were low and measurements were also limited for this sire. In order to carry out accurate analysis, the sire must be heterozygous at the markers, so that alternative paternal alleles inherited by the progeny can be compared. In the case of 429/98, the C and I alleles were associated with the q allele, and thus the estimates of the effects always appeared opposite to that for the other sires. For sire 429/98 C and I allele were inherited from its dam rather than from a common grandsire (150/96). Consequently 429/98 inherited C and I alleles from another "Texel ancestor".

The C and I alleles did occur within the dam population. However, they were at relatively low frequencies, with the C allele of marker BM81124 at a frequency of 9% (52 occurrences) and the I allele at marker BULGE20 at a frequency of 2% (13 occurrences). There were nine progeny that inherited the C1 haplotype from their dam, two progeny of sires 1170/00, 1199/00 and 535/98 and three progeny of sire 150/96 (Flocks 1 and 2). This would suggest that the haplotype is not unique to the Texel population but that it is rare in other populations with a less than one percent frequency. However, consideration must be given to whether or not the dam population may have had Texel genes introduced at some point. The breeders reported that the dams were

free of Texel genes but given that seven of the nine animals are off a single property it leaves this question open. Assuming that the haplotype is not unique to the Texel population, an important question is whether the haplotype is associated with increased muscling in non-Texel animals. Given the relatively small number of animals identified, the question cannot be resolved from the current data.

Overall, it would appear that a two marker haplotype (C and I alleles at markers BM81124 and Bulge20) can account for the presence/absence of the muscling/fat QTL identified within the region of Chr2 studied.

4.6 Conclusions

The following conclusions can be drawn from the search for a QTL affecting muscle and fat traits in the region of GDF8 on ovine Chr2.

- There is conclusive evidence for at least one (possibly two) QTL affecting muscles and fat depots within the leg.
  - The first and most consistent QTL maps to the region of markers BM81124 and BULGE20 (52cM from marker FCB128).
  - The second QTL, for which there is only limited evidence, maps to the region of marker TEXAN2-ILST30 (68-72cM from marker FCB128).
- The effect of both QTL is to increase leg muscle weight and decrease leg fat weight for a given carcass weight.
- In contrast to the work of Marcq et al. (2002), no conclusive QTL were detected in this study for any linear dimensions of the carcass or dressing-out percentage.
- In contrast to the work of Broad et al. (2000) and Walling et al. (2002), no conclusive QTL were detected in this study for measurement of M. *longissimus*, whether via ultrasound on the live animal, or post slaughter on the actual muscle.
- The favourable QTL allele affecting muscling and fat traits, did not have a significant negative effect on meat quality characteristics.
- Overall, a two marker haplotype has been identified for the first QTL:
  - The C and I alleles at markers BM81124 and BULGE20, respectively, are associated with the improved phenotype.
- The size of the effect for the first QTL is approximately:
  - 0.6 of an adjusted standard deviation unit or 5-8% of the phenotypic mean for

leg muscle traits.

- 0.8 of an adjusted standard deviation unit or 10-15% of the phenotypic mean for leg fat weight traits.

- Tests for a sex by QTL interaction and effects of linkage disequilibrium did not clarify the QTL detected.
- For one sire a QTL independent of the two previously mentioned, was detected for meat quality traits, and maps to marker RM356 (122cM from FCB128).

A major limitation of the present work is that the observations reflect the allele substitution effect of presumably one copy of the locus of interest from the Texel breed. While this is of great relevance to those farmers using Texels as a terminal sire, there is a need to ascertain the effect of two copies, particularly as composites dams consisting of Texel crosses are now widespread in the New Zealand industry. Given that such animals must exist in the Texel breed and Marcq et al. (2002) identified a additive mode of inheritance for their QTL, there is potential for much larger effects to be present in the homozygotes. However, in this case it would again be important to monitor any associated meat quality effects. A further limitation is the lack of knowledge of the QTL effects on other traits of productive importance including: hogget fertility, hogget and ewe prolificacy, host resistance to parasites, host resistance to facial eczema, wool production and quality. It is important that these effects are investigated prior to widespread use of MAS in industry.

These results are very similar to those obtained by Marcq et al. (2002) with the additional observation that the QTL does not appear to affect meat quality characteristics. This leads on to the question as to what is the underlying gene causing this effect, Marcq et al. (2002) has shown through sequencing that it is unlikely to be modification of the GDF8 gene itself (i.e. no protein coding differences). The alternative is that there is a promoter difference for this gene that controls expression under some as yet unidentified conditions. However, given that GDF8's role has not yet been confirmed, the possibility of other candidate genes exist in the region must be considered. This investigation is presented in Chapter 5 "Candidate genes for a Quantitative Trait Loci for increased muscling".

Chapter 5

Candidate Genes for a QTL for Increased Muscling

5.1 Introduction

Quantitative Trait Loci (QTL) studies aim to identify markers within a region of DNA that account for significant proportion of genetic variation in a trait. In comparison to the human gene map, in which 20,371 protein coding genes have been mapped (based on July 2003 RefSeq genes (http://genome.ucsc.edu), only 746 have been mapped in cattle and 370 in sheep as of April 2003 (www.ncbi.nlm.nih.gov, www.thearkdb.org). So although a region of DNA may be identified from QTL studies in sheep, it is unlikely that potential candidate genes will be identified using the current ovine gene map. Mapping of sheep, cattle, and human genes shows regions of conserved synteny (groups of genes located together), although rearrangement in their order, and exclusions exist (O'Brien et al., 1999). The number of large (>100-kb) conserved colinear blocks between mice and humans has been shown to be 344 (Kent, 2003). The size of these conserved blocks varies, and generally follows a Poisson distribution (Nadeau & Taylor, 1984). These conserved collinear syntenic blocks allow searches for candidate genes within a region of interest in the better studied species, usually humans.

The current study has located a QTL affecting muscling in the Texel breed of sheep (Chapter 4), and an obvious question to be addressed is: "what is the underlying gene causing this effect?". The area in which the search was carried out, is known to contain the Growth Differentiation Factor 8 (GDF8; also known as Myostatin) gene which has been shown to cause the muscle-hypertrophy phenotype in a number of beef breeds. However, previous work with sheep has shown GDF8 to not necessarily be the gene causing the muscle-hypertrophy phenotype in the Texel (Marcq et al., 2002). Additionally there is evidence for a second peak (Chapter 4), and thus there appears to be at least two genes in the region controlling the muscling phenotype.

Given that the size of the QTL effect is approximately one phenotypic standard deviation, it would suggest that the gene involved is one of the major genes in the signalling pathways involved in muscle growth and differentiation. There are several approaches to identifying potential candidate genes, which rely on the conserved synteny between the sheep, cattle and human genomes. The first is to identify genes that are known to influence muscle growth and development, and to search for their location on the human genome, to determine if they map to the region of interest. The second is to search within "Online Mendelian Inheritance in Man" (OMIM) for genes

which have been shown to impact on muscle growth or fat, and to determine if they map to a similar region to GDF8. The third option is to identify the comparative area of interest in the human genome, identify all genes within this area, and study all the information on these genes to determine if any play a role in muscle growth. Lexical analysis and the use of tissue expression data are useful techniques for this purpose.

The purpose of the work described in this chapter was to explore published material relating to regions of conserved synteny between the human, cattle and sheep genomes to determine the region on the human genome that corresponds to the site where the muscling QTL was located in sheep. Then, to search for genes affecting skeletal muscle growth within this conserved region, using a variety of techniques (described above).

There are other approaches that could have been taken including the use of a Gene Ontology (GO) annotated database (Harhay & Keele, 2003). The GO annotated database has been constructed to provide ontologies for gene function across species.

The section of this chapter on "Lexical Analysis" (5.6) was presented at the Fifteenth Conference of the Association for the Advancement on Animal Breeding and Genetics (Johnson et al., 2003). Since that paper was written further genes have been mapped to the region of interest, and thus a follow up (epilogue (5.11)) to this work is presented at the end of the chapter.

5.2 Likely Attributes of the Candidate Gene

The increased muscling phenotype in Chapter 4 is likely to result from either the up-regulation of a gene associated with protein deposition/accretion, or the down-regulation of a negative regulator of protein deposition/accretion. The effect on fat is likely to occur as a result of repartitioning of nutrients towards muscle growth, making less available for adipose growth.

If the mutation is to a gene already positively associated with muscle growth, it is likely to be a mutation in the promoter, which disrupts the binding site for a transcription factor which normally represses transcription. An example of a promoter mutation, is the promoter region variant of β-lactoglobulin in cattle which has a marked impact of the transcriptional regulation of the β-lactoglobulin gene (Lum et al., 1997; Folch et al.,

1999). Another recent example that has been identified, is an increased muscling phenotype in pigs which is due to a variant in a repressor region of the Insulin-Growth-Factor 2 (IGF2) gene resulting in increased expression of the gene in muscles of adult pigs (Andersson, 2003; Nezer et al., 2003).

If the mutation is to a gene negatively associated with muscle growth, it is likely to be a mutation which either knocks out or down-regulates the action of the gene. This type of mutation has already been shown for muscle in cattle and mice with the GDF8 gene, which is a negative regulator of skeletal muscle growth. Mutations to the protein coding region of the gene have been shown to turn down/off this regulatory role, resulting in an increased muscling phenotype (Charlier et al., 1995; Varga et al., 1997; Grobet et al., 1998; Marcq et al., 1998; Szabo et al., 1998; Varga et al., 2003).

Although currently there is no evidence to suggest that GDF8 is involved with the QTL described in Chapter 4 (based on the results of Marcq et al. 2002), it should not be excluded from a potential list of candidate genes, as further work could uncover differences in the promoter region which could in turn impact on the gene.

5.3 Conserved Regions of DNA for Ovine Chromosome 2

The region of interest, identified in Chapter 4, on ovine Chromosome2 (Chr2) is bounded by markers BM81124 and OARFCB20, which corresponds to 2q1.4-q3.2 or 146-193cM on the ovine map published by Maddox et al. (2001). Only 26 genes have been assigned to ovine Chr2, and of these only 12 have been accurately positioned within the chromosome, with the remainder only assigned to the chromosome.

Various mapping experiments have shown that genes mapped to the ovine 2q region also map to bovine Chr2, and specifically when multiple genes have been mapped they have shown conserved order, suggesting homology between the two species. (Broad et al., 1995; Goldhammer et al., 1995; Lopez-Corrales et al., 1998). Ovine Chr2q markers have also been shown to map to a similar region of bovine Chr2 (Broad et al., 2000). Similar homology has been shown with ovine Chr2 and human Chr2 (Ansari et al., 1994; Pitel et al., 1995). In work by Sonstegard et al. (Sonstegard et al., 1998) comparing human and bovine Chr2, although they showed homology, they also suggested there were rearrangements in the region of interest. However, more recent

work comparing the conserved regions between ovine, bovine and human suggests there is no indication of rearrangement within the region of interest although the genes are inverted, however, rearrangements do exist in distal areas (Maddox et al., 2003; van Stijn et al., 2003). These conserved regions are presented in Figure 5.1.

Given this homology between the species, even though relatively few genes and markers have been mapped in sheep, information can be gained from both the bovine and human maps. Ultimately a region of human Chr2 can be identified. However, it can not be directly derived from the ovine map, rather the bovine map must be used as an intermediate. The majority of markers used in Chapter 4 have also been mapped on bovine Chr2 (Broad et al. 2000), which has a greater number of genes accurately mapped on it, compared with the equivalent Chr2 in sheep. Given the bounding markers of BM81124 and OARFCB20, the closest genes mapped to these markers on bovine Chr2 are GDF8 and GCG respectively (http://www.thearkdb.org/anubis). These genes map to approximately 190 and 160 mega base pairs on human Chr2 respectively (http://genome.ucsc.edu). The candidate gene search area corresponds to human Chr2 160-190 mega base pairs or 2q24.2-2q32.3. Any genes uncovered in the following sections which map to this region present themselves as being strong candidate genes, mapping to the region where QTL peaks were detected in Chapter 4.

5.4 Major Genes Involved in Muscle and Fat Pathways

Growth is a complex integrated process that relies on interactions between nutrients, the environment, genotype, and is controlled via feedback loops involving different hormones and receptors in different tissues (Spencer, 1985). Traditional breeding methods have assumed an infinitesimal animal model, that states that genetic variation in any given trait (e.g. growth) is controlled by an infinite number of genes, each with a small contribution (Lynch & Walsh, 1998). However, as increasingly detailed studies of genes and their actions are carried out, it is apparent that for any given trait there may be "major" genes which have a large effect.

Major hormones affecting growth include: growth hormone, thyroid hormones, glucocorticoids, sex steroids, insulin and peptides (referred to as growth factors) (Spencer, 1985). In addition a number of other "non-hormonal" contributors have been

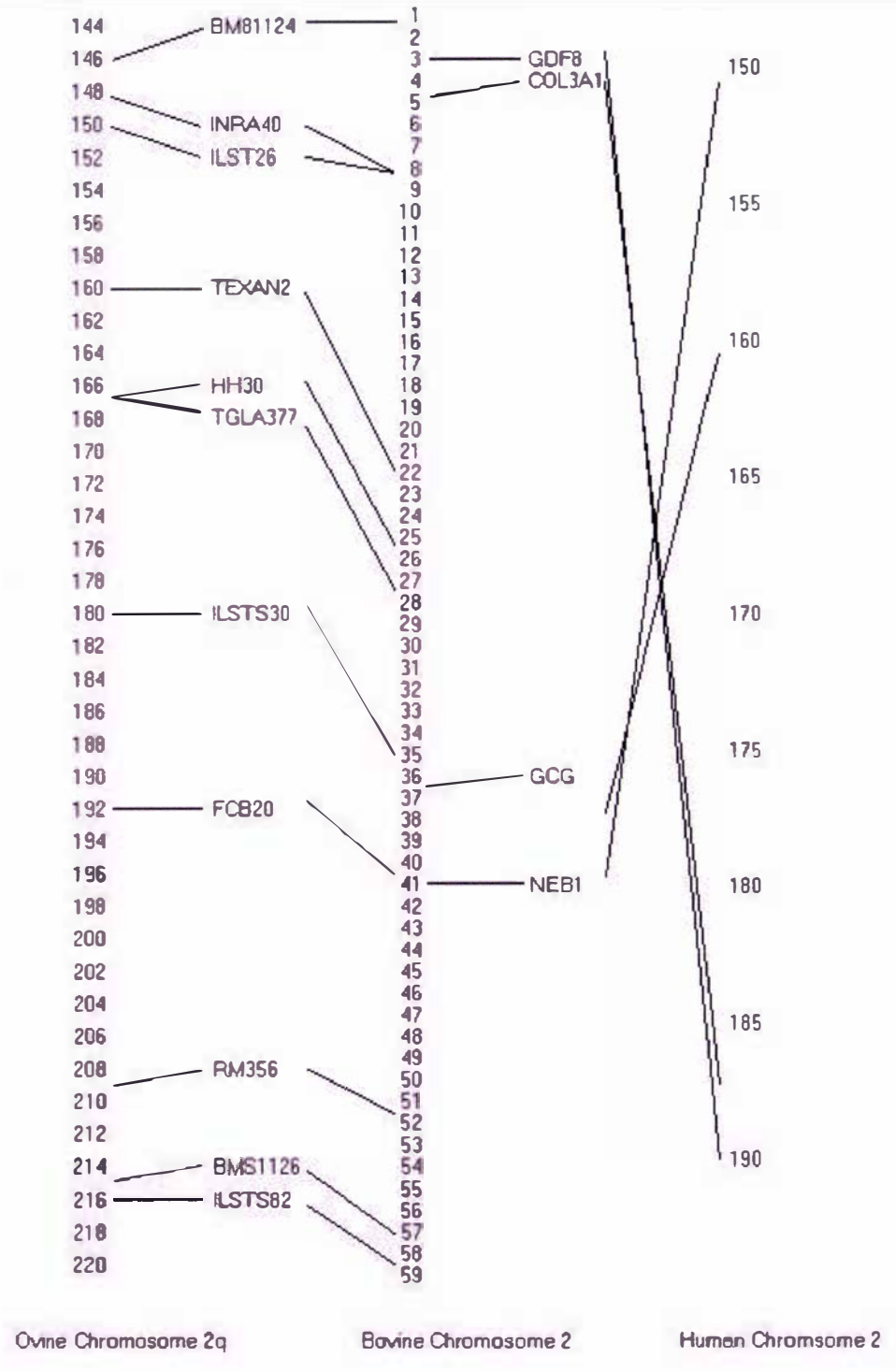


Figure 5.1 Maps showing comparative area of ovine chromosome 2q, bovine chromosome 2 and human chromosome 2. Distances for ovine and bovine are in centiMorgan, and for human in mega-base pairs (based on http://www.thearkdb.org/anubis; http://genome.uscs.edu October 2003).

identified as affecting growth in the form of muscle regulatory factors. For any one of these hormones or factors there are multiple genes coding, not only for the actual hormone/factor, but also for the receptors that bind the hormone to cause activation in a particular cell and the signalling pathways involved. There is currently considerable research being carried out into signalling pathways, and it is likely that in the near future

a much clearer picture of the interactions will exist. Mutations to any one of these genes could potentially have an impact on the growth and development of an animal.

5.4.1 Hormones

The somatotropic axis is involved in the regulation of the growth of muscle, fat and bone. Growth Hormone (GH or somatotrophin), is central to the somatotropic axis, and has both anabolic and catabolic effects (Florini et al., 1996; Etherton & Bauman, 1998). Release of GH from the anterior pituitary gland is predominately controlled by GH releasing hormone (somatocrinin) and GH release inhibiting factor (somatostatin) (Florini et al., 1996). Growth hormone is also involved with the activation of tyrosine kinase JAK2 (Janus kinase 2), which then binds to GH releasing hormone and is involved in a number of signalling pathways (Carter-Su et al., 1996). Other factors that influence plasma GH levels include free fatty acids, leptin and neuropeptide Y (Howard et al., 1996; Smith et al., 1996).

The insulin-like growth factor (IGF) family consists of three known ligands (IGF-I, IGF-2 and insulin), six characterized binding proteins (IGFBP-1 to 6) and associated receptors (Butler & Le Roith, 2001). Originally it was thought that the major growth promoting effect of GH were mediated via insulin-like growth factor 1 (IGF-1) produced exclusively from the liver (Daughaday et al., 1972), however, since then IGF-1 has been shown to be produced in most tissues, and so the role of autocrine/paracrine IGF-1 vs circulating IGF-1 is unclear (Le Roith et al., 2001). The predominant effect of IGFs is to mediate the anabolic effects of GH. A regulatory mutation in the insulin-like growth factor 2 (IGF2) gene has been shown to cause an increased muscling phenotype in pigs (Van Laere et al., 2003). Mutations to the IGF appear to be involved with overall growth (Bass et al., 1999).

Insulin is an anabolic hormone and acts to preserve nutrients in their storage forms by stimulating glycogenesis, lipogenesis, and glycerol synthesis and by inhibiting gluconeogenesis, glycogenolysis, and lipolysis (Brockman & Laarveld, 1986). Insulin concentrations have been shown to be positively correlated with the proportion of adipose tissue in the carcass and negatively correlated with the proportion of muscle in the carcass of adult cattle (Istasse et al., 1990; Hocquette et al., 1999).

Thyroid hormones such as triiodothyronine (T3) and thyroxine (T4) play an important role in post-natal growth of animals through stimulating oxidative metabolism and anabolic functions of cells by regulating oxygen consumption, mineral balance and the synthesis and metabolism of proteins, carbohydrates and lipids. To an extent these effects occur via an influence on GH (Samuels et al., 1979) and IGF (Furnlanetto et al., 1979) levels. Triiodothyronine concentrations have been shown to be positively related to the higher fat development in adult cattle (Hocquette et al., 1999).

Cortisol and corticosterone are glucocorticoids which are major catabolic hormones that inhibit muscle growth in immature animals, although they also have lipogenic actions (Daughaday et al., 1975). Such glucocorticoids have specifically been shown to decrease protein synthesis by affecting rates of translation (Sharpe et al., 1986).

Androgens and oestrogens are anabolic, growth promoting agents. Two possible modes of action for these hormones were proposed by Heitzman (1981). The first, is a direct effect on protein synthesis and/or degradation, mediated by direct entry into the muscle cells, the second is an indirect effect via other endocrine hormones of the hypothalamus, gonads, pancreas or the thyroid, which then exert an anabolic effect in muscle. Testosterone is a more potent stimulant of GH than the oestrogens (Jansson et al., 1985). Exogenous natural and synthetic estrogenic and androgenic steroid hormones are used commercially to stimulate metabolic processes associated with increased rate and efficiency of body growth in ruminants (Tucker & Merkel, 1987)

From the above discussion of hormones, it is apparent that control of growth is complex and that a disruption in the production of any of these hormones or their receptors could alter growth patterns. The chromosomal location of genes for some of these hormones and their receptors are in Table 5.1. With the exception of IGF-BP2 and IGF-BP5, none of these hormones or these receptors map to Chr2, and IGF-BP2 and IGF-BP5 map to 2q33-q34 and 2q33-q36, respectively, whereas the region of interest in this study is 2q32, a difference of approximately 30 mega base pairs (IGF-BP2 and IGF-BP5 map to 216 mega base pairs; region of interest 160-190 mega base pairs). Thus none of the major hormones or their receptors identified here are possible candidate genes.

Table 5.1 Human chromosomal locations of major hormones and their receptors involved in the growth pathway. Locations of genes were taken from http://bioinformatics.weizmann.ac.il/cards/

Hormone	Location on Human Chromosome
Growth Hormone 1	17q22-q24
Growth Hormone 2	17q22-q24
Growth Hormone Receptor	5p13-p12
Growth Hormone Releasing Hormone	20q11.2
Growth Hormone Releasing Hormone Receptor	7p15-p14
Somatostatin	3q28
Somatostatin Receptor 1	14q13
Somatostatin Receptor 2	17q24
Somatostatin Receptor 3	22q13.1
Somatostatin Receptor 4	20p11.2
Somatostatin Receptor 5	16p13.3
Janus kinase 2	9p24
Leptin	7q31.3
Leptin Receptor	1p31
Neuropeptide Y	7p15.1
Neuropeptide Y Receptor 1	4q31.3-q32
Neuropeptide Y Receptor 2	4q31
Neuropeptide Y Receptor 5	4q31-q32
Neuropeptide Y Receptor 6	5q31
Insulin-Like Growth Factor 1	12q22-q24.1
Insulin-Like Growth Factor 2	11p15.5
Insulin-Like Growth Factor 1 Receptor	15q25-q26
Insulin-Like Growth Factor 2 Receptor	6q26
Insulin-Like Growth Factor Binding Protein 1	7p14-p12
Insulin-Like Growth Factor Binding Protein 2	2q33-q34
Insulin-Like Growth Factor Binding Protein 3	7p14-p12
Insulin-Like Growth Factor Binding Protein 4	17q12-q21
Insulin-Like Growth Factor Binding Protein 5	2q33-q36
Insulin-Like Growth Factor Binding Protein 6	12
Insulin-Like Growth Factor Binding Protein 7	4q12
Insulin	11p15.5
Insulin Receptor	19p13.2
Androgen Receptor	Xq11-q12
Oestrogen Receptor 1	6q25.1
Oestrogen Receptor 2	14q

5.4.2 Muscle Regulatory Factors

The muscle regulatory factor (MRF) proteins are skeletal muscle tissue-specific transcription factors that activate skeletal muscle differentiation (Te Pas & Soumillion, 2001). The MRF gene family consists of four genes, MyoD (alternative names of MyoD1, or myf-3), myogenin (alternative name myf-4), myf-5, and MRF4 (also called myf-6 or herculin). MyoD and Myf-5 are expressed in proliferating myoblasts. The expression of Myf-5 precedes MyoD expression, whilst myogenin is expressed during differentiation (fusion) of myoblasts to myofibers. MRF4 is mainly expressed in postnatal skeletal muscle tissue, maintaining skeletal muscle fiber differentiation-specific phenotype. However, all contribute to postnatal hypertrophic skeletal muscle growth (Te Pas & Soumillion, 2001).

As reviewed by Te Pas and Soumillion (2001) knock out mice for these genes have insufficient muscle production, which is often lethal, thus demonstrating their importance. However, there were no reports of polymorphisms that merely altered the function of the genes.

Whether polymorphisms in any of these genes would lead to up-regulation of muscle production and the increased muscling phenotype observed is unclear, but in the absence of evidence they should be considered as candidate genes. The chromosomal locations of these MRF genes are presented in Table 5.2. None of the MRF genes map to human Chr2, the location of the candidate gene being sought and so it is unlikely that any of the MRF genes are the candidate gene involved in the observed phenotypes.

Table 5.2 Human chromosomal locations of the muscle regulation factors involved in the growth pathway

Muscle Regulation Factor	Location on Human Chromosome
Myogenic Differentiation Antigen 1	11q15.4
Myogenin	1q31-q41
Myogenic Factor 5	12q21
Myogenic Factor 6	12q21

5.5 Genes Known to Have a Direct Effect on Increasing Skeletal Muscle Mass and Decreasing Fat Mass

The approach taken in section 5.4 assumed that any candidate gene is likely to be a major gene/factor involved in the growth pathways of muscle, given the size of the effect. Although this was a suitable hypothesis, it is also possible that the gene may not generally have a large direct role in muscle growth, but that a mutation to it may result in a large effect. A non-muscle example is the Inverdale line of sheep, where a mutation to the BMP15 gene (also known as growth differentiation factor 9B) causes the increased ovulation rate associated with the Inverdales, yet BMP15 normally only plays a small role in reproduction (Galloway et al., 2002).

Online Mendelian Inheritance in Man (OMIM) is an online database tool that provides reference to known human genetic disorders (Hamosh et al., 2002). Through using search words related to a given phenotype, it will return a list of all genes where mutations to that gene cause the phenotype described, with subsequent links to further information about the gene. Thus, it can be used to search for genes/factors which result in the increased muscling phenotype observed in Chapter 4.

5.5.1 Search Words

The search words used in OMIM were "skeletal muscle hypertrophy"; "skeletal muscle hyperplasia", "reduced adipose" and "reduced fat" as these broadly reflected the phenotypes observed in Chapter 4.

5.5.2 Documented Genes Affecting Skeletal Muscle

For any of the search words described above, there were no direct "hits" for the phrases. Rather OMIM searched for entries that contained all of the individual words.

A search using "skeletal muscle hypertrophy" or "skeletal muscle hyperplasia" achieved 94 "hits" of genes where mutations to the gene have been associated with changes in muscle either in humans or in animal models (predominately mice). The full list of genes is given in Appendix 8.14. Of these, a number of the mutations were not directly related to skeletal muscle, rather a number referred to mutations with an effect on cardiac muscle, but also had reference to "skeletal" in the text. As a result only six genes, fitted the criteria of having a mutation leading to an effect on skeletal muscle, these genes and the resulting skeletal muscle phenotype are presented in Table 5.3.

Table 5.3 Details on candidate genes identified as having documented mutations (Online Mendelian Inheritance in Man) which result in changes to skeletal muscle

Abbreviation	Name	Description of Skeletal Muscle Effect	Position
HSPG2	Heparan Sulfate Proteoglycan of Basement Membrane	Skeletal dysplasia	*1q36.1*
GDF8	Growth Differentiation Factor 8	Skeletal muscle hypertrophy	*2q32.1*
CLCN1	Chloride Channel 1; skeletal muscle	In mouse model, when CLCN1 knocked out, muscle hypertrophy	*7q35*
IGF1	Insulin Growth Factor 1	Deletion leads to growth retardation with deafness or mental retardation	*12q22-q24.1*
LGMD2C	Muscular Dystrophy, Limb-Girdle, Type 2C	Muscular dystrophy	*13q12*
DMD	Dystrophin	Deteriation of muscle tissue; calf muscle hypertrophy	*Xp21.2*

Of the genes presented in this table, only GDF8 maps to Chr2 and specifically the region of interest. Of the remaining genes only Chloride Channel 1; skeletal muscle (CLCN1), which maps to 7q35, actually describes an increased muscling phenotype. However, upon reading further about this gene, it is actually associated with Myotonia congenita which is a non-dystrophic muscle disorder causing muscle stiffness (Sun et al., 2001) and is therefore unlikely to be a candidate gene.

5.5.3 Documented Genes Affecting Fat Development

For any of the search words described above, there were no direct "hits" for the phrases. Rather OMIM searched for entries that contained all of the individual words.

A search using "reduced adipose" and "reduced fat" achieved 40 "hits" of genes where mutations to the gene have been associated with changes in fat either in humans or in animal models (predominately mice). The full list of genes is given in Appendix 8.14. Of these, however, a number of the mutations were associated with increased fat deposition leading to obesity. As a result only 22 genes fitted the criteria of having a mutation which leads to a decrease in fat, these genes and the resulting fat phenotype are presented in Table 5.4.

Table 5.4 Details on candidate genes identified as having documented mutations (Online Mendelian Inheritance in Man) which decrease fat accumulation

Abbreviation	Name	Description	Position
PRKAR2B	Protein Kinase, cAMP-Dependent, Regulatory, Type II Beta	Knock-out mice have decreased fat	*1p31*
HSD11B1	Hydroxysteroid (11-beta) dehydrogenase 1	Mutations associated with increased fat catabolism	*1q32-q41*
GDF8	Growth Differentiation Factor 8	Knock-out mice have decreased adipogenesis	*2q32.1*
IRS1	Insulin Receptor Substrate 1	Mutations lead to decreased fat	*2q36*
GHR	Growth Hormone Receptor	Mutations causing over expression lead to decreased fat	*5p13-p12*
IL6	Interleukin 6	Mutations lead to decreased fat	*7p21*
CREBBP	Creb-Binding Protein	Mutations lead to decreased fat	*8q*
INS	Insulin	Mutations lead to decreased fat	*11p15.5*
UCP3	Uncoupling Protein 3	Mutations causing over expression lead to decreased fat	*11q13*
UCP2	Uncoupling Protein 2	Mutations associated with obesity	*11q13*
BSCL	Bernardinelli-Seip Congenital Lipodystrophy	Mutations lead to decreased fat	*11q13*
ACACB	Acetyl-CoA Carboxylase-Beta	Knock-out mice have decreased fat	*12q24.1*
KL	Klotho	Mutations lead to decreased white fat	*13q12*
CYP19A1	Cytochrome P450, Family 19, Subfamily A, Polypeptidel	Also known as eostrogen synthase, knock-out mice have high fat levels	*15q21.1*
PLIN	Perilipin	Knock-out mice have decreased fat	*15q26*
FOXC2	Forkhead Box C2	Mutations causing over expression lead to increased brown fat	*16p13.3*
LEPR	Leptin Receptor	Mutations lead to obesity	*16q24.3*
SREBF1	Sterol Regulatory Element-Binding Transcription Factor 1	Mutations causing over expression lead to decreased fat	*17p11.2*
GH1	Growth Hormone 1	Mutations causing over expression lead to decreased fat, whilst other mutations associated with obesity	*17q22-q24*
INSR	Defect in Insulin Receptor - Leprechaunism	Mutations lead to decreased fat	*19p13.2*
NCOA2	Nuclear Receptor Coactivator 2	Knock-out mice have decreased fat	
GPR24	G Protein-Coupled Receptor 24	Knock-out mice have decreased fat	*22q13.3*

The only genes presented in this table that map to Chr2 are GDF8 and Insulin Receptor Substrate 1 (IRS1), but only GDF8 maps to the specific region of interest. All of the remaining genes are associated with decreased fat production, but none directly referred to an associated increase in skeletal muscle mass.

5.6 Lexical Analysis

Lexical analysis is the study of words. It has previously been used to determine key words relating to the function of a gene in terms of protein regulation using relevant abstracts (Andrade & Valencia, 1998). The lexical analysis package used for this analysis was WordSmith (http://www.oup.com). The program is made up of three main tools: WordList, KeyWords and Concord. Wordlist creates a word frequency list for a given text or texts. KeyWords compares a user submitted word list with a used defined reference list and reports "Key Words" by comparing the frequency of a "search" word in the submitted and reference lists. Concord provides concordance for a "search" word, providing all instances of that word and those around it, thereby providing an idea of its context.

Given the nature of the QTL identified, the "search" words used in this lexical analysis were *muscle*, *lean*, *fat*, *adipose*, *hypertrophy*, *hyperplasia* and *growth*. Word lists were generated for each gene text file (all abstracts for the gene). To avoid frequent words such as "the, a, in" a *stop* list was used to exclude them, this list was created using the 1000 most common words of the British National Corpus (Leech et al., 2001). In addition *pmid*, *pubmed*, *medline*, and *publication* were added, as these words occurred within each abstract due to the source of the abstracts. Each list was lemmatised (words joined that belong together) using a text file, in this case the words joined were muscle (muscles, muscling, muscular, muscularity); adipose (adipocyte, adipocytes) and hypertrophy (hypertrophied). Word lists were sorted by frequency of word to identify the most frequently occurring words. The reference list used to identify *KeyWords* for each gene was based on a list created using all the abstracts for all genes. Within *Concord* the "search" words were used as search criteria for each gene, approximately five words either side of the search word were requested. The results of the concordance query were manually inspected to identify irrelevant entries.

5.6.1 Sourcing Gene List

The University of California, Santa Cruz Genome Bioinformatics website, (http://www.genome.ucsc.edu/, June 2002) was used to identify genes mapped to the region of interest on human Chr2 (160 – 190 mega base pairs). To eliminate the problem of alternative names for the same gene, the GeneCards™ website (http://bioinformatics.weizmann.ac.il/cards/), was used to create a list of alternative names for each gene. Each gene and its alternative names were used as search criteria within PubMed (http://www.ncbi.nih.gov/entrez/query.fcgi), the biomedical sciences literature search engine. Abstracts for each gene were saved as a text file.

The search within the USCS Genome Bioinformatics website identified 123 genes, of which only 78 had approved names in GeneCards (the remaining 45 only had identification numbers). 3515 papers related to the 78 approved genes were identified in PubMed (range 1-735). Details of potential candidate genes are presented after a discussion of the relevant results in Table 5.7 and Table 5.8, with the remainder of the 78 genes (non-candidates) found in Appendix 8.15. The average number of abstracts per gene was 45.

5.6.2 Word Lists

Using a stop list to eliminate common words is an important editing process. The results of Andrade and Valencia (1998), showed the most common words, by number of occurrences across the texts and mean number of occurrences within the texts, were "of", "the" and "and". The inclusion of such words will distort any analysis.

Graphing the results of the word lists (Figure 5.2) shows the most frequent words (i.e. those that occur in a high proportion of abstracts about a gene or have a high frequency within a small number of genes) and where the "search" words were. The word that occurred in the largest number of lists with moderate frequency within these lists was "gene". The word that had the greatest mean number of occurrences (about 450) in its respective list was proglucagon, reflecting the high number of abstracts on this gene (300). *Growth* occurred in the most number of gene lists (40) of the words relevant to this study, with most only occurring in up to 10 of the texts, suggesting that there was potential for narrowing the range of putative candidate genes.

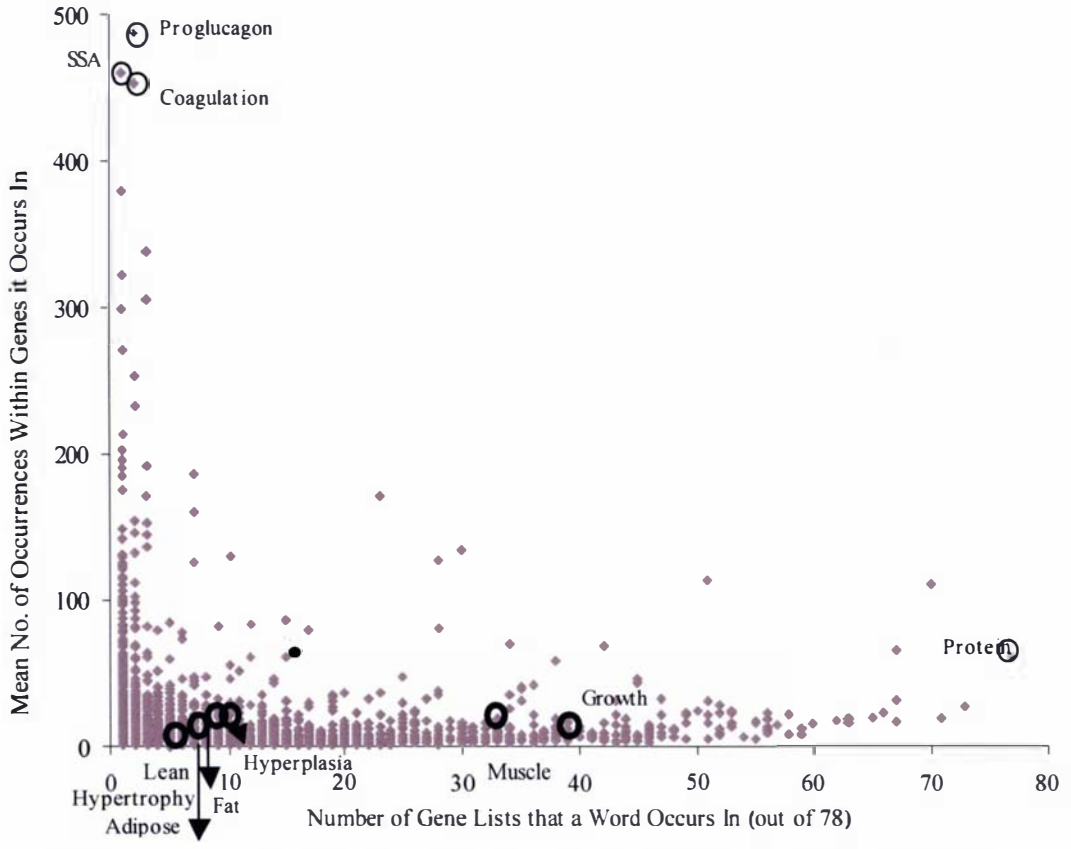


Figure 5.2 Distribution of words in abstracts of 78 genes from a region of human Chr. 2. Frequent words (occur in many lists or at a high frequency in only a few lists) are presented in light ovals. Words describing the desired attributes of a candidate gene are in dark ovals.

5.6.3 Key Words

The purpose of *KeyWords* is to compare the frequency of a word in the submitted list, with the reference list, using chi-squared analysis, where the information provided is the number of occurrences of each word relative to the total number of words for that list. For example the word 'muscle' occurred 124 times within the GDF8 word list which contained 7541 words in total, and 371 times within the reference list which contained 929,928 words. Chi-squared analysis of these numbers produces a likelihood ratio chi-squared value of 643 ($P<0.001$). A high likelihood ratio chi-squared value indicates a words uniqueness to that gene, and it is reported as a key word. Four genes had as a key word, the "search" words outlined in this study, these genes and their ten most frequent key words are presented in Table 5.5. The only gene to have a "search" word as the most unique keyword was GDF8, likewise it was the only gene to have two "search" words in the top ten key words. However, any gene which has a "search" word as a keyword (irrespective of its ranking) must be considered as a potential candidate gene. Therefore, GDF8, NAB1, PDK1 and TTN should all be considered as candidate genes.

Table 5.5 Results from KeyWord (WordSmith Tool) for genes with a "search" word as a keyword. The likelihood ratio chi-squared value is given in brackets, the higher this value the more unique the word is to that gene

Key Word	Gene Abbreviations[a]							
(ranking)	GDF8		NAB1		PDK1		TTN	
1	Muscle[b]	(643)	Egr	(561)	Pyruvate	(536)	titin	(285)
2	Myostatin	(480)	Nab	(222)	dehydrogenase	(437)	ttn	(264)
3	mstn	(431)	Ngfi	(214)	Pdk	(322)	muscle	(110)
4	cattle	(169)	Gnrh	(115)	isoenzymes	(258)	mdm	(102)
5	mh	(137)	Transcriptional	(95)	Pdc	(136)	dystrophy	(82)
6	growth	(118)	Mkp	(85)	phosphorylation	(124)	cardiomyopathy	(80)
7	breeds	(110)	Cocaine	(85)	starvation	(102)	nebulin	(78)
8	double	(85)	Growth	(77)	Ppar	(89)	dilated	(78)
9	belgian	(72)	Corepressors	(72)	Muscle	(89)	neb	(70)
10	blue	(71)	corepressor	(69)	Fed	(70)	tmd	(66)

[a] GDF8-Growth Differentiation Factor 8; NAB1 – NGFI-A binding protein (EGR1 binding protein 1); PDK1 – Pyruvate dehydrogenase kinase, isoenzyme1; TTN - Titin

[b] Words describing the desired attributes of the candidate gene being sought are highlighted

5.6.4 Concordance

The Concord Tool was used to examine the "search" words in their context within the abstracts. Through using this tool, genes that include "search" words, in the correct context, can be identified. This approach considers every entry of a "search" word. There were 1032 occurrences of the "search" words across all genes, however, many were not in the correct context. The majority of references to muscle were in the context of "smooth muscle" (intestinal muscle) (e.g. ITGA4). Given that the QTL identified affects skeletal muscle, genes impacting only on smooth muscle are unlikely to be the gene involved with this QTL. There were a number of derivatives of growth including tumor growth (FAP), epidermal growth (WASPIP), growth of cultures on plates (WASPIP) all of which did not fit with the likely attributes of any candidate gene.

Exclusion of words in the incorrect context left only 393 occurrences of "search" words across the genes. Only 24 of the original 78 genes contained the "search" words in what appeared to be an appropriate context. Thus, from the total number of occurrences it was obvious that some words occurred frequently within certain abstracts, examples of the occurrences of the "search" words for these 24 genes are provided in Table 5.6.

From Table 5.5 NAB1 was considered a candidate gene for its high frequency of the "search" word "growth", however, concordance showed all instances of this word were not in the correct context, with the majority referring to "filamentous growth", and therefore this gene does not appear in Table 5.6.

Details of the 24 genes identified as being possible candidates are provided in Table 5.7.

Table 5.6 Examples from the concordance study of genes in the region of interest. The 24 shown are examples for each "search" word that occurred in the correct context for a gene (if a word occurred multiple times within a gene one example of the context in which it was used is presented)

Gene	Words Surrounding "*Search Word*"
ATF2	important in the regulation of cell *growth* and apoptosis, including activation
	levels were found in heart and skeletal *muscle*. Like p38, p38-2 is activated by
CED-6	cers. AKT2 is activated by a variety of *growth* factors and insulin via
	APPL is highly expressed in skeletal *muscle*, heart, ovary and pancreas,
COL3A1	including transcription factors and *growth*-regulatory genes that had not
DIRC1	in fetal kidney, spleen, and skeletal *muscle*. A GFP-Dirc1 fusion protein was
FRZB	and dysregulation of sFRP-2 in *fat* and obesity also suggest a potential
	high levels of hFRP-2 were found in *adipose* tissue. In addition, low levels
	have been implicated in regulating *growth* and pattern formation in a varied
	shortened trunks and decreased skeletal *muscle*. This phenotype is nearly identic
GAD1	receptor alpha (RARA), transforming *growth* factor beta3 (TGFB3), and msh
GCG	the diabetic and obesity phenotype in *fat/fat* mice. PMID: 11375130 [PubMed
	also regulate glycogen synthesis in *adipose* tissue and *muscle*; however, the
	in a syndrome of severe postnatal *growth* impairment and multiple defects
	These data indicate that the skeletal *muscle* is one of the target tissues for
	rats (17.0 +/- 2.8 pmol) compared with *lean* Zucker rats (12.4 +/- 1.8 pmol). In
GDF8	mice have a significant reduction in *fat* accumulation with increasing age
	20%, due to general skeletal-*muscle hyperplasia*-that is, an increase in the
	skeletal *muscle hypertrophy* and/or *hyperplasia*. MSTN orthologues have been
	is a negative regulator of *muscle growth* in cattle as well as mice. PMI
	as a negative regulator of skeletal *muscle growth*, suggests that it may sim
	relationship with the marked difference in *lean* meat mass between Western and
GRB14	issues such as human skeletal *muscle* and *fat* cells. Here we report the cloning
	including skeletal *muscle*, liver, and *adipocyte* cells. hGrb10gamma is also
	cDNA library, we identified the human *growth* factor receptor bound 14 (hGrb14)
	family (hGrb10gamma) from human skeletal *muscle* and its localization to human
HAT	stone H4. hat1 mutants have no obvious *growth* defects or phenotypes other than
	is expressed mainly in skeletal *muscle*, heart and brain, whereas citrin
	was expressed in diaphragm, skeletal *muscle*, heart, brain and kidney, but not
HOXD13	role for cFHF-2 in the patterning and *growth* of skeletal elements is implied
NEUROD1	adrenocorticotropic hormone and *growth* hormone concomitantly: aberrant
	H factor, neuroD1/BETA2, but not by the *muscle* bHLH factor, MyoD. However, the
PDE11A	DE11A1 was mainly detected in skeletal *muscle*. The human PDE11A gene was invest
PDE1A	was present in both heart and skeletal *muscle*. Multiple forms of human PDE1A a
PDK1	predominantly expressed in skeletal *muscle* and heart. The specific activities
SARCOSIN	increased expression of skeletal *muscle* alpha-actin (ACTA1), myosin light
SCN1A	episodic dysfunction of heart, skeletal *muscle*, and brain. Lossin and colleagues
SLC11A3	large effects on glycogen content in *muscle* and meat quality. Ten microsatellite
STK39	expression starts in the exponential *growth* phase and precedes that of the Ni
TANK	Inhibition of transforming *growth* factor beta signaling and Smad-
	heart, liver, placenta, brain, skeletal *muscle*, kidney and the pancreas;
TFPI	followed by the heart, adrenal, and *adipose* tissue. Since little has been
	an tissues, such as the liver, skeletal *muscle*, heart, kidney, and pancreas.
TTN	approximately 2 mD is expressed in skeletal *muscle*, but western blot studies with
UBE2E3A1	strong 1.9-kb band of UBE2E3 in skeletal *muscle*. Recombinant fusion protein of GS
ZAK	of several cardiac and skeletal *muscle* genes. Previously, we identified

Table 5.7 Details on candidate genes identified through lexical analysis

Abbreviation	Name	Description	Position
ATF2	activating transcription factor 2	Binds the camp response element. A sequence present in many viral and cellular promoters	*2q31.1*
*CED-6**	*No name*		*2q32.2*
*COL3A1**	collagen, type III, alpha 1	Collagen Type III occurs in most soft connective tissues along with Type I Collagen	*2q32.2*
DIRC1	disrupted in renal carcinoma 1		*2q32.2*
*FRZB**	frizzled-related protein	May be involved in morphogenesis of skeleton	*2q32.1*
GAD1	Glutamate Decarboyxlase 1	Catalyzes the production of GABA	*2q31.1*
GCG	glucagon	Glucagon promotes hydrolysis of glycogen and lipids, and raises the blood sugar level	*2q24.2*
GDF8	growth differentiation factor 8	Acts specifically as a negative regulator of skeletal muscle growth	*2q32.2*
GRB14	growth factor receptor-bound protein 14	Inhibits (by interaction) the cytoplasmic domain of the autophosphorlyated insulin receptor	*2q24.3*
*HAT1**	Histone acetyltransferase1	May play a role in telomeric silencing. HAT1 has intrinsic substrate specificity that modifies lysine in recognition sequence GXGKXG.	*2q31.1*
HOXD13	Homeobox D13	Sequence-specific transcription factor which is part of a developmental regulatory system that provides cells with specific positional identifiers on the anterior-posterior axis	*2q31.1*
NEUROD1	neurogenic differentiation 1	Acts as a differentiation factor during neurogenesis. Transcriptional activator.	*2q31.3*
PDE11A	phosphodiesterase 11A		*2q31.2*
PDE1A	phosphodiesterase 1A, calmodulin-dependent	Has a higher affinity for CGMP than for CAMP	*2q32.1*
*PDK1**	Pyruvate dehydrogenase kinase, isoenzyme1	Inhibits the mitochondrial pyruvate dehydrogenase complex, thus contributing to the regulation of glucose metabolism	*2q31.1*
*SARCOSIN**	Sarcomeric muscle protein		*2q31.1*
*SCN1A**	Sodium Channel, Voltage-gated, type1, alpha polypeptide	This protein mediates the voltage-dependent sodium ion permeability of excitable membranes.	*2q24.3*
*SLC11A3**	solute carrier family 11 (proton coupled divalent metal ion transporters), member 3		*2q32.2*
STK39	Serine theronine kinase 39	May act as a mediator of stress-activated signals	*2q24.3*
*TANK**	TRAF- family member-associated NFKB activator	Acts as a regulator of TRAF function by maintaining them in a latent state.	*2q24.2*
*TFPI**	tissue factor pathway inhibitor (lipoprotein-associated coagulation inhibitor)	It possess an antithrombotic action and also the ability to associate with lipoproteins in plasma	*2q32.1*
*TTN**	Titin		*2q31.2*
UBE2E3	ubiquitin-conjuugatin enzyme E2E3		*2q31.3*
ZAK	sterile alpha motif and leucine zipper containing kinase AZK		*2q31.1*

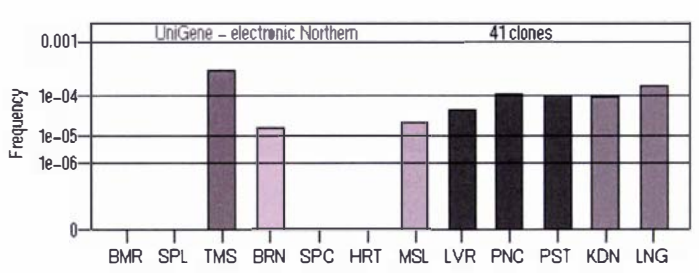
* these genes are also expressed at least in skeletal muscle (refer Table 5.8)

5.7 Tissue Expression

Whether or not a gene is expressed in any given tissue provides evidence as its likely involvement, with either the growth, development or function of the tissue. One of the features of the GeneCards™ website are enriched expression profiles (Safran et al., 2003). These expression profiles focus on normal human tissues: bone marrow, spleen, thymus, brain, spinal cord, heart, skeletal muscle, liver, pancreas, prostate, kidney and lung. Data are collected on these tissues from NCBI's Unigene dataset (*Hs. Data*), which is a system for automatically partitioning GenBank mRNA sequences and Expressed Sequence Tags (EST's) into non-redundant sets of gene-oriented clusters (Schuler, 1997; Wheeler et al., 2002). This data set is mined for information about the number of unique EST clones per gene per tissue. The results used for this study were from the February 2003 downloaded results (based on Build 155 *Homo sapiens*). The results are calculated by dividing the number of EST clones per gene by the number of clones per tissue which are then expressed on a root scale (Schuler, 1997; Wheeler et al., 2002). If any of the genes from the equivalent human region identified in Table 5.7 are expressed in skeletal muscle, it would provide additional evidence to that gained from the lexical analysis that the gene plays an important role in that tissue.

5.7.1 Example of Tissue Expression Profile

An example of expression profiles are shown in Figure 5.3. Figure 5.3 shows the tissues in which gene CIR is expressed, in this instance it is expressed in seven of the twelve tissues including skeletal muscle which is the tissue of interest for this work. That no expression was seen in, for example, the spleen, does not mean that it is not expressed in that tissue, just that its level of expression may be low and was thus not detected, or that as the total number of clones for this tissues is 13,489, it has not been fully tested. If in contrast a gene is not shown to be expressed in the brain, it is more likely that it is not actually expressed in the brain given that the total number of clones tested on the brain is 274,393.



Tissue		Clones per gene	Total clones
BMR	Bone marrow	0	22,540
SPL	Spleen	0	10,567
TMS	Thymus	2	6,376
BRN	Brain	2	122,062
SPC	Spinal cord	0	574
HRT	Heart	0	5,047
MSL	Skeletal muscle	1	45,723
LVR	Liver	4	86,604
PNC	Pancreas	6	55,487
PST	Prostate	6	58,380
KDN	Kidney	6	65,470
LNG	Lung	14	86,529

Figure 5.3 Example of the enriched expression profile provided by the GeneCards™ website for gene CIR, showing that the gene is expressed in a number of different tissues

5.7.2 Candidate Gene Expression in Skeletal Muscle

The enriched protein expression profiles were downloaded for each of the 78 genes in the region of interest (discussed 5.6.1) and whether or not expression of the gene in skeletal muscle occurred noted, these were then matched up the lexical analysis results.

Thirty-five of the 78 genes in the region of interest showed evidence of skeletal muscle expression. However, of these, only eleven genes also had relevant abstracts relating to their function. The remaining 24 genes, although showing evidence of being expressed in skeletal muscle, do not have supporting abstracts (Table 5.8). Of these 24 genes, one (AGPS) did not have any abstracts relating to it and a further 14 had less than ten abstracts, which suggests that relatively little is known about their function and they could therefore be involved in skeletal muscle growth and development or function once further research is carried out. Of the eleven which showed expression and had relevant abstracts, four also appeared in the original concordance results (CALCRL, FAP, ITGA4 and NAB1), however, their reference was to smooth not skeletal muscle.

Of the genes identified via lexical analysis as being involved with skeletal muscle growth (24), 13 did not show evidence for being expressed in skeletal muscle, of these, however, four did not have any clones expressed in any of the tissues. That there is no current evidence from these clones that these 13 genes are expressed in skeletal muscle does not mean that they are not expressed in this tissue. Rather it could be that no clones derived from skeletal muscle, to date, provide matches to these genes. It is possible that clones produced in the future could yet provide a match. Also the time of expression (e.g. pre-natal or post-natal) will influence whether or not results are seen, because the clones are based on EST's which are mRNA derived, as the above are based on adult tissues and the results are therefore limited to genes expressed post-natally.

Growth differentiation factor 8 is in fact an example of one gene which is predominantly expressed pre-natally with limited post natal expression (Shibata et al., 2003).

Table 5.8 Details of candidate genes identified through tissue expression profiles from GeneCards™ [a] as being expressed in skeletal muscle

Abbreviation	Name	Description	Position
AGPS	alkylglycerone phosphate synthase		2q31.2
ATP5G3	ATP synthase, H+ transporting, mitochondrial F0 complex, subunit c (subunit9) isoform 3	This protein is one of the chains of the nonenzymatic membrane component of mitochondrial ATPase	2q31.1
CALCRL	calcitonin receptor-like	Receptor for calcitonin gene-related peptide type 1.	2q32.1
CIR	CBF1 interacting corepressor		2q31.1
FAP	Fibroblast Activation Protein, Alpha	May have a role in tissue remodelling during development and wound healing.	2q24.2
GALNT3	UDP-N-Acetyl-Alpha-D-Galactosamine:polypeptide N-acetylgalactosaminyltransferase 3		2q24.3
GLS	glutaminase	Catalyzes the first reaction in the primary pathway for renal catabolism of glutamine.	2q32.2
HOXD8	Homeobox D8	Sequence-specific transcription factor, part of a developmental regulatory system.	2q31.1
IGRP	Islet-specific glucose-6-phosphate catalytic subunit-related protein		2q31.1
INPP1	inositol polyphosphate-1-phosphatase		2q32.2
ITGA4	integrin, alpha 4		2q31.3
ITGA6	Integrin, alpha 6	Receptor for laminin on platelets.	2q31.1
ITGAV	integrin, alpha V	Receptors for vitronectin, cytotactin, fibronectin, fibrinogen, laminin, matrix.	2q32.1
LRP2	Low density lipoprotein-related protein 2	Binds specifically clustering with high affinity, but also ligands in common with other family members .	2q31.1
NAB1	NGFI-A binding protein (EGR1 binding protein 1)	Acts as a transcriptional repressor for zinc finger transcription factors EGR1 and EGR2.	2q32.2
NCKAP1	NCK-associated protein 1	Associates with the first SH3 domain of NCK	2q32.1
NFE2L2	nuclear factor (erythroid-derived 2)- like 2	May be involved in the transcriptional activation of genes of the beta-globin cluster.	2q31.2
PMS1	postmeiotic segregation increased 1 (S. cerevisiae)	Probably involved in the repair of mismatches in DNA.	2q32.2
PPIG	Peptidyl-prolyl isomerase G		2q31.1
PRKRA	protein kinase, interferon-inducible double stranded RNA dependent activator		0
SCN3A	Sodium Channel, Voltage-gated, type III, alpha polypepetide	This protein mediates the voltage-dependent Na+ permeability of excitable membranes.	2q24.3
SLC25A12	Solute carrier family 25 (mitochondrial carrier, Aralar), member 12	Calcium-dependent mitochondiral solute carried. High levels in heart and skeletal muscle, low in brain and very low in kidney.	2q31.1
SB	Sjogren syndrome antigen B (autoantigen La)	LA protein plays a role in the transcription of RNA polymerase III.	2q31.1
SSFA2	sperm specific antigen 2	Sperm surface antigen.	2q32.1

[a] Additional candidate genes were found, however, their details can be found in Table 5.7, these genes were CED-6, COL3A1, FRZB, HAT1, PDK1, SARCOSIN, SCN1A, SLC11A3, TANK, TFPI and TTN

5.8 Follow up of Candidate Genes

Both the review of genes involved with the major pathways influencing with muscle development and a search in OMIM for genes where mutations to the genes are known to impact on skeletal muscle growth or fat deposition revealed GDF8 as the only high quality candidate gene. Lexical analysis identified 24 genes which warranted further investigation including GDF8, and tissue expression results added a further 24 which were not identified via the lexical analysis. The abstracts relating to these 48 genes were read to further determine their relevance to the candidate gene search. Because of the content of the search engine, PubMed, most related to well-studied species e.g. *Homo sapiens*, *Mus musculus* and *Xenopus*, although reference to other species such as cattle do occur occasionally.

Of these 48 genes, only three provided evidence of involvement in skeletal muscle growth or differentiation, all three of which were detected in the lexical analysis and two of these also appeared within the tissue expression profiles. Two of the genes FRZB and TTN are unlikely to be the candidate gene involved in the phenotypes seen (see discussion below), with the third, GDF8, having previously been considered a candidate gene for the phenotype. The other 45 were tentatively excluded, due to the content of the abstracts. The most frequent reason for exclusion was because the majority of their functions were related to either cancer (e.g. ATF2 and DIRC1) and/or immune response situations (GAD1 and TANK) with no discussion of a direct effect on skeletal muscle growth or development.

The titin gene (TTN) maps to human chromosome 2q31.2 or 177,453,253-177,734,685 base pairs) and is a giant, filamentous protein expressed in all vertebrate striated muscles (Labeit & Kolmerer, 1995). No reports of mutations to the TTN gene resulting in increased muscling have been found, however, researchers have reported mutations to TTN that are associated with muscular dystrophy in humans which results in progressive muscular degeneration (Fougerousse et al., 1998; Garvey et al., 2002). Whether or not alternative mutations to this gene could result in increased muscling is unclear, however, given the active role of TTN from early embryonic stages (Fougerousse et al., 1998) and its known involvement in muscle development its potential involvement in an increased muscling phenotype should be considered.

The frizzled-related protein gene (FRZB or FRZB-1) maps to human chromosome

2q32.1 or 182,382,364 bp to 182,414,562 bp. This gene has an antagonistic effect on the family of WNT genes (Borello et al., 1999). The WNT genes are a family of genes that encode secreted glycoproteins that modulate cell fate and behaviour in embryos through activation of receptor-mediated signalling pathways (Moon et al., 1997). FRZB-1 exerts strong influences on skeletogenesis and is a powerful and direct modulator of chondrocyte maturation, phenotype and function (Enomoto-Iwamoto et al., 2002). Misexpression of Frzb-1 in cultured chondrocytes shows that it boosts expression of traits associated with the immature phenotype, but inhibits traits associated with maturation, resulting in shortening of skeletal elements (Enomoto-Iwamoto et al., 2002). Over-expression of Frzb-1 *in vivo* has also been shown to strongly reduce and even abolish myogenesis (Borello et al., 1999). Whether or not alterative mutations to the Frzb-1 gene could result in increased muscling have not been reported, however, given its active role in skeletal and muscle development its potential role in an increased muscling phenotype must be considered.

Growth Differentiation Factor 8, a member of the transforming-growth-factor-β-family, in contrast to the two genes discussed above, does have known effects that result in an increased muscling phenotype. The effects of GDF8 have previously been discussed in Chapter 4. Briefly the generalised role of GDF8 is that of a negative regulator of skeletal muscle growth, with it being expressed specifically in developing and adult skeletal muscle (McPherron et al., 1997). Knocking out of the GDF8 gene was carried out in mice and caused a widespread increase in skeletal muscle mass (McPherron et al., 1997). A naturally occurring mutation to the GDF8 gene was subsequently shown to be responsible for the double-muscling phenotype in cattle (McPherron & Lee, 1997). A similar mutation (compact) has been demonstrated in mice (Varga et al., 1997; Szabo et al., 1998)

Of the three possible candidate genes identified in the relevant region of human Chr2, GDF8 presents itself as the most likely candidate in that there is evidence from two other species (mice and cattle) that mutations to this gene result in an increased muscling phenotype (Charlier et al., 1995; Varga et al., 1997; Grobet et al., 1998; Marcq et al., 1998; Szabo et al., 1998; Varga et al., 2003). However, despite evidence for a location association being presented by Marcq et al. (1998; 2002), they also sequenced GDF8, comparing the sequence of the Texel with that of the Romanov (control) and

found no coding differences between the two. This brings into question whether GDF8 is actually involved with the increased muscling seen. To date only the coding region of the gene has been sequenced, and it could yet be that there are mutations in the promoter or repressor regions which could be having an effect (as discussed in 5.2). Some of these regions could be some distance from the promoter coding region, therefore further work needs to be carried out with GDF8 and its surrounding sequence to fully establish whether or not it is the gene involved.

5.9 Assessment of the Relative Merits of Various Methods for Searching for Candidate Genes

Four methods of candidate gene identification have been presented in this chapter, providing a comprehensive search for candidate genes. The first two involved identifying genes known to influence muscle growth through known pathways and a search in OMIM, and then identifying whether or not any of the identified genes mapped to the region of interest identified. The other two, involved identifying all of the genes in the region of interest and using either lexical analysis or tissue expression profiles to identify likely candidates. All methods have advantages and disadvantages.

Attempting to identify the genes involved in the major pathways of skeletal muscle growth was somewhat difficult as there are very few recent reviews which cover all protein coding genes and signalling pathways involved, which made it a time consuming procedure to collect an up-to-date list of genes. Additionally this approach is likely to have excluded a number of "related" genes that are involved in the pathways.

Using OMIM to search for genes was problematic, in that it was hard to accurately determine "search phrases". For example the use of "skeletal muscle hypertrophy" yielded no "hits" and therefore, the program searched for the occurrence of each individual word, yielding a number of descriptions that referred to cardiac muscle hypertrophy, but elsewhere in the article referred to "skeletal". There is also the problem, that no two genes had the same phrases to describe the same thing, for example the description of GDF8 referred to "increased skeletal muscle mass" whilst others referred to "muscle cell hypertrophy". Thus the success of this approach was limited to the coverage of the "search phrases" used. Provided accurate "search phrases" could be determined though, it provides examples where mutations to genes

have resulted in the phenotype of interest.

Using Lexical Analysis to analyse the abstracts of genes in the region of interest, had both advantages and disadvantages. The major advantages are that it is comprehensive, and relatively easy to update and rerun (see 5.11), and has the potential to be automated. Its disadvantages are that it relies on genes having been mapped to the area, and that such mapped genes have known function. It is also extremely "sensitive" and picks up the occurrence of any "search" word, therefore time is required to manually review the results in the Concord feature to ensure that the "search" words are in context.

Using tissue expression data to determine genes in the region of interest that are also expressed in skeletal muscle likewise has advantages and disadvantages. The major advantage of this approach is that it is independent of the literature written on the gene. It has disadvantages in that a gene has to have been thoroughly tested across a variety of tissues. Given the number of tissues and genes this is unlikely with current technology. It also requires that the gene being tested is expressed at the appropriate stages at which the tissue was collected. For example, if the gene has pre natal effects, but adult tissues are used when it is no longer expressed, it would be concluded that it was not expressed, when in actual fact may be important. As a result rarely expressed genes may not be adequately resolved by this method.

5.10 Conclusions

All of the methods used (review known genes; OMIM; lexical analysis; tissue expression) have the potential to provide useful information, with each having advantages and disadvantages. The major limiting factor to any of these approaches is that the current gene map is still incomplete and that for many genes, relatively little is known about their function.

The overall findings from each of theses methods were:

- A review of genes known to be integrally involved with muscle/fat growth and development did not reveal any potential candidate genes,
    - Most genes did not map to the correct chromosome, and for the few that did, the location was not the location of interest.

- A search in Online Mendelian Inheritance in Man (OMIM) did not reveal any genes mapping to the correct chromosomal region and having mutations that had been shown to be associated with changes in muscle or fat.
- From the lexical analysis and tissue expression data, three candidate genes emerged:
  - GDF8, TTN and FRZB.

Based on the associated literature GDF8 is the most likely candidate. However, due to information in the literature (Marcq et al., 2002) the situation remains unclear as to whether or not GDF8 is the gene of interest. Even if further investigations do show GDF8 to be the gene, Chapter 4 provided evidence for a second QTL peak. Work to date, for the other two genes (TTN, FRZB) does not provide evidence as to whether or not mutations to these genes could result in an increased muscling phenotype.

Research is constantly being carried out and new mutations and gene functions determined, and in addition new genes which had previously not been described are characterised. Thus there remains the possibility that information from sheep or other species may become available that will provide further evidence about the involvement, of these, or for that matter any of the genes in the region, in an increased muscling phenotype.

5.11 Epilogue

Considerable time and money is currently being spent by a number of groups world wide to complete the human genome mapping project to map all genes, and to additionally provide information on their function. As a result there is new information being released in published material or via the web sites dedicated to this topic. At the time that the majority of the current work was being carried out the most recent list of genes came from the June 2002 map from the University of California Santa Cruz, Genome Browser; whilst the information on tissue expression came from the February 2003 download (based on Build 155 *Homo sapiens*). Since this time there have been further human genome maps created (November 2002, April 2003 and July 2003), and five further downloads of tissue expression (now based on Build 160, *Homo sapiens*). A comparison between the current and previously collected information reveals

differences, however, the overall conclusions remain the same.

Compared to the June 2002 human genome map, the equivalent region on the July 2003 map contained fifteen further genes. Details of these genes are presented in Appendix 8.16. Eleven of the 15 genes are "newly mapped" genes that have been newly discovered at the time of writing, with four having no abstracts relating to them and six having only one abstract relating to them. Lexical analysis carried out on those with abstracts showed that only one contained reference to any of the "search words". This gene was SP3 (Sp3 transcription factor), however, upon using the Concordance feature, references to muscle, involved smooth or cardiac muscle.

The results of the tissue expression varied considerably between the two different downloads in terms of the number of clones involved in the studies. Between Build 155 and Build 160 of the *Homo sapiens*, a new format for the UniGene's library of information was implemented, enabling the GeneCards data-mining techniques to only report gene expression in adult human tissues (as opposed to fetal/infant). As a result the total number of clones reported decreased from approximately 860,000 to 554,000, although the number for skeletal muscle actually increased from approximately 23,000 to 25,000. However, the actual expression results did not change.

Whilst the gene underlying the QTL effect remains unknown, the websites used in this study should be re-visited on a regular basis, in the hope that either a new gene will be mapped to this area that has known effects on muscling, or that a new function of a previously mapped gene will be discovered.

Chapter 6

General Discussion

A number of New Zealand meat processing companies are developing or implementing technology to provide producers information on lean-meat yield and meat quality. Such information will provide better feedback to lamb producers and stud breeders, as to the demands of the end consumers, who are seeking improved lean (muscle) and quality in the meat that they purchase. It is likely that the "ideal" lamb which receives the highest returns per kilogram, when this information is used as payment criteria for lamb, will differ to that which receives the highest value in the current market.

As a result of the proposed changes, ram breeders will need to change their breeding plans. Lamb producers, in addition to modifying their choice of rams, will need to re-consider some of their management procedures. The main objective of the work outlined in this thesis was to investigate further the potential of Quantitative Trait Loci (QTL) affecting muscling within the Texel breed, by following up on the work of Broad et al. (2000). Such a QTL would offer an alternative selection method for ram breeders. A further objective related to this was to identify candidate genes for the QTL, as in order to fully exploit any QTL, the gene or genes involved and their mode of action should ideally be known. Another objective was to investigate gender differences in carcass composition and meat quality using the population generated for the QTL search. Although comparisons between gender groups for lambs have been regularly reported in scientific literature, few differences in meat quality characteristics have been reported. There is evidence from New Zealand industry data that meat quality traits for ram lambs are inferior to ewe lambs. Such an observation over a large data set would provide important information for lamb producers. A further objective was to use reported scientific literature to assess how well the Texel may meet the requirements of the proposed payment systems based on measures of lean-meat yield and/or meat quality.

6.1 Choice of Breed

Based on the review of breed comparison studies (Section 2.3), Texel-sired lambs are likely to be rewarded for their improved dressing-out-percentage under current New Zealand payment systems based on carcass weight and GR. However, Texel-sired lambs are not specifically advantaged for their lower carcass fat, because it is not always reflected in lower GR measurements. Some studies have shown the Texel to have superior carcass conformation relative to other breeds, although this confers no

advantage to the breed using the basic grading system based on carcass weight and GR. The Alliance Group have a specific "Market Choice" grade which rewards carcass conformation, and anecdotally Texel-sired lambs achieve this grade more readily, although to ensure they meet the grade, the lambs need to be at least ¾ Texel.

With the introduction of technology that allows estimation of lean-meat-yield, the advantage conferred by Texel sires is likely to become more evident. Texel-sired lambs have consistently been shown to have increased carcass lean-meat-yield relative to other terminal-sired lambs, so they are likely to receive a price premium relative to other breeds. Additionally, Texel-sired lambs have been shown to have meat quality characteristics that are not significantly different to other terminal sire breeds, despite their increased muscling, which means they will not be disadvantaged when meat quality is introduced into grading systems.

Thus, the introduction of the Texel into New Zealand has provided breeders/producers an opportunity to increase returns in future markets without being penalised in current markets. However, as will be discussed below, there is variation in muscling within the Texel breed which also needs to be addressed.

6.2 Gender Differences

Based on the review of gender comparisons (Section 2.4), there are tendencies which hold true across the majority of studies. In comparisons between ewe and ram lambs at the same carcass weight, ram lambs had lower dressing-out percentages, smaller *M. longissimus* dimensions and poorer conformation scores. However, they also had lower fat percentages and higher lean-meat yields. There were few statistically significant differences reported for meat quality traits, with the exception of Bickerstaff et al. (2000) who found ram lambs to have significantly higher pH.

In the comparison of ram and ewe lambs (Chapter 3), the majority of the findings from the literature review were supported, however, there was one notable exception. Significant differences between the sexes were detected for several meat quality traits. Such differences have not often been reported in the scientific literature, although there is evidence from non-published industry data that differences in pH exist. A possible reason why differences have not always reported is that the numbers of animals used in

other studies have been insufficient to detect small differences. In the current study, ram lambs had higher pH, higher cooking loss (%), greater Warner-Bratzler shear force measurements (tougher) and the meat was less red, when compared with ewe lambs. Whether, these differences could be attributed to the increased muscle of the ram lambs was tested, but no consistent, significant correlations between muscle traits and quality traits were detected. This implies that there is some intrinsic characteristic of rams which results in decreased meat quality, most likely due to the effects of testosterone production.

Under current payment systems, although ewe lambs would to some extent be rewarded at a given liveweight, because of their increased dressing-out percentage, the ram lambs would have a significant advantage through having lower GR measurements. The existence of meat quality differences is of little importance to the lamb producers within the current carcass classification system in New Zealand because they are not assessed at grading.

The introduction of technology that estimates lean-meat yield will see ram lambs to have a continued advantage, as their lower GR measurements are reflective of their overall lower carcass fat and higher carcass muscle. However, if technology is introduced that assesses meat quality, it is likely that more ram than ewe lambs will fail to meet the quality criteria and their value will be discounted. It will then depend on the relative premium/discount that is applied to lean-meat yield and meat quality as to whether or not farmers will be encouraged to castrate their lambs to produce wethers. From the review of literature, wether lambs tend to be more like ewe lambs for most carcass traits. However, wethers do not have the superior lean-meat yield of ram lambs.

Although techniques to control sex ratios exist in the dairy industry, they are unlikely to be used in the sheep industry. Therefore, the farmer is left with management options – including castration and slaughtering at different ages.

6.3 Search for a QTL

Despite the Texel having an overall advantage in muscling, anecdotal evidence suggests that there is considerable variation within the breed. The data set collected in this study is currently the most comprehensive available worldwide to answer whether or not a QTL exists within the GDF8 region in the Texel breed.

This study has shown limited evidence for a QTL affecting ultrasound measurements of *M. longissimus* traits. This is in general agreement with the work of Broad et al. (2000) and Walling et al. (2002). There was significant evidence for a QTL affecting leg muscle and fat traits in four of the six sires used. Specifically the QTL resulted in an increase of 5-8% for leg muscle traits and a decrease of 10-15% for leg fat traits. The region to which the QTL mapped was similar to that described by Marcq et al. (2002), in that it was bounded by markers BM81124 and BULGE20. A novel observation in this study was that this QTL does not have an effect on meat quality traits. This is an important observation as the Callipyge increased muscling locus, is also associated with decreased tenderness (e.g. Koohmaraie et al. 1995).

If the existing payment systems were to remain in use, genetic selection of such a QTL would likely be of little benefit to the producers who used it. Although there is firm evidence that the QTL decreases leg fat, there is only limited evidence for it affecting fat measures of the loin such as GR. Thus, the cost and time associated with implementing the QTL would not be repaid through improved payment.

However, for the proposed new payment systems, the QTL is likely to result in increased returns for animals carrying the favourable form of the QTL. This is because the proposed new payment systems are likely to specifically reward increased lean-meat yield, which is the main feature of the QTL identified. Furthermore, the QTL does not appear to have a negative impact on meat quality. This is an important observation, as a number of meat processors are additionally developing technology which will assess meat quality and so although an animal may have improved carcass lean, if it was associated with decreased meat quality the carcass would be downgraded.

Very few QTL detected for meat traits in sheep and cattle have been implemented via Marker Assisted Selection (MAS) to date. A widely recognised QTL detected in cattle

has been the muscle-hypertrophy phenotype in cattle, however, this phenotype was already widespread, and did not require the use of MAS. The most obvious QTL detected in sheep has been the increased muscling phenotype associated with the Callipyge locus. Although marker tests do exist for this QTL, it has not been used in industry because of its association with poorer meat quality. The Rib-eye-muscling locus, associated with increased *M. longissimus* area, has as an associated marker test, however, there has not been industry-wide uptake of this QTL. Recently a QTL has been identified in cattle which results in increased tenderness, the gene involved has been shown to be Calpain-1, and a gene test has been developed. A screen of the New Zealand beef population is being carried out to assess the frequency of this mutation (Cullen et al., 2003), with the hope that MAS (or Gene Assisted Selection (GAS)) can be used to increase the frequency of this QTL.

Whether or not the QTL detected in this study can be used in industry via MAS is currently unclear. There is a two marker haplotype, which appears to be consistent, however, its use would likely be limited to descendents of sire 150/96. Before there is industry-wide uptake of the QTL there are a number of areas that need to be followed up in further studies. These include studies to determine:

- The effect of the QTL on other industry important traits (such as disease traits) which are an existing part of many breeding objectives. Will it complement them, or will there be antagonistic affects?
- The extent to which the effects shown here on leg composition extend to all carcass cuts, and whether or not they result in differences under yield-based payment systems.
- The effect of one versus two copies of the favourable haplotype (i.e. what is the mode of gene action).
- The prevalence of this haplotype within the Texel population and whether it is unique to the Texel population.
- Whether the likely returns, relative to the costs make the use of the information economically worthwhile, from breeder and processor perspectives.
- Locating the exact gene that is involved, which first requires further fine mapping of its location

6.4 Identification of Candidate Genes

Although the marker evidence from the QTL study (Chapter 4) would suggest that the gene involved is GDF8, work by Marcq et al. (2002) comparing the sequence of the GDF8 gene with controls has shown there to be no sequence differences. This does not exclude GDF8, as the effects reported here and elsewhere could be the result of a mutation in a promoter region which has not yet been sequenced. However, consideration needs to be given to the question of whether or not there are any other genes in the region that could cause the observed phenotype. Additionally, for one sire there appeared to be evidence of a second QTL, and therefore at least one other gene which maps to the region which affects muscle and fat.

The search for a candidate gene relies on the conserved co-linear regions of synteny that exists between the sheep/cattle and human gene maps. Using this synteny a region of human Chr2 was determined which corresponds to the region of interest on ovine Chr2. A number of approaches were taken to determine whether or not any other genes of interest map to the region. These included:-

- Creating a list of genes known to be involved with muscle and fat development, and determining their location.
- Searching On-Line Mendelian Inheritance in Man (OMIM) to identify genes where mutations are known to impact on muscle and fat development, and determining their location.
- Identification of all genes that map to the region of interest on human Chr2 and use of lexical analysis and gene expression data to determine whether or not the genes have a role which may lead to the observed phenotype.

However, no candidate genes with the exception of GDF8 were identified. This is despite over 70 genes mapping to the region of interest by using all of the above approaches. Regular revisiting of the genes mapped to this region is required, as new genes are continually being mapped and new roles of existing genes are being uncovered, thus other candidate genes will become apparent in the future.

Determining the gene involved with the increased muscling phenotype described in Chapter 4 is important if full exploitation of the potential of the gene is to be made. The

most obvious application, would be to screen the NZ sheep population for other functional variants. There would also be the opportunity to develop vaccines that could promote muscle growth, an example of which was the immunization of mice against GDF8 which led to increased muscle development (Whittemore et al., 2003).

6.5 Overall

The payment system used by meat processors to pay producers, is likely to change in the near future. This thesis identifies a number of opportunities for producers and breeders to make improved returns:

- Choice of breed is an obvious way to manipulate muscle characteristics. This thesis has shown that the Texel, of the terminal sire breeds available in New Zealand, provides producers with a particularly good option for increasing lean-meat yield without adversely affecting meat quality.
- Although gender is not easy to manipulate, this thesis has shown that ram lambs are likely to better rewarded relative to ewes lambs. However, if quality is also integrated into the payment systems, and the quality differences prove to be commercially important, an option for producers would be to castrate their ram lambs.
- The use of new technologies to select directly on the genetic makeup of the animal, offers the most potential of any of the options. This thesis has shown that a QTL affecting muscling, without affecting meat quality exists within the Texel breed. Whether or not this QTL is suitable for use with MAS in the industry, is currently unclear and requires further investigation.
- Optimum use of this QTL would come from identifying the gene involved, at this point in time GDF8 would appear to be the best candidate.

Chapter 7

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Chapter 8

Appendices

8.1 DNA Procedures

8.1.1 DNA Extraction Technique

DNA was extracted from FTA paper.

- 2.5mm^2 samples of blood soaked DNA labels were punched into sterile 1.5ml microcentrifuge tubes.
- 200µl of distilled water was added, after two hours at room temperature, the water was pipette off and the punch dried at room temperature.
- 20 µl of 0.2M NaOH (0.8g NaOH + 100ml distilled water) was added to the punch, once soaked it was incubated in the tube at 75°C for five minutes.
- After incubation 80µl 45mM Tris-HCl, pH 7.6 was added to stop the extraction
    - *45mM Tris-HCl, pH 7.6* - 450µl 1.0M Tris-HCl, pH 7.6 + 9.55ml water; *1.0M Tris-HCl, pH 7.6* – 12.11g Tris dissolved in 80ml water, pH 7.6 using HCL and top up to 100ml
- 10µl 0.2% Azide was added to the solution to prevent contamination.

8.1.2 DNA Amplification Reagents

The PCR reaction required a radioactive label, cocktail and DNA.

- Radioactive Label:
    - 98µl of distilled water; 10µl of primer B; 9µl of buffer; 9µl of T4kinase; 9µl of P33; Total volume 135µl.
    - Labels were incubated for one hour in a 37°C water-bath, and then stored in freezer until required. When required they were allowed to warm to room temperature before use.
- Cocktail for 384 well plates:
    - 3300µl of distilled water; 76µl of primer A; 315µl of DNTP's; 315µl of Tricine (tween); 15µl of BSA; 15µl of red hot TAQ; 135µl of the label prepared above is added just prior to plating.
- 10µl of cocktail and 1.5µl of DNA followed by 10µl of paraffin oil were added per wells in 384 well plates, a wax lid was placed over the plate to prevent evaporation.
- The PCR reaction took place in a Techne Thermal Cycler running a 32 cycle touchdown program made up of the following cycles which took approximately two hours to run: 3 x 95°C, 45 sec then 60°C, 1 min; 3 x 95°C, 45 sec then 57°C, 1 min;

3 x 95°C, 45 sec then 54°C, 1min; 3 x 95°C, 45 sec then 51°C, 1min; 20x 95°C, 45 sec then 48°C, 1 min.

8.1.3 Gel used for Electrophoresis

Gels were run on homemade plates, with 48 lane combs used.

- Gel:
  - 50ml acrylamide gel; 290µl ammonium persulphate and 36µl Temed.
  - Poured into the plate ensuring that no air locks formed and allowed to set.
- The plates were attached to plastic backing and placed in bases connected to an electrophoresis power pack. 1:10 dilution of x10 TBE buffer was added to the base and back of the plates.
- 10µl of azide was added to each well of amplified DNA, which was then heated at 95°C for four minutes prior to loading the gels.
- Four microliters of amplified DNA was added to the lanes on the gels using a channel Hamilton syringe.

8.1.4 Alternative Protocol for Marker TGLA10

- New primers were used
- Radioactive Label:
  - 60µl of distilled water; 10µl of primer B; 10µl of buffer; 16µl of T4kinase; 20µl of P33; Total volume 116µl.
  - Labels were incubated for one hour in a 37°C water-bath, and then stored in freezer until required. When required they were allowed to warm to room temperature before use.
- Cocktail for 384 well plates:
  - 2200µl of distilled water; 178µl of primer; 320µl of DNTP's; 320µl of Tricine (tween); 24µl of BSA; 18µl of red hot TAQ; 116µl of the label prepared above is added just prior to plating the cocktail.
- 10µl of cocktail and 1.5µl of DNA followed by 10µl of paraffin oil were added per wells in 384 well plates, a wax lid was placed over the plate to prevent evaporation.
- The PCR reaction was the same as described in 8.1.2

8.2 Models Fitted for QTL Analysis

8.2.1 Definition of Fixed Effects and Covariates

Fitted	**Definition**
Covariates	
Birthday deviation	Deviation from overall average birth date
Scan weight	Liveweight at scanning
Carcass weight	Cold carcass weight, taken after chilling
Fixed Effects	
Sex	Sex of lamb: ram or ewe
Birth/Rearing Rank	Born Single/Reared Single (11); Born Twin/ Reared Single (21); Born Twin/Triplet Reared Twin (22)
Slaughter Group	One of four groups in which the lambs were slaughtered, as defined in Table 4.2
Dissector	One of five people who carried out the dissection of the leg and loin
Sire Number	One of seven sire number, as defined in Table 4.2

8.2.2 Models Fitted

Table 8.1 Covariates and fixed effects fitted for different trait groups. Definitions of the trait groups are provided in Section 4.2.16. Sire number was fitted in all model

Group	**Covariate**	**Fixed Effects**
Liveweight traits	Birthday deviation	Birth/rearing rank Sex Sire number
Ultrasound traits	Scan weight	Sex Sire number
Carcass linear traits	Carcass weight	Sex Slaughter group Sire number
Leg muscle traits	Carcass weight	Sex Slaughter group Dissector Sire number
Leg fat traits	Carcass weight	Sex Slaughter group Dissector Sire number
Leg bone traits	Carcass weight	Sex Slaughter group Dissector Sire number
Loin muscle traits	Carcass weight	Sex Slaughter group Sire number
Loin fat traits	Carcass weight	Sex Slaughter group Sire number
Ratio traits	-	Sex Slaughter group Dissector Sire number
Meat quality traits	-	Sex Slaughter group Sire number

8.3 Pedigree Diagrams

Figure 8.1 Pedigree diagram for Sire 535/98. Dam lines are represented in red and sire lines in blue

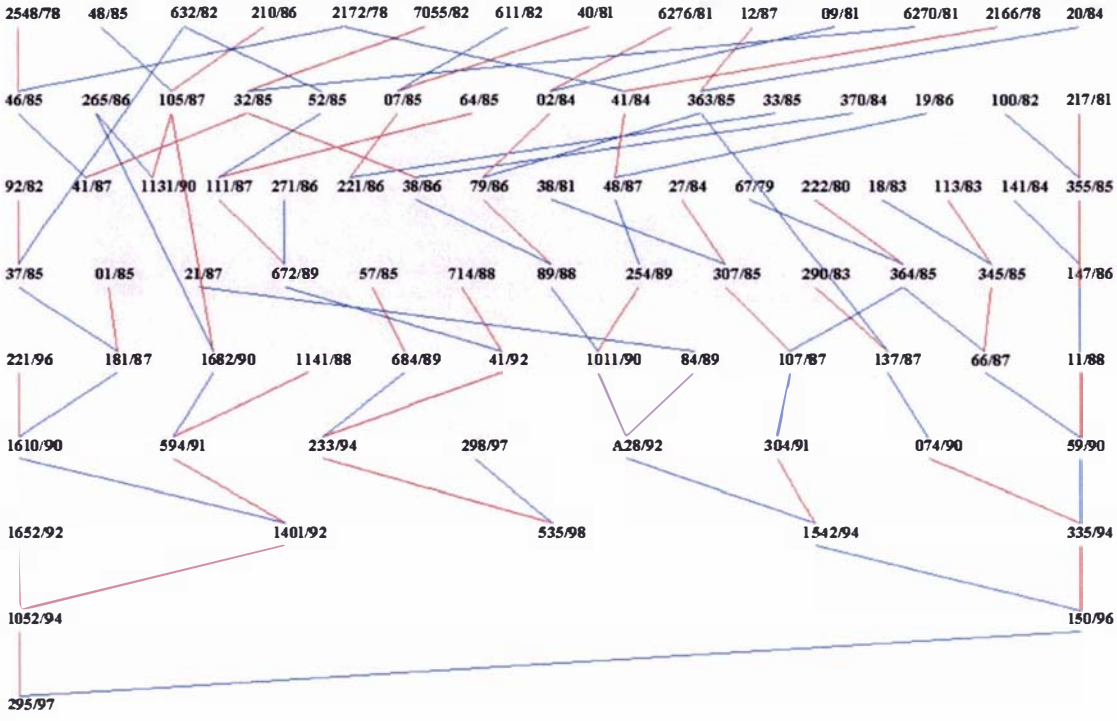


Figure 8.2 Pedigree diagram for Sire 15/98. Dam lines are represented in red and sire lines in blue

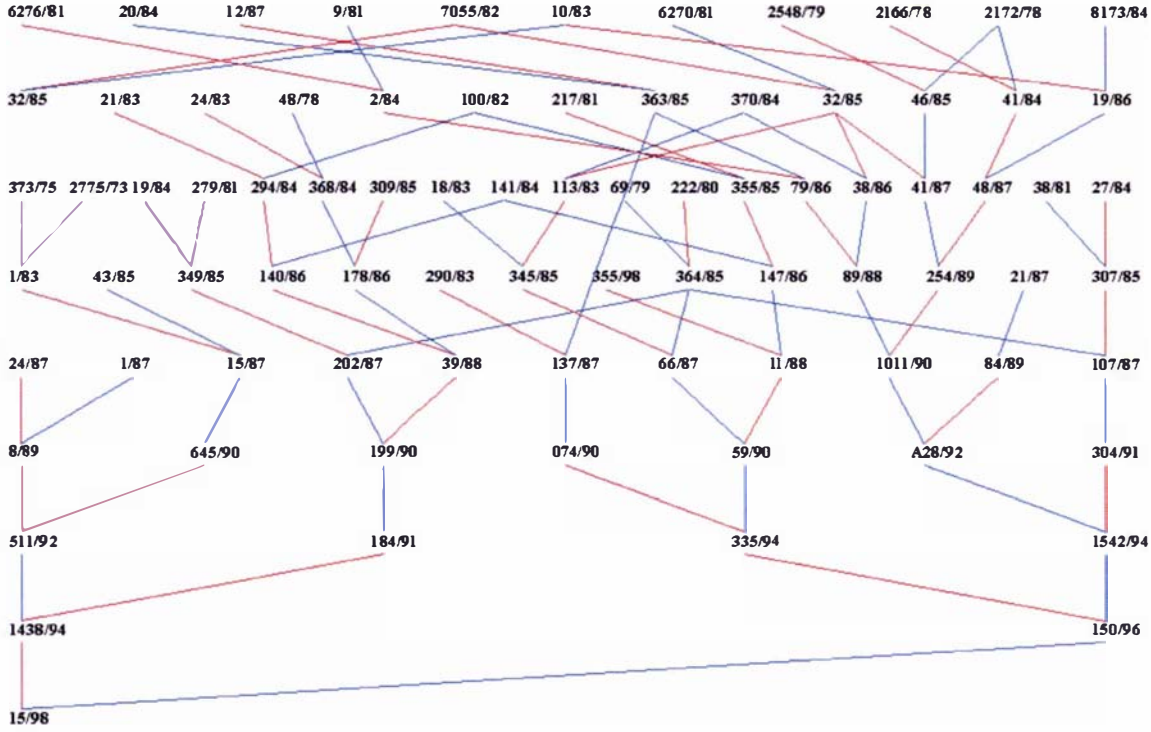
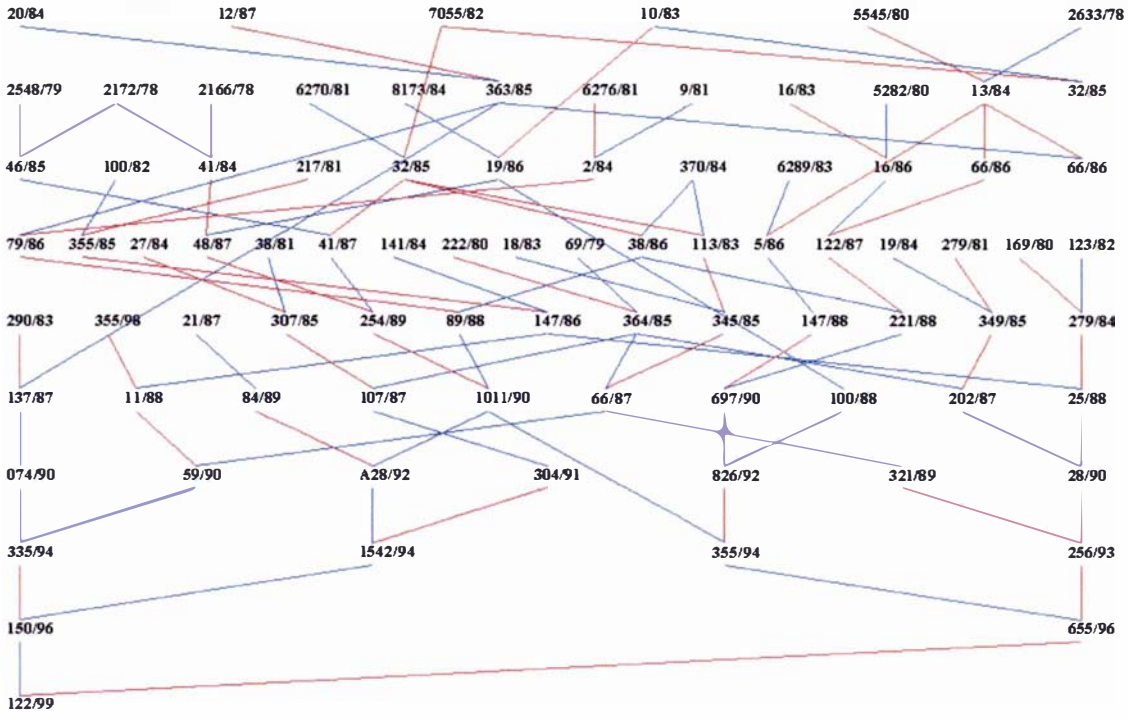


Figure 8.3 Pedigree diagram for Sire 122/99. Dam lines are represented in red and sire lines in blue



8.4 Sire 1190/00 Non-Significant Single Trait QTL Results

Table 8.2 Details of peaks for sire 1199/00 Texel cross half-sib QTL analysis for a region of ovine chromosome two – liveweight traits[1]

Trait	Phen mean ± adj. std	Estimate ± stderr	SD units	Permutation 95%	Permutation 99%	max -log$_{10}$ prob	pos (cM) [CI]
Birth weight (kg)	5.3 ± 0.8	-0.2 ± 0.2	0.2	1.9	2.7	0.5	66
Wean weight (kg)	24.0 ± 2.8	0.8 ± 0.8	0.3	1.9	2.6	0.4	20
Scan weight (kg)	34.0 ± 4.1	1.2 ± 0.9	0.3	2.1	2.9	0.8	62
Daily gain birth-wean (g)	0.3 ± 0.04	0.0 ± 0.0	0.3	1.8	2.5	0.5	20
Daily gain wean-scan (g)	0.1 ± 0.04	0.0 ± 0.0	0.4	2.0	2.7	1.0	76

[1] Descriptions of column headings are given in Table 4.16

Table 8.3 Details of peaks for sire 1199/00 Texel cross half-sib QTL analysis for a region of ovine chromosome two – ultrasound muscle traits[1]

Trait	Phen mean ± adj. std	Estimate ± stderr	SD units	Permutation 95%	Permutation 99%	max -log$_{10}$ prob	pos (cM) [CI]
M. longissimus width (mm)	58.5 ± 3.9	1.1 ± 0.8	0.3	1.9	2.5	0.8	112
M. longissimus depth (mm)	21.4 ± 2.0	0.6 ± 0.4	0.3	2.0	2.8	1.0	104
M. longissimus area (mm^2)	971.3 ± 147.0	46.6 ± 27.6	0.3	2.0	2.7	1.0	104

[1] Descriptions of column headings are given in Table 4.16

Table 8.4 Details of peaks for sire 1199/00 Texel cross half-sib QTL analysis for a region of ovine chromosome two – ultrasound fat traits[1]

Trait	Phen mean ± adj. std	Estimate ± stderr	SD units	Permutation 95%	Permutation 99%	max -log$_{10}$ prob	pos (cM) [CI]
Fat depth C (mm)	1.6 ± 0.5	-0.3 ± 0.1	0.7	1.9	2.6	2.1 *	20 [2 -113]

[1] Descriptions of column headings are given in Table 4.16

Table 8.5 Details of peaks for sire 1199/00 Texel cross half-sib QTL analysis for a region of ovine chromosome two – dressing-out percentage[1]

Trait	Phen mean ± adj. std	Estimate ± stderr	SD units	Permutation 95%	Permutation 99%	max -log$_{10}$ prob	pos (cM) [CI]
Dressing-out percentage	38.5 ± 1.7	0.3 ± 0.2	0.2	1.8	2.5	0.9	104

[1] Descriptions of column headings are given in Table 4.16

Table 8.6 Details of peaks for sire 1199/00 Texel cross half-sib QTL analysis for a region of ovine chromosome two – carcass linear traits[1]

Trait	Phen mean ± adj. std	Estimate ± stderr	SD units	Permutation 95%	Permutation 99%	max -log$_{10}$ prob	pos (cM) [CI]
Carcass length (cm)	79.8 ± 2.1	-0.3 ± 0.4	0.1	2.3	3.2	0.3	104
Width of forequarter (cm)	16.6 ± 0.8	-0.3 ± 0.2	0.4	2.4	3.3	1.4	2
Width of thorax (cm)	16.0 ± 0.7	-0.2 ± 0.1	0.3	1.8	2.4	0.8	2
Gigot width (cm)	21.7 ± 0.7	0.3 ± 0.1	0.4	2.0	2.6	1.7	66

[1] Descriptions of column headings are given in Table 4.16

Table 8.7 Details of peaks for sire 1199/00 Texel cross half-sib QTL analysis for a region of ovine chromosome two – loin muscle traits[1]

Trait	Phen mean ± adj. std	Estimate ± stderr	SD units	Permutation 95%	99%	max $-\log_{10}$ prob	pos (cM) [CI]
M. longissimus width (mm)	55.3 ± 3.5	1.7 ± 0.7	0.5	2.4	3.3	2.0	52
M. longissimus depth (mm)	25.8 ± 2.2	0.9 ± 0.5	0.4	2.1	2.8	1.2	56
M. longissimus area (mm^2)	1,101.3 ± 127.5	71.1 ± 27.0	0.6	2.2	3.0	2.0	54
M. longissimus weight (g)	193.2 ± 18.4	9.7 ± 3.4	0.5	2.1	2.8	2.3 *	52 [2 - 122]

[1] Descriptions of column headings are given in Table 4.16

Table 8.8 Details of peaks for sire 1199/00 Texel cross half-sib QTL analysis for a region of ovine chromosome two - leg bone traits[1]

Trait	Phen mean ● adj. std	Estimate ● stderr	SD units	Permutation 95%	99%	max $-\log_{10}$ prob	pos (cM) [CI]
Pelvic bone weight (g)	107.8 ± 8.6	3.1 ± 2.0	0.4	2.1	2.8	0.9	122
Femur bone weight (g)	154.4 ± 11.7	-2.7 ± 2.8	0.2	2.4	3.2	0.5	2
Total bone weight (g)	420.1 ± 32.0	7.3 ± 7.0	0.2	2.1	2.9	0.5	122
Femur bone length (mm)	172.8 ± 4.0	-1.5 ± 0.9	0.4	2.0	2.8	1.0	58

[1] Descriptions of column headings are given in Table 4.16

Table 8.9 Details of peaks for sire 1199/00 Texel cross half-sib QTL analysis for a region of ovine chromosome two – meat quality traits[1]

Trait	Phen mean ± adj. std	Estimate ± stderr	SD units	Permutation 95%	99%	max $-\log_{10}$ prob	pos (cM) [CI]
M. semimembranosus							
pH	5.6 ± 0.1	0.0 ± 0.0	0.3	1.7	2.2	0.8	116
Colour "L*"	29.8 ± 1.9	1.0 ± 0.7	0.6	1.8	2.4	1.0	18
Colour "a*"	14.9 ± 1.2	0.4 ± 0.3	0.3	1.9	2.6	0.7	114
Colour "b*"	6.3 ± 0.8	0.3 ± 0.2	0.4	1.9	2.6	0.9	104
Sarcomere lng. (μm)	1.7 ± 0.4	0.1 ± 0.1	0.3	2.3	3.1	0.6	122
Cooking loss (%)	37.2 ± 1.2	0.3 ± 0.3	0.3	2.1	2.8	0.5	2
W-B peak force	11.3 ± 3.0	-0.8 ± 0.7	0.3	2.0	2.6	0.6	122
W-B yield force	8.6 ± 2.6	-0.8 ± 0.6	0.3	2.0	2.8	0.8	122
M. longissimus							
pH	5.7 ± 0.2	-0.1 ± 0.1	0.3	2.0	2.6	0.8	116
Colour "L*"	31.3 ± 2.2	0.8 ± 0.6	0.3	1.8	2.5	0.6	0
Colour "a*"	14.7 ± 1.5	0.9 ± 0.4	0.6	2.0	2.7	1.7	122
Colour "b*"	6.2 ± 1.1	0.5 ± 0.2	0.5	2.5	3.5	1.7	122
Sarcomere lng. (μm)	1.8 ± 0.1	0.0 ± 0.0	0.5	1.0	1.3	0.5	110
Cooking loss (%)	30.0 ± 2.1	1.2 ± 0.7	0.6	1.8	2.5	1.0	24
W-B peak force	7.1 ± 1.9	1.0 ± 0.4	0.5	2.3	3.2	2.0	56
W-B yield force	5.6 ± 1.2	0.6 ± 0.3	0.5	2.3	3.2	1.7	56

[1] Descriptions of column headings are given in Table 4.16

8.5 Sire 1170/00 Non-Significant Single Trait QTL Results

Table 8.10 Details of peaks for sire 1170/00 Texel cross half-sib QTL analysis for a region of ovine chromosome two – liveweight traits[1]

Trait	Phen mean ● adj. std	Estimate ± stderr	SD units	Permutation 95%	Permutation 99%	max $-\log_{10}$ prob	pos (cM) [CI]
Birth weight (kg)	5.3 ± 0.8	-0.1 ± 0.2	0.2	2.0	2.8	0.3	88
Wean weight (kg)	22.8 ± 2.7	0.8 ± 0.6	0.3	1.8	2.4	0.8	66
Scan weight (kg)	32.8 ± 3.8	1.5 ± 0.9	0.4	1.6	2.3	1.0	66
Daily gain birth-wean (g)	0.3 ± 0.04	0.0 ± 0.0	0.4	1.7	2.4	1.0	66
Daily gain wean-scan (g)	0.1 ± 0.03	0.0 ± 0.0	0.4	1.4	1.9	0.9	0

[1] Descriptions of column headings are given in Table 4.16

Table 8.11 Details of peaks for sire 1170/00 Texel cross half-sib QTL analysis for a region of ovine chromosome two – ultrasound fat traits[1]

Trait	Phen mean ● adj. std	Estimate ± stderr	SD units	Permutation 95%	Permutation 99%	max $-\log_{10}$ prob	pos (cM) [CI]
Fat depth C (mm)	1.5 ± 0.5	0.1 ± 0.1	0.3	1.9	2.6	0.7	122

[1] Descriptions of column headings are given in Table 4.16

Table 8.12 Details of peaks for sire 1170/00 Texel cross half-sib QTL analysis for a region of ovine chromosome two – dressing-out percentage[1]

Trait	Phen mean ● adj. std	Estimate ± stderr	SD units	Permutation 95%	Permutation 99%	max $-\log_{10}$ prob	pos (cM) [CI]
Dressing-out percentage	39.1 ± 1.9	0.4 ● 0.2	0.2	2.3	3.1	1.2	60

[1] Descriptions of column headings are given in Table 4.16

Table 8.13 Details of peaks for sire 1170/00 Texel cross half-sib QTL analysis for a region of ovine chromosome two – carcass linear traits[1]

Trait	Phen mean ● adj. std	Estimate ± stderr	SD units	Permutation 95%	Permutation 99%	max $-\log_{10}$ prob	pos (cM) [CI]
Carcass length (cm)	81.1 ± 2.0	0.6 ± 0.5	0.3	1.9	2.7	0.7	24
Width of forequarter (cm)	16.4 ± 0.6	-0.2 ± 0.1	0.3	1.4	2.0	0.7	58
Width of thorax (cm)	15.8 ± 0.7	-0.3 ± 0.1	0.4	1.9	2.7	1.4	56
Gigot width (cm)	21.4 ± 0.7	0.3 ± 0.1	0.4	2.2	3.1	2.0	82

[1] Descriptions of column headings are given in Table 4.16

Table 8.14 Details of peaks for sire 1170/00 Texel cross half-sib QTL analysis for a region of ovine chromosome two – loin muscle traits[1]

Trait	Phen mean ± adj. std	Estimate ± stderr	SD units	Permutation 95%	Permutation 99%	max $-\log_{10}$ prob	pos (cM) [CI]
M. longissimus width (mm)	53.9 ± 2.8	1.8 ± 0.8	0.6	1.3	1.8	1.5 *	38 [15 - 122]
M. longissimus depth (mm)	24.7 ± 2.2	0.7 ● 0.5	0.3	1.8	2.5	0.8	122
M. longissimus area (mm^2)	1,026.7 ± 117.8	39.2 ± 35.2	0.3	1.5	2.2	0.6	26
M. longissimus weight (g)	175.8 ± 16.5	-5.2 ± 3.7	0.3	1.4	1.8	0.8	82

[1] Descriptions of column headings are given in Table 4.16

Table 8.15 Details of peaks for sire 1170/00 Texel cross half-sib QTL analysis for a region of ovine chromosome two – loin fat traits[1]

Trait	Phen mean ● adj. std	Estimate ± stderr	SD units	Permutation 95%	Permutation 99%	max $-\log_{10}$ prob	pos (cM) [CI]
GR (mm)	5.2 ± 1.9	-0.9 ± 0.5	0.5	1.9	2.6	1.3	34
Fat depth C (mm)	2.7 ± 0.9	-0.4 ± 0.2	0.4	1.6	2.2	1.2	54
Loin fat weight (g)	79.8 ± 20.0	-9.3 ± 5.8	0.5	1.0	1.4	1.0	52

[1] Descriptions of column headings are given in Table 4.16

Table 8.16 Details of peaks for sire 1170/00 Texel cross half-sib QTL analysis for a region of ovine chromosome two - leg bone traits[1]

Trait	Phen mean ± adj. std	Estimate ± stderr	SD units	Permutation 95%	Permutation 99%	max $-\log_{10}$ prob	pos (cM) [CI]
Pelvic bone weight (g)	107.0 ± 9.6	4.6 ± 2.1	0.5	1.7	2.4	1.5	118
Femur bone weight (g)	147.3 ± 11.1	3.4 ± 2.4	0.3	1.7	2.4	0.8	58
Total bone weight (g)	405.1 ± 30.0	15.0 ± 7.6	0.5	1.8	2.5	1.3	110
Femur bone length (mm)	172.0 ± 4.1	-1.1 ± 1.0	0.3	1.9	2.7	0.6	60

[1] Descriptions of column headings are given in Table 4.16

Table 8.17 Details of peaks for sire 1170/00 Texel cross half-sib QTL analysis for a region of ovine chromosome two – meat quality traits[1]

Trait	Phen mean ● adj. std	Estimate ± stderr	SD units	Permutation 95%	Permutation 99%	max $-\log_{10}$ prob	pos (cM) [CI]
M. semimembranosus							
pH	5.6 ± 0.1	0.0 ± 0.0	0.4	1.8	2.5	0.9	92
Colour "L*"	30.7 ± 2.2	0.7 ± 0.5	0.3	2.1	3.0	1.0	0
Colour "a*"	15.1 ± 1.2	0.4 ± 0.3	0.4	1.7	2.3	0.9	122
Colour "b*"	6.6 ± 0.7	0.1 ± 0.2	0.2	1.7	2.5	0.4	2
Sarcomere lng. (μm)	1.8 ± 0.3	-0.1 ± 0.1	0.4	1.1	1.5	0.7	66
Cooking loss (%)	36.9 ± 1.1	0.5 ± 0.3	0.5	1.6	2.2	1.2	58
W-B peak force	10.0 ± 2.6	0.5 ± 0.7	0.2	1.6	2.2	0.4	66
W-B yield force	7.6 ± 2.2	0.7 ± 0.6	0.3	1.6	2.2	0.6	68
M. longissimus							
pH	5.7 ± 0.2	0.1 ± 0.1	0.3	1.4	2.0	0.6	90
Colour "L*"	32.3 ± 2.4	0.6 ± 0.5	0.3	1.9	2.7	0.6	66
Colour "a*"	14.9 ± 1.4	0.7 ± 0.4	0.5	1.8	2.6	1.2	122
Colour "b*"	6.7 ± 0.8	-0.5 ± 0.3	0.6	1.3	1.8	1.0	30
Sarcomere lng. (μm)	1.8 ± 0.2	0.1 ± 0.0	0.3	2.0	2.5	1.0	58
Cooking loss (%)	29.7 ± 2.1	-0.7 ± 0.6	0.4	1.6	2.3	0.7	88
W-B peak force	6.1 ± 1.3	-0.7 ± 0.5	0.6	1.1	1.5	0.8	18
W-B yield force	5.0 ± 0.9	-0.7 ± 0.3	0.8	1.3	1.8	1.5 *	14 [2 - 122]

[1] Descriptions of column headings are given in Table 4.16

8.6 Sire 150/96 Non-Significant Single Trait QTL Results

Table 8.18 Details of peaks for sire 150/96 Texel cross half-sib QTL analysis for a region of ovine chromosome two – liveweight traits[1]

Trait	Phen mean ± adj. std	Estimate ± stderr	SD units	Permutation 95%	Permutation 99%	max -log$_{10}$ prob	pos (cM) [CI]
Birth weight (kg)	5.4 ± 0.7	0.2 ± 0.2	0.3	1.9	2.6	1.0	66
Wean weight (kg)	23.8 ± 3.4	0.4 ± 0.6	0.1	2.3	3.0	0.3	66
Scan weight (kg)	34.0 ± 4.1	1.4 ± 0.8	0.3	2.2	2.9	1.1	66
Daily gain birth-wean (g)	0.3 ± 0.05	0.0 ± 0.0	0.2	2.4	3.2	0.4	122
Daily gain wean-scan (g)	0.1 ± 0.04	0.0 ± 0.0	0.2	2.5	3.3	0.5	66

[1] Descriptions of column headings are given in Table 4.16

Table 8.19 Details of peaks for sire 150/96 Texel cross half-sib QTL analysis for a region of ovine chromosome two – ultrasound muscle traits[1]

Trait	Phen mean ± adj. std	Estimate ± stderr	SD units	Permutation 95%	Permutation 99%	max -log$_{10}$ prob	pos (cM) [CI]
M. longissimus width (mm)	59.7 ± 4.0	1.1 ± 0.7	0.3	2.0	2.6	0.9	122
M. longissimus depth (mm)	22.6 ± 2.0	0.3 ± 0.3	0.2	1.9	2.6	0.5	66
M. longissimus area (mm^2)	1,044.3 ± 148.1	31.3 ± 25.5	0.2	2.0	2.7	0.7	122

[1] Descriptions of column headings are given in Table 4.16

Table 8.20 Details of peaks for sire 150/96 Texel cross half-sib QTL analysis for a region of ovine chromosome two – ultrasound fat traits[1]

Trait	Phen mean ± adj. std	Estimate ± stderr	SD units	Permutation 95%	Permutation 99%	max -log$_{10}$ prob	pos (cM) [CI]
Fat depth C (mm)	1.8 ± 0.4	0.1 ● 0.1	0.2	2.0	2.6	0.5	106

[1] Descriptions of column headings are given in Table 4.16

Table 8.21 Details of peaks for sire 150/96 Texel cross half-sib QTL analysis for a region of ovine chromosome two – dressing-out percentage[1]

Trait	Phen mean ± adj. std	Estimate ± stderr	SD units	Permutation 95%	Permutation 99%	max -log$_{10}$ prob	pos (cM) [CI]
Dressing-out percentage	40.3 ± 1.9	-0.1 ± 0.2	0.1	1.9	2.7	0.4	122

[1] Descriptions of column headings are given in Table 4.16

Table 8.22 Details of peaks for sire 150/96 Texel cross half-sib QTL analysis for a region of ovine chromosome two – carcass linear traits[1]

Trait	Phen mean ± adj. std	Estimate ± stderr	SD units	Permutation 95%	Permutation 99%	max -log$_{10}$ prob	pos (cM) [CI]
Carcass length (cm)	80.4 ± 2.0	-0.3 ± 0.4	0.2	1.7	2.3	0.4	104
Width of forequarter (cm)	17.4 ± 0.8	-0.2 ± 0.1	0.3	2.0	2.8	1.2	122
Width of thorax (cm)	16.5 ± 0.7	-0.3 ± 0.1	0.4	2.1	2.9	1.5	104
Cigot width (cm)	22.2 ± 0.6	-0.1 ± 0.1	0.2	1.7	2.2	0.4	122

[1] Descriptions of column headings are given in Table 4.16

Table 8.23 Details of peaks for sire 150/96 Texel cross half-sib QTL analysis for a region of ovine chromosome two – leg muscle weight traits[1]

Trait	Phen mean ± adj. std	Estimate ± stderr	SD units	Permutation 95%	99%	max $-\log_{10}$ prob	pos (cM) [CI]
Trimmed leg (g)	2,726.3 ± 141.8	36.7 ± 19.2	0.3	2.1	2.7	1.2	104
M. semimembranosus (g)	252.2 ± 17.5	3.3 ± 3.8	0.2	1.8	2.3	0.4	116
M. semitendinosus (g)	95.8 ± 9.7	-2.8 ± 1.8	0.3	2.2	2.9	0.9	122
M. biceps femoris (g)	256.7 ± 19.7	3.9 ± 3.9	0.2	2.0	2.7	0.5	104
M. quadriceps (g)	369.9 ± 28.1	6.8 ± 5.0	0.2	2.4	3.3	0.7	108
M. adductor (g)	120.9 ± 9.8	6.6 ± 2.3	0.7	2.2	3.0	2.4 *	96 [39 - 122]
M. gluteus medius (g)	166.7 ± 16.6	-2.6 ± 3.0	0.2	2.2	2.9	0.4	66
Muscle trim (g)	683.6 ± 46.7	8.6 ± 9.2	0.2	2.0	2.7	0.5	104
Total leg muscle (g)	1,945.8 ± 119.7	28.2 ± 20.6	0.2	1.9	2.6	0.8	104

[1] Descriptions of column headings are given in Table 4.16

Table 8.24 Details of peaks for sire 150/96 Texel cross half-sib QTL analysis for a region of ovine chromosome two – other muscle related traits[1]

Trait	Phen mean ± adj. std	Estimate ± stderr	SD units	Permutation 95%	99%	max $-\log_{10}$ prob	pos (cM) [CI]
Leg muscularity	0.5 ± 0.02	0.01 ± 0.005	0.6	1.7	2.3	1.3	88
Leg muscle percent	71.4 ± 1.7	0.3 ± 0.4	0.2	1.7	2.2	0.3	54
Femur muscle to bone	4.7 ± 0.3	0.1 ± 0.1	0.4	1.8	2.4	0.8	54

[1] Descriptions of column headings are given in Table 4.16

Table 8.25 Details of peaks for sire 150/96 Texel cross half-sib QTL analysis for a region of ovine chromosome two – leg fat weight traits[1]

Trait	Phen mean ± adj. std	Estimate ± stderr	SD units	Permutation 95%	99%	max $-\log_{10}$ prob	pos (cM) [CI]
Subcutaneous fat (g)	204.4 ± 34.4	6.9 ± 8.2	0.2	2.1	2.8	0.4	104
Intermuscular fat (g)	141.7 ± 23.6	6.3 ± 6.0	0.3	2.1	2.9	0.5	114
Total leg fat (g)	346.1 ± 45.2	12.5 ± 10.8	0.3	2.0	2.8	0.6	104

[1] Descriptions of column headings are given in Table 4.16

Table 8.26 Details of peaks for sire 150/96 Texel cross half-sib QTL analysis for a region of ovine chromosome two – loin muscle traits[1]

Trait	Phen mean ± adj. std	Estimate ± stderr	SD units	Permutation 95%	99%	max $-\log_{10}$ prob	pos (cM) [CI]
M. longissimus width (mm)	57.1 ± 3.3	-1.2 ± 0.8	0.4	2.2	2.9	0.8	92
M. longissimus depth (mm)	26.7 ± 2.2	0.3 ± 0.5	0.1	2.0	2.8	0.3	56
M. longissimus area (mm^2)	1,177.2 ± 130.9	-18.4 ± 28.2	0.1	2.2	3.0	0.3	72
Loin muscle weight (g)	202.7 ± 19.7	3.2 ± 3.8	0.2	2.5	3.4	0.4	104

[1] Descriptions of column headings are given in Table 4.16

Table 8.27 Details of peaks for sire 150/96 Texel cross half-sib QTL analysis for a region of ovine chromosome two – loin fat traits[1]

Trait	Phen mean ± adj. std	Estimate ± stderr	SD units	Permutation 95%	99%	max $-\log_{10}$ prob	pos (cM) [CI]
GR (mm)	5.1 ± 1.7	0.7 ± 1.0	0.4	1.9	2.5	0.3	0
Fat depth C (mm)	2.2 ± 1.0	0.2 ± 0.2	0.2	2.2	2.9	0.3	50
Loin fat (g)	78.4 ± 34.9	-12.3 ± 5.7	0.4	2.5	3.1	1.5	66
Leg fat percent	12.7 ± 1.8	0.3 ± 0.5	0.3	1.4	2.0	1.1	68

[1] Descriptions of column headings are given in Table 4.16

Table 8.28 Details of peaks for sire 150/96 Texel cross half-sib QTL analysis for a region of ovine chromosome two - leg bone traits[1]

Trait	Phen mean ± adj. std	Estimate ● stderr	SD units	Permutation 95%	Permutation 99%	max $-\log_{10}$ prob	pos (cM) [CI]
Pelvic bone weight (g)	110.9 ± 8.9	-4.7 ± 3.6	0.5	2.1	2.8	0.7	24
Femur bone weight (g)	152.3 ± 10.4	-2.4 ± 5.9	0.2	1.8	2.4	0.2	10
Total bone weight (g)	418.8 ± 30.3	-14.8 ± 11.6	0.5	1.9	2.7	0.7	28
Femur bone length (mm)	171.1 ± 4.0	-1.4 ± 0.9	0.4	1.9	2.5	1.0	62

[1] Descriptions of column headings are given in Table 4.16

Table 8.29 Details of peaks for sire 150/96 Texel cross half-sib QTL analysis for a region of ovine chromosome two – meat quality traits[1]

Trait	Phen mean ± adj. std	Estimate ± stderr	SD units	Permutation 95%	Permutation 99%	max $-\log_{10}$ prob	pos (cM) [CI]
M. semimembranosus							
pH	5.7 ± 0.1	0.0 ± 0.0	0.4	2.1	2.8	1.0	116
Colour "L*"	30.6 ± 1.8	0.3 ± 0.5	0.2	1.8	2.4	0.3	60
Colour "a*"	15.0 ± 1.4	0.5 ± 0.3	0.4	2.2	3.0	1.1	104
Colour "b*"	6.4 ± 0.8	0.4 ± 0.2	0.4	2.2	2.9	1.2	106
Sarcomere lng. (μm)	1.7 ± 0.4	0.2 ± 0.1	0.4	2.5	3.3	0.9	88
Cooking loss (%)	37.3 ± 1.2	-0.4 ± 0.3	0.3	2.2	3.0	0.7	122
W-B peak force	10.3 ± 3.1	-0.8 ± 0.8	0.3	2.3	3.1	0.5	90
W-B yield force	7.7 ± 2.6	-0.6 ± 0.6	0.2	2.2	2.9	0.5	48
M. longissimus							
pH	5.7 ± 0.2	0.0 ± 0.0	0.1	2.3	3.0	0.3	122
Colour "L*"	32.0 ± 2.3	-0.7 ± 0.6	0.3	2.0	2.7	0.7	104
Colour "a*"	14.6 ± 1.5	-0.3 ± 0.3	0.2	2.0	2.7	0.5	56
Colour "b*"	6.2 ± 1.0	-0.2 ± 0.2	0.2	2.1	2.8	0.5	56
Sarcomere lng. (μm)	1.8 ± 0.1	0.0 ± 0.0	0.4	1.1	1.5	0.5	122
Cooking loss (%)	30.0 ± 2.3	-1.2 ± 1.4	0.5	2.2	2.9	0.4	2
W-B peak force	6.5 ± 1.9	-1.0 ± 0.4	0.6	2.3	3.1	2.0	52
W-B yield force	5.0 ± 1.2	-1.4 ± 0.6	1.2	2.3	3.1	2.0	14

[1] Descriptions of column headings are given in Table 4.16

8.7 Sire 15/98 Non-Significant Single Trait QTL Results

Table 8.30 Details of peaks for sire 15/98 Texel cross half-sib QTL analysis for a region of ovine chromosome two – liveweight traits[1]

Trait	Phen mean ± adj. std	Estimate ● stderr	SD units	Permutation 95%	Permutation 99%	max $-\log_{10}$ prob	pos (cM) [CI]
Birth weight (kg)	4.8 ± 0.8	-0.4 ± 0.2	0.5	1.9	2.6	1.3	122
Wean weight (kg)	23.5 ± 3.8	-1.0 ± 0.7	0.3	2.0	2.7	0.9	82
Scan weight (kg)	34.2 ± 4.5	1.0 ± 0.9	0.2	1.8	2.6	0.5	104
Daily gain birth-wean (g)	0.2 ± 0.04	0.0 ± 0.0	0.2	2.0	2.8	0.6	82
Daily gain wean-scan (g)	0.1 ± 0.03	0.0 ± 0.0	0.5	1.6	2.2	1.6	104

[1] Descriptions of column headings are given in Table 4.16

Table 8.31 Details of peaks for sire 15/98 Texel cross half-sib QTL analysis for a region of ovine chromosome two – ultrasound fat traits[1]

Trait	Phen mean ± adj. std	Estimate ± stderr	SD units	Permutation 95%	Permutation 99%	max $-\log_{10}$ prob	pos (cM) [CI]
Fat depth C (mm)	2.3 ± 0.7	0.1 ± 0.2	0.2	2.2	3.1	0.3	122

[1] Descriptions of column headings are given in Table 4.16

Table 8.32 Details of peaks for sire 15/98 Texel cross half-sib QTL analysis for a region of ovine chromosome two – dressing-out percentage[1]

Trait	Phen mean ● adj. std	Estimate ± stderr	SD units	Permutation 95%	Permutation 99%	max $-\log_{10}$ prob	pos (cM) [CI]
Dressing-out percentage	41.8 ± 2.4	0.7 ± 0.2	0.3	2.0	2.8	2.3*	72

Table 8.33 Details of peaks for sire 15/98 Texel cross half-sib QTL analysis for a region of ovine chromosome two – carcass linear traits[1]

Trait	Phen mean ± adj. std	Estimate ± stderr	SD units	Permutation 95%	Permutation 99%	max $-\log_{10}$ prob	pos (cM) [CI]
Carcass length (cm)	79.8 ± 1.9	0.2 ± 0.3	0.1	2.1	2.9	0.3	50
Width of forequarter (cm)	17.5 ± 0.8	-0.4 ± 0.2	0.6	1.8	2.5	1.4	122
Width of thorax (cm)	16.5 ± 0.7	-0.3 ± 0.2	0.5	1.8	2.4	1.2	122
Gigot width (cm)	22.7 ± 0.6	0.3 ± 0.1	0.5	2.0	2.9	2.2 *	34 [2 -104]

[1] Descriptions of column headings are given in Table 4.16

Table 8.34 Details of peaks for sire 15/98 Texel cross half-sib QTL analysis for a region of ovine chromosome two – loin muscle traits[1]

Trait	Phen mean ± adj. std	Estimate ● stderr	SD units	Permutation 95%	Permutation 99%	max $-\log_{10}$ prob	pos (cM) [CI]
M. longissimus width (mm)	57.9 ± 2.5	1.0 ± 0.6	0.4	2.4	3.3	1.0	72
M. longissimus depth (mm)	27.0 ± 2.0	0.8 ± 0.4	0.4	2.3	3.2	1.4	56
M. longissimus area (mm^2)	1,204.9 ± 110.6	47.9 ± 20.6	0.4	2.3	3.2	1.7	60
Loin muscle weight (g)	216.0 ± 20.4	12.4 ± 3.3	0.6	1.9	2.6	3.5 ** sug	66 [39 - 104]

[1] Descriptions of column headings are given in Table 4.16

Table 8.35 Details of peaks for sire 15/98 Texel cross half-sib QTL analysis for a region of ovine chromosome two – loin fat traits[1]

Trait	Phen mean ± adj. std	Estimate ± stderr	SD units	Permutation 95%	Permutation 99%	max -log$_{10}$ prob	pos (cM) [CI]
GR (mm)	8.8 ± 2.4	-0.8 ± 0.5	0.3	1.8	2.4	1.0	68
Fat depth C (mm)	2.2 ● 0.9	-0.4 ± 0.2	0.5	1.6	2.2	2.1 *	62 [34 -104]
Loin fat (g)	91.1 ± 52.7	9.8 ± 14.4	0.2	1.4	1.7	0.3	22 [50 -104]

[1] Descriptions of column headings are given in Table 4.16

Table 8.36 Details of peaks for sire 15/98 Texel cross half-sib QTL analysis for a region of ovine chromosome two - leg bone traits[1]

Trait	Phen mean ± adj. std	Estimate ± stderr	SD units	Permutation 95%	Permutation 99%	max -log$_{10}$ prob	pos (cM) [CI]
Pelvic bone weight (g)	100.9 ± 9.6	3.1 ± 2.1	0.3	1.9	2.7	0.8	78
Femur bone weight (g)	137.5 ± 10.9	2.2 ± 3.2	0.2	1.9	2.7	0.3	122
Total bone weight (g)	403.0 ± 32.6	8.2 ± 8.4	0.3	1.9	2.7	0.5	116
Femur bone length (mm)	167.5 ± 4.4	2.0 ± 1.4	0.5	1.9	2.7	0.9	122

Table 8.37 Details of peaks for sire 15/98 Texel cross half-sib QTL analysis for a region of ovine chromosome two – meat quality traits[1]

Trait	Phen mean ± adj. std	Estimate ± stderr	SD units	Permutation 95%	Permutation 99%	max -log$_{10}$ prob	pos (cM) [CI]
M. semimembranosus							
pH	5.4 ± 1.2	-0.5 ± 0.3	0.4	1.7	2.3	1.2	64
Colour "L*"	29.0 ± 5.5	-1.8 ± 1.3	0.3	1.8	2.2	0.8	66
Colour "a*"	15.5 ± 3.1	-1.3 ± 0.7	0.4	1.8	2.3	1.0	66
Colour "b*"	6.7 ± 1.5	-0.4 ± 0.3	0.3	1.9	2.4	0.7	52
Sarcomere lng. (μm)	1.8 ± 0.1	0.0 ± 0.0	0.3	2.0	2.7	0.7	82
Cooking loss (%)	36.7 ± 1.2	-0.3 ± 0.3	0.3	1.9	2.8	0.6	52
W-B peak force	9.7 ± 2.0	0.9 ± 0.7	0.4	2.0	2.7	0.6	10
W-B yield force	6.8 ± 1.7	-0.6 ± 0.5	0.4	1.9	2.7	0.6	120
M. longissimus							
pH	5.7 ± 0.6	0.1 ± 0.2	0.2	1.3	1.6	0.4	96
Colour "L*"	30.6 ± 2.3	-0.6 ± 0.6	0.3	1.9	2.7	0.5	22
Colour "a*"	14.5 ± 1.2	0.2 ± 0.3	0.2	1.9	2.6	0.3	58
Colour "b*"	6.1 ± 1.2	-0.2 ± 0.3	0.2	1.9	2.7	0.4	48
Sarcomere lng. (μm)	1.7 ± 0.1	0.0 ± 0.0	0.4	2.0	2.8	0.9	40
Cooking loss (%)	29.7 ± 2.8	-0.5 ± 0.6	0.2	2.0	2.7	0.4	50
W-B peak force	6.2 ± 1.5	0.6 ± 0.5	0.4	1.9	2.7	0.6	120
W-B yield force	4.8 ± 1.1	0.4 ± 0.3	0.4	1.9	2.6	0.6	120

[1] Descriptions of column headings are given in Table 4.16

8.8 Sire 122/99 Non-Significant Single Trait QTL Results

Table 8.38 Details of peaks for sire 122/99 Texel cross half-sib QTL analysis for a region of ovine chromosome two – liveweight traits[1]

Trait	Phen mean ± adj. std	Estimate ± stderr	SD units	Permutation 95%	Permutation 99%	max $-\log_{10}$ prob	pos (cM) [CI]
Birth weight (kg)	5.0 ± 0.8	-0.4 ± 0.4	0.5	2.0	2.8	0.5	36
Wean weight (kg)	24.8 ± 3.2	1.5 ± 1.1	0.5	1.8	2.4	0.8	88
Scan weight (kg)	35.2 ± 4.5	0.7 ± 1.2	0.2	2.0	2.7	0.2	64
Daily gain birth-wean (g)	0.2 ± 0.03	0.0 ± 0.0	0.6	1.8	2.5	1.2	78
Daily gain wean-scan (g)	0.1 ± 0.04	0.0 ± 0.0	0.4	2.3	3.0	0.8	82

[1] Descriptions of column headings are given in Table 4.16

Table 8.39 Details of peaks for sire 122/99 Texel cross half-sib QTL analysis for a region of ovine chromosome two – ultrasound muscle traits[1]

Traits	Phen mean ● adj. std	Estimate ● stderr	SD units	Permutation 95%	Permutation 99%	max $-\log_{10}$ prob	pos (cM) [CI]
M. longissimus width (mm)	57.1 ± 4.6	-0.9 ± 1.2	0.2	1.9	2.6	0.3	106
M. longissimus depth (mm)	21.5 ± 2.3	-0.4 ± 0.5	0.2	2.0	2.7	0.4	122
M. longissimus area (mm^2)	959.9 ± 164.0	-35.7 ± 38.1	0.2	1.9	2.6	0.5	114

[1] Descriptions of column headings are given in Table 4.16

Table 8.40 Details of peaks for sire 122/99 Texel cross half-sib QTL analysis for a region of ovine chromosome two – ultrasound fat traits[1]

Trait	Phen mean ± adj. std	Estimate ± stderr	SD units	Permutation 95%	Permutation 99%	max $-\log_{10}$ prob	pos (cM) [CI]
Fat depth C (mm)	2.1 ± 0.6	0.1 ± 0.2	0.1	1.6	2.1	0.1	58

[1] Descriptions of column headings are given in Table 4.16

Table 8.41 Details of peaks for sire 122/99 Texel cross half-sib QTL analysis for a region of ovine chromosome two – dressing-out percentage[1]

Trait	Phen mean ± adj. std	Estimate ± stderr	SD units	Permutation 95%	Permutation 99%	max $-\log_{10}$ prob	pos (cM) [CI]
Dressing-out percentage	41.9 ± 2.2	-2.1 ± 0.7	1.0	1.7	2.8	2.7* sug	76 [50-104]

[1] Descriptions of column headings are given in Table 4.16

Table 8.42 Details of peaks for sire 122/99 Texel cross half-sib QTL analysis for a region of ovine chromosome two – carcass linear traits[1]

Trait	Phen mean ● adj. std	Estimate ± stderr	SD units	Permutation 95%	Permutation 99%	max $-\log_{10}$ prob	pos (cM) [CI]
Carcass length (cm)	80.4 ± 2.0	-1.2 ± 0.6	0.6	1.9	2.6	1.2	50
Width of forequarter (cm)	17.4 ● 0.7	0.3 ± 0.2	0.5	2.0	2.8	1.0	64
Width of thorax (cm)	16.2 ± 0.6	-0.4 ± 0.2	0.6	2.0	2.8	1.4	122
Gigot width (cm)	23.1 ± 0.6	-0.3 ± 0.1	0.5	1.9	2.6	1.2	118

[1] Descriptions of column headings are given in Table 4.16

Table 8.43 Details of peaks for sire 122/99 Texel cross half-sib QTL analysis for a region of ovine chromosome two – loin muscle traits[1]

Trait	Phen mean ± adj. std	Estimate ± stderr	SD units	Permutation 95%	99%	max $-\log_{10}$ prob	pos (cM) [CI]
M. longissimus width (mm)	56.3 ± 3.1	-2.4 ± 1	0.8	1.6	2.2	1.9 *	96 [54 - 122]
M. longissimus depth (mm)	25.3 ● 2.1	3.0 ± 1.9	1.4	1.8	2.4	0.9	4
M. longissimus area (mm^2)	1,097.2 ± 108.2	102.7 ± 98.9	1.0	1.8	2.5	0.5	4

[1] Descriptions of column headings are given in Table 4.16

Table 8.44 Details of peaks for sire 122/99 Texel cross half-sib QTL analysis for a region of ovine chromosome two – loin fat traits[1]

Trait	Phen mean ± adj. std	Estimate ± stderr	SD units	Permutation 95%	99%	max $-\log_{10}$ prob	pos (cM) [CI]
GR (mm)	7.8 ± 2.2	1.3 ± 0.7	0.6	2.0	2.9	1.2	64
Fat depth C (mm)	2.0 ± 0.7	-0.3 ± 0.2	0.4	2.1	3.0	0.7	122

[1] Descriptions of column headings are given in Table 4.16

8.9 Sire 429/98 Non-Significant Single Trait QTL Results

Table 8.45 Details of peaks for sire 429/98 Texel cross half-sib QTL analysis for a region of ovine chromosome two – liveweight traits[1]

Trait	Phen mean ± adj. std	Estimate ● stderr	SD units	Permutation 95%	Permutation 99%	max $-\log_{10}$ prob	pos (cM) [CI]
Birth weight (kg)	5.1 ± 0.8	-0.3 ± 0.1	0.3	2.2	3.0	1.8	52
Wean weight (kg)	23.5 ± 2.9	-1.3 ± 0.4	0.4	2.2	3.0	2.7 *	48 [29 - 122]
Scan weight (kg)	37.2 ± 3.5	-1.2 ± 0.5	0.3	2.1	2.9	1.6	46
Daily gain birth-wean (g)	0.2 ± 0.04	0.0 ± 0.0	0.5	2.2	3.1	2.1	32
Daily gain wean-scan (g)	0.2 ± 0.03	0.0 ± 0.0	0.1	2.2	2.9	0.3	54

[1] Descriptions of column headings are given in Table 4.16

Table 8.46 Details of peaks for sire 429/98 Texel cross half-sib QTL analysis for a region of ovine chromosome two – ultrasound muscle traits[1]

Trait	Phen mean ± adj. std	Estimate ± stderr	SD units	Permutation 95%	Permutation 99%	max $-\log_{10}$ prob	pos (cM) [CI]
M. longissimus width (mm)	59.4 ± 3.8	-1.3 ± 0.6	0.3	2.1	2.8	1.4	34
M. longissimus depth (mm)	23.8 ± 1.6	-0.4 ± 0.3	0.3	2.1	2.9	0.9	14
M. longissimus area (mm^2)	1,093.8 ± 132.2	-45.1 ± 23.8	0.3	2.1	2.9	1.2	24

[1] Descriptions of column headings are given in Table 4.16

Table 8.47 Details of peaks for sire 429/98 Texel cross half-sib QTL analysis for a region of ovine chromosome two – ultrasound fat traits[1]

Trait	Phen mean ± adj. std	Estimate ± stderr	SD units	Permutation 95%	Permutation 99%	max $-\log_{10}$ prob	pos (cM) [CI]
Fat depth C (mm)	1.7 ± 0.7	0.3 ± 0.1	0.5	1.9	2.6	2.6 *	64 [15 - 113]

[1] Descriptions of column headings are given in Table 4.16

Table 8.48 Details of peaks for sire 429/98 Texel cross half-sib QTL analysis for a region of ovine chromosome two – dressing-out percentage[1]

Trait	Phen mean ● adj. std	Estimate ± stderr	SD units	Permutation 95%	Permutation 99%	max $-\log_{10}$ prob	pos (cM) [CI]
Dressing-out percentage	40.8 ± 1.7	-0.6 ± 0.2	0.3	1.6	2.0	2.2 **	96

[1] Descriptions of column headings are given in Table 4.16

Table 8.49 Details of peaks for sire 429/98 Texel cross half-sib QTL analysis for a region of ovine chromosome two – carcass linear traits[1]

Trait	Phen mean ± adj. std	Estimate ± stderr	SD units	Permutation 95%	Permutation 99%	max $-\log_{10}$ prob	pos (cM) [CI]
Carcass length (cm)	80.5 ± 1.9	-0.4 ± 0.3	0.2	2.0	2.8	0.8	36
Width of forequarter (cm)	17.4 ± 0.8	-0.4 ± 0.1	0.5	2.0	2.6	2.6 *	42 [28 -122]
Width of thorax (cm)	16.4 ± 0.7	-0.1 ± 0.1	0.1	2.0	2.8	0.3	28
Gigot width (cm)	22.1 ± 0.6	-0.2 ± 0.1	0.4	2.1	2.8	1.4	32

[1] Descriptions of column headings are given in Table 4.16

Table 8.50 Details of peaks for sire 429/98 Texel cross half-sib QTL analysis for a region of ovine chromosome two – loin muscle traits[1]

Trait	Phen mean ± adj. std	Estimate ● stderr	SD units	Permutation 95%	99%	max -$\log_{10}$ prob	pos (cM) [CI]
M. longissimus width (mm)	56.6 ± 3.5	-1.0 ± 0.5	0.3	2.3	3.1	1.3	82
M. longissimus depth (mm)	27.0 ± 2.2	0.2 ± 0.3	0.1	2.1	2.9	0.3	122
M. longissimus area (mm^2)	1,179.5 ± 126.3	-21.4 ± 19.8	0.2	2.1	2.9	0.6	82
Loin muscle weight (g)	207.3 ± 15.6	4.0 ± 3.5	0.3	1.7	2.2	0.6	122

[1] Descriptions of column headings are given in Table 4.16

Table 8.51 Details of peaks for sire 429/98 Texel cross half-sib QTL analysis for a region of ovine chromosome two – loin fat traits[1]

Trait	Phen mean ● adj. std	Estimate ± stderr	SD units	Permutation 95%	99%	max -$\log_{10}$ prob	pos (cM) [CI]
GR (mm)	5.6 ± 1.6	0.5 ± 0.2	0.3	2.1	2.8	1.7	58
Fat depth C (mm)	2.8 ± 1.0	0.5 ± 0.2	0.5	2.1	2.9	2.0	32
Loin fat (g)	80.9 ± 21.8	19.1 ± 5.5	0.9	2.3	3.3	3.2 95 , [sug]	28 [2 - 122]

[1] Descriptions of column headings are given in Table 4.16

Table 8.52 Details of peaks for sire 429/98 Texel cross half-sib QTL analysis for a region of ovine chromosome two – leg bone traits[1]

Trait	Phen mean ± adj. std	Estimate ± stderr	SD units	Permutation 95%	99%	max -$\log_{10}$ prob	pos (cM) [CI]
Pelvic bone weight (g)	105.3 ± 7.9	3.3 ± 2.0	0.4	1.9	2.5	1.0	42
Femur bone weight (g)	147.2 ± 9.6	4.0 ± 2.5	0.4	1.9	2.6	0.9	8
Total bone weight (g)	407.6 ± 28.2	14.2 ± 8.1	0.5	1.8	2.5	1.1	14
Femur bone length (mm)	173.6 ± 4.2	1.7 ± 1.0	0.4	2.1	2.9	1.0	104

[1] Descriptions of column headings are given in Table 4.16

8.10 Sire 535/98 Non-Significant Single Trait QTL Results

Table 8.53 Details of peaks for sire 535/98 Texel cross half-sib QTL analysis for a region of ovine chromosome two – liveweight traits[1]

Trait	Phen mean ± adj. std	Estimate ± stderr	SD units	Permutation 95%	Permutation 99%	max $-\log_{10}$ prob	pos (cM) [CI]
Birth weight (kg)	5.3 ± 0.8	0.0 ± 0.1	0.1	1.8	2.5	0.1	122
Wean weight (kg)	24.0 ± 2.8	0.3 ± 0.4	0.1	1.9	2.6	0.3	122
Scan weight (kg)	34.0 ± 4.1	0.2 ± 0.5	0.1	2.0	2.7	0.2	122
Daily gain birth-wean (g)	0.3 ± 0.03	0.0 ± 0.0	0.1	1.9	2.5	0.3	80
Daily gain wean-scan (g)	0.1 ± 0.03	0.0 ± 0.0	0.1	1.9	2.6	0.2	82

[1] Descriptions of column headings are given in Table 4.16

Table 8.54 Details of peaks for sire 535/98 Texel cross half-sib QTL analysis for a region of ovine chromosome two – ultrasound muscle traits[1]

Trait	Phen mean ± adj. std	Estimate ± stderr	SD units	Permutation 95%	Permutation 99%	max $-\log_{10}$ prob	pos (cM) [CI]
M. longissimus width (mm)	58.5 ± 3.9	1.3 ± 0.7	0.3	2.0	2.7	1.1	36
M. longissimus depth (mm)	21.4 ± 2.0	0.7 ± 0.4	0.3	2.0	2.7	0.9	18
M. longissimus area (mm^2)	971.3 ± 147.0	54.7 ± 30.7	0.4	2.0	2.6	1.1	26

[1] Descriptions of column headings are given in Table 4.16

Table 8.55 Details of peaks for sire 535/98 Texel cross half-sib QTL analysis for a region of ovine chromosome two – ultrasound fat traits[1]

Trait	Phen mean ± adj. std	Estimate ± stderr	SD units	Permutation 95%	Permutation 99%	max $-\log_{10}$ prob	pos (cM) [CI]
Fat depth C (mm)	1.6 ± 0.5	-0.3 ± 0.1	0.6	2.3	3.0	1.6	106

[1] Descriptions of column headings are given in Table 4.16

Table 8.56 Details of peaks for sire 535/98 Texel cross half-sib QTL analysis for a region of ovine chromosome two – dressing-out percentage[1]

Trait	Phen mean ± adj. std	Estimate ± stderr	SD units	Permutation 95%	Permutation 99%	max $-\log_{10}$ prob	pos (cM) [CI]
Dressing-out percentage	41.6 ± 1.9	-1.1 ± 0.5	0.6	1.6	2.0	1.5	2

[1] Descriptions of column headings are given in Table 4.16

Table 8.57 Details of peaks for sire 535/98 Texel cross half-sib QTL analysis for a region of ovine chromosome two – carcass linear traits[1]

Trait	Phen mean ± adj. std	Estimate ± stderr	SD units	Permutation 95%	Permutation 99%	max $-\log_{10}$ prob	pos (cM) [CI]
Carcass length (cm)	79.8 ± 2.1	-0.4 ± 0.3	0.2	2.0	2.8	0.7	84
Width of forequarter (cm)	16.6 ± 0.8	0.2 ± 0.1	0.2	2.0	2.8	0.8	122
Width of thorax (cm)	15.9 ± 0.7	0.17 ± 0.1	0.3	2.0	2.7	1.4	122
Gigot width (cm)	21.6 ± 0.7	-0.1 ± 0.1	0.2	2.0	2.7	0.9	122

[1] Descriptions of column headings are given in Table 4.16

Table 8.58 Details of peaks for sire 535/98 Texel cross half-sib QTL analysis for a region of ovine chromosome two – leg muscle weight traits[1]

Trait	Phen mean ± adj. std	Estimate ● stderr	SD units	Permutation 95%	99%	max $-\log_{10}$ prob	pos (cM) [CI]
Trimmed leg (g)	2,541.0 ± 136.3	30.9 ± 24.5	0.2	2.5	3.4	0.7	54
M. semimembranosus (g)	232.8 ± 18.8	-4.0 ± 4.2	0.2	2.2	3.0	0.5	82
M. semitendinosus (g)	80.9 ± 8.1	1.7 ± 2.1	0.2	2.0	2.6	0.4	72
M. biceps femoris (g)	224.1 ± 18.0	3.9 ± 3.7	0.2	2.3	3.2	0.5	54
M. quadriceps (g)	342.6 ± 24.1	-3.9 ± 4.3	0.2	2.3	3.1	0.4	122
M. adductor (g)	106.4 ± 9.6	-6.1 ± 2.7	0.6	1.9	2.6	1.6	88
M. gluteus medius (g)	155.8 ± 15.1	-3.0 ± 3.7	0.2	2.1	2.9	0.4	112
Muscle trim (g)	620.0 ± 46.0	10.0 ± 9.3	0.2	2.1	2.8	0.5	58
Total leg muscle (g)	1,762.7 ± 115.1	16.1 ± 19.7	0.1	2.2	3.0	0.4	54

[1] Descriptions of column headings are given in Table 4.16

Table 8.59 Details of peaks for sire 535/98 Texel cross half-sib QTL analysis for a region of ovine chromosome two – other muscle related traits[1]

Trait	Phen mean ± adj. std	Estimate ± stderr	SD units	Permutation 95%	99%	max $-\log_{10}$ prob	pos (cM) [CI]
Leg muscularity	0.4 ● 0.02	-0.004 ± 0.0004	0.2	1.8	2.4	0.4	66
Leg muscle percent	69.3 ● 2.2	-0.54 ● 0.4	0.2	1.8	2.5	0.7	68
Femur muscle to bone	4.2 ± 0.4	0.1 ● 0.1	0.3	2.1	2.8	0.7	116

[1] Descriptions of column headings are given in Table 4.16

Table 8.60 Details of peaks for sire 535/98 Texel cross half-sib QTL analysis for a region of ovine chromosome two – leg fat weight traits[1]

Trait	Phen mean ± adj. std	Estimate ± stderr	SD units	Permutation 95%	99%	max $-\log_{10}$ prob	pos (cM) [CI]
Subcutaneous fat (g)	201.1 ± 35.3	22.4 ± 8.4	0.6	2.0	2.8	2.1 *	64
Intermuscular fat (g)	141.0 ± 23.4	2.5 ± 4.5	0.1	2.0	2.6	0.2	122
Total leg fat (g)	342.1 ± 49.2	22.3 ± 10.7	0.5	2.0	2.7	1.4	64
Leg fat %	11.7 ± 1.8	0.7 ± 0.4	0.4	1.2	1.5	0.3	112

[1] Descriptions of column headings are given in Table 4.16

Table 8.61 Details of peaks for sire 535/98 Texel cross half-sib QTL analysis for a region of ovine chromosome two – loin muscle traits[1]

Trait	Phen mean ● adj. std	Estimate ● stderr	SD units	Permutation 95%	99%	max $-\log_{10}$ prob	pos (cM) [CI]
M. longissimus width (mm)	55.3 ± 3.5	0.6 ± 0.5	0.2	1.6	2.1	0.6	122
M. longissimus depth (mm)	25.8 ± 2.2	-0.5 ± 0.3	0.2	2.0	2.7	0.7	122
M. longissimus area (mm^2)	1,101.3 ± 127.5	20.9 ± 20.4	0.2	1.9	2.6	0.5	52
Loin muscle weight (g)	193.2 ± 18.4	-2.8 ± 3.5	0.2	2.3	3.2	0.4	120

[1] Descriptions of column headings are given in Table 4.16

Table 8.62 Details of peaks for sire 535/98 Texel cross half-sib QTL analysis for a region of ovine chromosome two – loin fat traits[1]

Trait	Phen mean ● adj. std	Estimate ± stderr	SD units	Permutation 95%	99%	max $-\log_{10}$ prob	pos (cM) [CI]
GR (mm)	4.6 ± 1.8	0.4 ● 0.3	0.2	2.0	2.7	0.9	106
Fat depth C (mm)	2.6 ± 1.0	0.4 ● 0.4	0.4	1.9	2.6	0.5	12
Loin fat (g)	71.8 ± 19.4	9.5 ± 10.1	0.5	1.7	2.3	0.5	10

[1] Descriptions of column headings are given in Table 4.16

Table 8.63 Details of peaks for sire 535/98 Texel cross half-sib QTL analysis for a region of ovine chromosome two – leg bone traits[1]

Trait	Phen mean ± adj. std	Estimate ± stderr	SD units	Permutation 95%	Permutation 99%	max $-\log_{10}$ prob	pos (cM) [CI]
Pelvic bone weight (g)	107.8 ± 8.6	-4.1 ± 1.8	0.5	2.2	3.1	1.7	118
Femur bone weight (g)	154.4 ± 11.7	-1.7 ± 2.0	0.2	2.2	2.9	0.4	122
Total bone weight (g)	420.1 ± 32.0	-10.4 ± 5.9	0.3	2.2	3.1	1.1	122
Femur bone length (mm)	172.8 ± 4.0	0.8 ● 1.0	0.2	1.9	2.6	0.3	48

[1] Descriptions of column headings are given in Table 4.16

Table 8.64 Details of peaks for sire 535/98 Texel cross half-sib QTL analysis for a region of ovine chromosome two – meat quality traits[1]

Trait	Phen mean ± adj. std	Estimate ● stderr	SD units	Permutation 95%	Permutation 99%	max $-\log_{10}$ prob	pos (cM) [CI]
M. semimembranosus							
pH	5.6 ± 0.1	0.0 ± 0.0	0.3	1.8	2.4	0.8	78
Colour "L*"	29.8 ± 1.9	-0.7 ± 0.3	0.4	1.9	2.6	1.7	122
Colour "a*"	14.9 ± 1.2	0.1 ± 0.2	0.1	2.1	2.7	0.2	122
Colour "b*"	6.3 ± 0.8	-0.1 ± 0.2	0.2	2.0	2.6	0.3	112
Sarcomere lng. (µm)	1.7 ± 0.4	0.1 ± 0.2	0.3	2.0	2.8	0.3	104
Cooking loss (%)	37.2 ± 1.2	-0.3 ± 0.3	0.3	1.9	2.5	0.6	122
W-B peak force	11.3 ± 3.0	1.7 ± 0.7	0.6	2.4	3.2	2.0	122
W-B yield force	8.6 ± 2.6	1.5 ± 0.6	0.6	2.3	3.2	2.0	122
M. longissimus							
pH	5.7 ± 0.2	0.0 ± 0.0	0.2	2.0	2.7	0.5	80
Colour "L*"	31.3 ± 2.2	-1.1 ± 0.4	0.5	2.2	3.0	2.0	116
Colour "a*"	14.7 ± 1.5	-0.3 ± 0.4	0.2	1.7	2.4	0.5	50
Colour "b*"	6.2 ± 1.1	-0.6 ± 0.2	0.5	2.1	2.7	1.7	86
Sarcomere lng. (µm)	1.8 ± 0.1	0.0 ± 0.0	0.3	2.2	3.0	0.5	98
Cooking loss (%)	30.0 ± 2.1	-0.6 ± 0.3	0.3	1.9	2.5	1.2	120
W-B peak force	7.1 ± 1.9	1.0 ± 0.4	0.6	2.3	3.1	1.7	50
W-B yield force	5.6 ± 1.2	1.8 ± 0.8	1.5	2.3	3.1	1.7	2

[1] Descriptions of column headings are given in Table 4.16

8.11 Weighting Factors for Multi-Trait Analysis

8.11.1 Leg Muscle Traits

Table 8.65 Weighting factors for multi-trait analysis (multivariate (Multi) and principal component (PC)) of leg muscle traits

	Sire and Weighting Factor											
	1199/00		1170/00		150/96		15/98		429/98		535/98	
Traits	**PC**	**Multi**	**PC**	**Multi**	**PC**	**Multi**	**PC**	**Multi**	**PC**	**Multi**	**PC**	**Multi**
M. semimembranosus	0.6	1.1	0.4	1.3	0.4	0.4	0.3	0.5	0.4	0.4	0.4	0.6
M. semitendinosus	0.4	0.8	0.3	-0.1	0.2	-1.2	0.3	1.0	0.3	0.5	0.4	-1.0
M. biceps femoris	0.4	0.8	0.4	0.0	0.5	-0.5	0.4	-0.4	0.4	0.5	0.4	0.3
M. quadriceps	0.4	-1.2	0.4	-0.0	0.4	0.2	0.4	1.3	0.5	0.3	0.4	0.3
M. adductor	0.3	-0.1	0.3	0.5	0.4	0.9	0.2	0.4	0.4	0.9	0.3	1.1
M. gluteus medius	0.4	0.5	0.4	-0.2	0.4	0.3	0.3	-0.5	0.0	-0.5	0.3	-0.6

8.11.2 Leg fat weight traits

Table 8.66 Weighting factors for multi-trait analysis (multivariate (Multi) and principal component (PC)) of leg fat weight traits

	Sire and Weighting Factor											
	1199/00		1170/00		150/96		15/98		429/98		535/98	
Traits	**PC**	**Multi**	**PC**	**Multi**	**PC**	**Multi**	**PC**	**Multi**	**PC**	**Multi**	**PC**	**Multi**
Subcutaneous fat	0.7	1.1	0.7	2.2	0.7	0.9	0.7	1.3	0.7	1.7	0.7	1.3
Intermuscular fat	0.7	0.7	0.7	0.4	0.7	0.9	0.7	0.5	0.7	0.2	0.7	-0.5

8.11.3 Loin Muscle Traits

Table 8.67 Weighting factors for multi-trait analysis (multivariate (Multi) and principal component (PC)) of loin muscle traits

	Sire and Weighting Factor											
	1199/00		1170/00		150/96		15/98		429/98		535/98	
Traits	**PC**	**Multi**	**PC**	**Multi**	**PC**	**Multi**	**PC**	**Multi**	**PC**	**Multi**	**PC**	**Multi**
M. longissimus width	0.5	0.7	0.3	1.3	0.5	1.4	0.5	0.3	0.4	-0.7	0.4	-1.0
M. longissimus depth	-0.1	0.9	0.0	-0.4	0.2	0.9	-0.1	-0.1	-0.3	-0.2	0.1	-0.8
M. longissimus area	0.7	-0.6	0.7	0.1	0.7	-1.2	0.7	0.1	0.7	1.0	0.7	1.2
M. longissimus	0.6	1.0	0.6	-0.2	0.6	-0.1	0.6	2.0	0.6	0.5	0.6	0.5

8.11.4 Loin Fat Traits

Table 8.68 Weighting factors for multi-trait analysis (multivariate (Multi) and principal component (PC)) of loin fat traits

	Sire and Weighting Factor											
	1199/00		1170/00		150/96		15/98		429/98		535/98	
Traits	**PC**	**Multi**	**PC**	**Multi**	**PC**	**Multi**	**PC**	**Multi**	**PC**	**Multi**	**PC**	**Multi**
M. longissimus fat	0.6	1.2	0.6	1.1	0.5	-1.6	0.1	-0.2	0.6	0.5	0.6	-0.3
M. longissimus fat depth	0.6	0.3	0.6	0.2	0.6	0.3	0.7	1.3	0.6	0.8	0.6	0.2
GR fat depth	0.6	-0.3	0.5	0.8	0.6	1.4	0.7	-0.3	0.5	0.5	0.5	1.2

8.12 Linkage Disequilibrium Non-Significant Results

8.12.1 Tanbar Flock

Table 8.69 Results where adjusting the QTL analysis for the effects of linkage disequilibrium did not result in an increase in significance thresholds for the Tanbar Flock

	QTL Analysis			QTL Analysis Adjusted for Linkage Disequilibrium		
	Marker	$-\log_{10}P$	sug/sig[1]	Marker	$-\log_{10}P$	sug/sig[1]
Birth weight (kg)	TEXAN2	0.3		INRA40	0.4	
Weaning weight (kg)	BM81124	0.3		TEXAN2	0.4	
Scan weight (g)	FCB128	0.8		TEXAN2	1.2	
Daily gain birth to weaning (g)	BM81124	0.4		TEXAN2	0.4	
Daily gain weaning to scanning (g)	TEXAN2	0.9		FCB20	0.9	
Ultrasound *M. longissimus* width (mm)	FCB20	0.4		RM356	1.6	
Ultrasound *M. longissimus* depth (mm)	BM81124	0.5		RM356	0.9	
Ultrasound *M. longissimus* area (mm^2)	BM81124	0.3		RM356	1.4	
Ultrasound fat depth C (mm)	FCB128	1.0		FCB128	0.6	
Carcass length (cm)	FCB128	0.3		FCB20	0.2	
Width of forequarter (cm)	RM356	0.4		FCB128	0.8	
Width of thorax (cm)	BM81124	0.8		BULGE20	1.2	
Gigot width (cm)	ILST30	1.1		BULGE20	1.6	
Trimmed leg weight (g)	BULGE20	1.2		BULGE20	1.1	
M. semitendinosus weight (g)	BM81124	3.8	sug	BM81124	3.5	sug
M. quadriceps weight (g)	ILST30	0.9		BULGE20	0.9	
M. adductor weight (g)	BULGE20	2.3		BULGE20	2.4	
M. gluteus medius weight (g)	BULGE20	3.1	sug	BM81124	3.1	sug
Total muscle weight (g)	BULGE20	3.6	sug	BULGE20	3.9	sug
Leg muscularity	BULGE20	3.1	sug	BULGE20	3.3	sug
Leg muscle percent	BULGE20	5.5	sug	BULGE20	5.5	sug
Femur muscle to bone	BULGE20	3.1	sug	BULGE20	3.7	sug
Leg subcutaneous fat weight (g)	BULGE20	2.7		BULGE20	2.3	
Leg intermuscular fat weight (g)	BULGE20	1.9		ILST30	2.6	
Leg total fat weight (g)	BULGE20	3.1	sug	BULGE20	2.4	
M. longissimus width (mm)	BULGE20	1.5		BM81124	2.0	
M. longissimus depth (mm)	BULGE20	0.7		INRA40	0.9	
M. longissimus area (mm^2)	BULGE20	1.3		BM81124	1.4	
M. longissimus weight (g)	BULGE20	1.5		BM81124	2.0	
GR (mm)	BM81124	1.1		TEXAN2	1.4	
Fat depth C (mm)	BULGE20	0.6		BM81124	0.6	
Loin subcutaneous fat weight (g)	BULGE20	1.1		TEXAN2	1.5	
Pelvic bone weight (g)	ILST30	0.6		FCB20	0.6	
Femur bone weight (g)	ILST30	0.3		TEXAN2	0.5	
Total leg bone weight (g)	BM81124	0.5		RM356	0.5	
Femur length (cm)	TEXAN2	0.5		INRA40	0.9	
pH *M. semimembranosus*	ILST30	0.6		RM356	0.6	
Colour "L*" *M. semimembranosus*	INRA40	0.7		FCB128	1.0	
Colour "a*" *M. semimembranosus*	FCB20	0.6		FCB20	0.7	
Colour "b*" *M. semimembranosus*	FCB20	0.6		FCB20	0.7	
Sarcomere length *M. semi* (μm)	TEXAN2	0.4		FCB20	0.8	
Cooking loss *M. semi.* (%)	BM81124	0.7		BM81124	1.2	
Warner Bratzler peak force *M. semi.*	FCB20	0.4		RM356	0.5	

	QTL Analysis			QTL Analysis Adjusted for Linkage Disequilibrium		
	Marker	-log$_{10}$P	sug/sig[1]	Marker	-log$_{10}$P	sug/sig[1]
Warner Bratzler yield force *M. semi.*	RM356	0.6		RM356	0.5	
pH *M. longissimus*	FCB128	0.3		BM81124	0.4	
Colour "L*" *M. longissimus*	FCB128	0.3		ILST30	0.9	
Colour "a*" *M. longissimus*	BULGE20	0.8		RM356	1.3	
Colour "b*" *M. longissimus*	BM81124	0.9		RM356	1.1	
Sarcomere length *M. long.* (μm)	BM81124	0.6		RM356	0.8	
Cooking loss *M. longissimus* (%)	BULGE20	0.5		INRA40	0.5	
Warner Bratzler peak force *M. long.*	BM81124	1.3		BM81124	1.2	
Warner Bratzler yield force *M. long.*	BM81124	1.1		FCB128	1.0	

[1]The significance threshold defined in Table 4.16

8.12.2 Skye Flock

Table 8.70 Results where adjusting the QTL analysis for the effects of linkage disequilibrium did not result in an increase in significance thresholds for the Skye Flock

	QTL Analysis			QTL Analysis Adjusted for Linkage Disequilibrium		
	Marker	-log$_{10}$P	sug/sig[1]	Marker	-log$_{10}$P	sug/sig[1]
Birth weight (kg)	TGLA10	0.9		TGLA10	1.9	
Weaning weight (kg)	ILST30	1.5		ILST30	1.8	
Scan weight (g)	FCB20	0.7		FCB20	0.9	
Daily gain birth to weaning (g)	ILST30	1.3		ILST30	2.0	
Daily gain weaning to scanning (g)	FCB20	1.5		FCB20	1.8	
Ultrasound *M. longissimus* width (mm)	BULGE20	1.8		BULGE20	2.2	
Ultrasound *M. longissimus* depth (mm)	BULGE20	2.7		BULGE20	2.8	
Ultrasound *M. longissimus* area (mm2)	BULGE20	3.0	sug	BULGE20	3.2	sug
Ultrasound fat depth C (mm)	FCB20	0.6		FCB20	0.6	
Carcass length (cm)	TEXAN2	0.9		TEXAN2	0.8	
Width of forequarter (cm)	FCB20	1.3		FCB20	1.2	
Width of thorax (cm)	FCB20	1.3		FCB20	1.4	
Gigot width (cm)	TEXAN2	1.4		BULGE20	1.3	
Trimmed leg weight (g)	BULGE20	2.4		TGLA10	2.1	
M. semimembranosus weight (g)	INRA40	2.9	sug	FCB20	3.3	sug
M. semitendinosus weight (g)	BULGE20	3.1	sug	TEXAN2	3.2	sug
M. biceps femoris weight (g)	TGLA10	1.7		TGLA10	1.7	
M. quadriceps weight (g)	BULGE20	4.0	sug	BULGE20	3.2	sug
M. adductor weight (g)	BULGE20	1.7		BM81124	1.3	
M. gluteus medius weight (g)	INRA40	1.0		TGLA10	1.4	
Muscle trim weight (g)	BULGE20	3.4	sug	TEXAN2	3.1	sug
Total muscle weight (g)	BULGE20	4.7	sig	BULGE20	3.6	sug
Leg muscle percent	BULGE20	4.3	sug	TEXAN2	3.2	sug
Femur muscle to bone	BULGE20	2.4		TEXAN2	2.3	
Leg subcutaneous fat weight (g)	BULGE20	2.4		BULGE20	1.7	
Leg intermuscular fat weight (g)	TEXAN2	1.3		TGLA10	1.6	
Leg total fat weight (g)	TEXAN2	2.6		TEXAN2	2.3	
M. longissimus width (mm)	RM356	1.3		RM356	1.1	
M. longissimus depth (mm)	INRA40	1.4		INRA40	1.2	
M. longissimus area (mm^2)	INRA40	1.7		BULGE20	1.4	

	QTL Analysis			QTL Analysis Adjusted for Linkage Disequilibrium		
	Marker	**-log$_{10}$P**	**sug/sig[1]**	**Marker**	**-log$_{10}$P**	**sug/sig[1]**
M. longissimus weight (g)	TEXAN2	3.5	sug	TEXAN2	4.0	sug
GR (mm)	TEXAN2	1.2		TEXAN2	0.5	
Fat depth C (mm)	BULGE20	2.2		BULGE20	1.5	
Loin subcutaneous fat weight (g)	FCB20	0.3		TGLA10	0.5	
Pelvic bone weight (g)	ILST30	1.1		ILST30	1.0	
Femur bone weight (g)	ILST30	0.3		TGLA10	0.5	
Total leg bone weight (g)	ILST30	0.4		TGLA10	0.7	
Femur length (cm)	FCB20	1.0		FCB20	1.1	
pH *M. semimembranosus*	TEXAN2	1.0		TEXAN2	1.1	
Colour "L*" *M. semimembranosus*	BULGE20	0.7		BULGE20	0.9	
Colour "a*" *M. semimembranosus*	TEXAN2	0.9		BULGE20	1.2	
Colour "b*" *M. semimembranosus*	BULGE20	0.8		TGLA10	1.0	
Sarcomere length *M. semi* (μm)	TGLA10	0.5		TGLA10	1.4	
Cooking loss *M. semi.* (%)	BM81124	0.6		BULGE20	1.0	
Warner Bratzler peak force *M. semi.*	BULGE20	0.7		TGLA10	0.4	
Warner Bratzler yield force *M. semi.*	BULGE20	0.6		INRA40	0.7	
pH *M. longissimus*	FCB20	0.5		TEXAN2	0.6	
Colour "L*" *M. longissimus*	BULGE20	0.3		TGLA10	0.4	
Colour "a*" *M. longissimus*	INRA40	0.3		INRA40	0.4	
Colour "b*" *M. longissimus*	BULGE20	0.3		TGLA10	0.4	
Sarcomere length *M. long.* (μm)	BM81124	1.1		BULGE20	1.3	
Cooking loss *M. longissimus* (%)	BM81124	0.4		BULGE20	0.4	
Warner Bratzler peak force *M. long.*	ILST30	0.7		ILST30	1.3	
Warner Bratzler yield force *M. long.*	ILST30	0.6		ILST30	1.4	

[1]The significance threshold defined in Table 4.16

8.12.3 Woodlands Flock

Table 8.71 Results where adjusting the QTL analysis for the effects of linkage disequilibrium did not result in an increase in significance thresholds for the Woodlands Flock

	QTL Analysis			QTL Analysis Adjusted for Linkage Disequilibrium		
	Marker	**-log$_{10}$P**	**sug/sig[1]**	**Marker**	**-log$_{10}$P**	**sug/sig[1]**
Birth weight (kg)	BM81124	1.3		BM81124	1.5	
Weaning weight (kg)	BM81124	2.2		BM81124	2.3	
Scan weight (g)	TEXAN2	1.2		BULGE20	1.0	
Daily gain birth to weaning (g)	BM81124	1.4		BM81124	1.4	
Daily gain weaning to scanning (g)	FCB128	0.3		FCB128	0.4	
Ultrasound *M. longissimus* width (mm)	BM81124	1.2		BM81124	1.3	
Ultrasound *M. longissimus* depth (mm)	INRA40	0.4		BULGE20	0.6	
Ultrasound *M. longissimus* area (mm^2)	BM81124	0.9		BM81124	1.0	
Ultrasound fat depth C (mm)	BM81124	1.8		BULGE20	1.9	
Carcass length (cm)	ILST30	0.5		FCB128	0.6	
Width of forequarter (cm)	BULGE20	1.8		BULGE20	2.0	
Width of thorax (cm)	FCB128	0.4		BULGE20	0.3	
Gigot width (cm)	BULGE20	0.8		BM81124	0.9	
Trimmed leg weight (g)	INRA40	0.5		INRA40	1.1	
M. semimembranosus weight (g)	BM81124	1.6		BM81124	2.3	

	QTL Analysis			QTL Analysis Adjusted for Linkage Disequilibrium		
	Marker	**-log$_{10}$P**	**sug/sig[1]**	**Marker**	**-log$_{10}$P**	**sug/sig[1]**
M. semitendinosus weight (g)	BM81124	1.6		BM81124	1.3	
M. biceps femoris weight (g)	FCB20	1.0		INRA40	1.1	
M. quadriceps weight (g)	BM81124	0.7		BM81124	0.9	
M. gluteus medius weight (g)	BM81124	0.5		BM81124	0.7	
Muscle trim weight (g)	INRA40	1.3		INRA40	2.0	
Total muscle weight (g)	BM81124	1.7		BM81124	2.3	
Leg muscularity	BM81124	2.6		BM81124	2.6	
Leg muscle percent	BM81124	4.5	sig	BM81124	4.7	sig
Femur muscle to bone	BM81124	3.8	sug	BM81124	3.9	sug
Leg subcutaneous fat weight (g)	BULGE20	2.7		BULGE20	2.3	
Leg intermuscular fat weight (g)	BM81124	0.9		FCB20	0.6	
Leg total fat weight (g)	BM81124	2.9	sug	BULGE20	2.4	
M. longissimus width (mm)	ILST30	0.7		FCB128	0.6	
M. longissimus depth (mm)	INRA40	0.4		INRA40	0.4	
M. longissimus area (mm^2)	INRA40	0.4		FCB20	0.4	
M. longissimus weight (g)	BM81124	0.5		BM81124	0.6	
GR (mm)	BM81124	1.4		BM81124	1.5	
Fat depth C (mm)	BULGE20	1.4		BULGE20	1.2	
Loin subcutaneous fat weight (g)	BULGE20	2.1		BULGE20	1.8	
Pelvic bone weight (g)	RM356	1.2		RM356	1.0	
Femur bone weight (g)	BM81124	0.5		RM356	0.1	
Total leg bone weight (g)	FCB128	0.8		RM356	0.6	
Femur length (cm)	TEXAN2	0.4		INRA40	0.4	
pH *M. semimembranosus*	FCB20	1.4		ILST30	1.2	
Colour "L*" *M. semimembranosus*	BM81124	1.8		BM81124	1.4	
Colour "a*" *M. semimembranosus*	RM356	1.7		RM356	1.8	
Colour "b*" *M. semimembranosus*	RM356	1.5		RM356	1.4	
Sarcomere length *M. semi* (μm)	FCB20	0.4		FCB128	0.5	
Cooking loss *M. semi.* (%)	BULGE20	1.1		BULGE20	1.0	
Warner Bratzler peak force *M. semi.*	ILST30	1.2		ILST30	1.0	
Warner Bratzler yield force *M. semi.*	ILST30	1.2		ILST30	1.1	
pH *M. longissimus*	BM81124	0.9		FCB20	0.7	
Colour "L*" *M. longissimus*	INRA40	1.9		BULGE20	1.3	
Colour "a*" *M. longissimus*	BULGE20	1.0		BULGE20	0.9	
Colour "b*" *M. longissimus*	INRA40	1.5		INRA40	1.4	
Sarcomere length *M. long.* (μm)	RM356	0.3		FCB20	0.2	
Cooking loss *M. longissimus* (%)	RM356	1.1		RM356	1.1	
Warner Bratzler peak force *M. long.*	RM356	1.6		RM356	1.8	
Warner Bratzler yield force *M. long.*	RM356	1.2		RM356	1.2	

[1]The significance threshold defined in Table 4.16

8.13 Sex Interaction Analysis Non-Significant Results

8.13.1 Sire 1199/00

Table 8.72 Details of non-significant QTL-sex interaction analysis results for sire 1199/00 [1]

Trait	QTL - Sex Int., Signif. of Peaks		Sex Int., Maximum Signif.		Sex Int., Signif. at QTL - Sex Int. Peak
	Position	$-\log_{10}P$	Position	P Value[2]	
Birth weight (kg)	66	0.7	66	+	no
Scan weight (g)	62	0.9	50	ns	no
Slaughter weight (kg)	70	2.3	50	ns	no
Daily gain birth to weaning (g)	2	0.9	2	+	no
Daily gain weaning to scanning (g)	76	1.1	48	ns	no
Ultrasound *M. longissimus* width (mm)	56	0.7	56	+	no
Ultrasound *M. longissimus* depth (mm)	104	1.4	104	+	no
Ultrasound *M. longissimus* area (mm2)	104	0.9	56	ns	no
Ultrasound fat depth C (mm)	14	1.7	122	ns	no
Carcass length (cm)	76	0.8	76	+	no
Width of thorax (cm)	82	0.8	104	ns	no
Width of forequarter (cm)	12	1.2	56	+	no
Gigot width (cm)	64	0.2	104	ns	no
Dressing-out percentage	104	0.7	96	ns	no
Trimmed leg weight (g)	122	1.3	122	*	no
M. semimembranosus weight (g)	52	4.5	56	ns	ns
M. semitendinosus weight (g)	48	3.7	122	ns	ns
M. biceps femoris weight (g)	72	5.0	74	***	yes
M. quadriceps weight (g)	64	1.4	58	+	no
M. adductor weight (g)	68	1.1	66	ns	no
M. gluteus medius weight (g)	54	3.3	14	ns	ns
Muscle trim weight (g)	72	3.3	94	*	no
Total muscle weight (g)	54	5.5	90	+	ns
Leg muscularity	62	4.7	62	+	ns
Leg muscle percent	68	8.1	0	ns	ns
Femur muscle to bone	38	3.5	66	ns	ns
Leg subcutaneous fat weight (g)	52	5.0	0	ns	ns
Leg intermuscular fat weight (g)	78	3.6	122	ns	ns
Leg total fat weight (g)	70	6.1	122	ns	ns
Leg fat percentage	70	5.0	0	ns	ns
M. longissimus weight (g)	48	3.4	62	**	no
M. longissimus width (mm)	28	1.5	2	*	no
M. longissimus width (mm)	56	1.9	100	ns	no
M. longissimus area (mm^2)	58	2.5	98	*	no
GR (mm)	66	2.5	104	ns	no
Fat depth C (mm)	52	1.3	104	ns	no
Loin subcutaneous fat weight (g)	54	1.9	100	ns	no
Pelvic bone weight (g)	122	0.8	122	ns	no
Femur bone weight (g)	66	0.3	66	ns	no
Total leg bone weight (g)	122	0.5	122	ns	no
pH *M. semimembranosus*	114	0.9	94	ns	no
Colour "L*" *M. semimembranosus*	10	0.5	82	ns	no
Colour "a*" *M. semimembranosus*	118	1.1	120	+	no
Colour "b*" *M. semimembranosus*	104	1.3	106	+	no

Trait	QTL - Sex Int., Signif. of Peaks		Sex Int., Maximum Signif.		Sex Int., Signif. at QTL - Sex Int. Peak
	Position	$-\log_{10}P$	Position	P Value[2]	
Sarcomere length *M. semi.* (μm)	60	0.9	74	+	no
Cooking loss *M. semi.* (%)	64	1.2	64	+	no
Warner Bratzler peak force *M. semi.*	122	0.4	56	ns	no
Warner Bratzler yield force *M. semi.*	122	0.5	30	ns	no
pH *M. longissimus*	118	0.7	120	ns	no
Colour "L*" *M. longissimus*	82	0.6	82	ns	no
Sarcomere length *M. long.* (μm)	64	1.3	106	*	no
Colour "a*" *M. longissimus*	122	2.0	122	ns	no
Colour "b*" *M. longissimus*	122	1.9	122	+	no
Cooking loss *M. longissimus* (%)	28	1.3	66	+	no
Warner Bratzler peak force *M. long.*	56	1.3	82	ns	no
Warner Bratzler yield force *M. long.*	56	1.1	82	ns	no
Warner Bratzler mean force *M. long.*	56	1.1	86	ns	no

[1] Firstly a test for a QTL – Sex Interaction QTL was carried out, then a test for the presence of a sex interaction. In order to report a QTL – Sex interaction, there must be the presence of a significant QTL peak, at which point the interaction is also significant
[2] NS non-significant; + P<0.1; * P<0.05; ** P<0.01; *** P<0.001

8.13.2 Sire 1170/00

Table 8.73 Details of non-significant QTL-sex interaction analysis results for sire 1170/00 [1]

Trait	QTL - Sex Int., Signif. of Peaks		Sex Int., Maximum Signif.		Sex Int., Signif. at QTL - Sex Int. Peak
	Position	$-\log_{10}P$	Position	P Value[2]	
Birth weight (kg)	56	0.5	56	ns	no
Scan weight (g)	26	1.6	22	ns	no
Slaughter weight (kg)	8	1.0	66	ns	no
Daily gain birth to weaning (g)	52	1.7	52	*	no
Daily gain weaning to scanning (g)	0	2.6	0	*	no
Ultrasound *M. longissimus* width (mm)	122	1.2	56	Ns	no
Ultrasound *M. longissimus* depth (mm)	122	0.6	88	ns	no
Ultrasound *M. longissimus* area (mm2)	122	0.9	94	ns	no
Ultrasound fat depth C (mm)	116	0.5	108	ns	no
Carcass length (cm)	24	0.8	18	+	no
Width of thorax (cm)	56	1.2	122	ns	no
Width of forequarter (cm)	14	0.7	0	ns	no
Gigot width (cm)	86	1.1	24	ns	no
Dressing-out percentage	104	1.6	100	*	no
Trimmed leg weight (g)	62	1.8	122	ns	no
M. semimembranosus weight (g)	52	3.4	0	ns	ns
M. semitendinosus weight (g)	122	1.6	122	**	no
M. biceps femoris weight (g)	34	2.2	14	*	no
M. quadriceps weight (g)	70	1.2	122	ns	no
M. adductor weight (g)	52	2.1	56	ns	no
M. gluteus medius weight (g)	58	0.7	66	ns	no
Muscle trim weight (g)	52	1.8	4	+	no
Total muscle weight (g)	52	2.8	6	ns	ns
Leg muscularity	58	1.9	84	*	no
Leg muscle percent	40	3.0	14	*	no
Femur muscle to bone	20	1.1	8	+	no
Leg subcutaneous fat weight (g)	48	3.1	6	ns	ns

Trait	QTL - Sex Int., Signif. of Peaks		Sex Int., Maximum Signif.		Sex Int., Signif. at QTL - Sex Int. Peak
	Position	$-\log_{10}P$	Position	P Value[2]	
Leg intermuscular fat weight (g)	122	1.0	122	*	no
Leg total fat weight (g)	50	2.9	8	+	ns
Leg fat percentage	42	2.1	12	+	no
M. longissimus weight (g)	82	1.1	108	+	no
M. longissimus width (mm)	46	1.8	48	ns	no
M. longissimus width (mm)	122	0.9	116	ns	no
M. longissimus area (mm^2)	0	0.7	0	ns	no
GR (mm)	34	1.5	26	ns	no
Fat depth C (mm)	30	1.5	90	ns	no
Loin subcutaneous fat weight (g)	38	2.0	96	ns	no
Pelvic bone weight (g)	114	1.8	112	ns	no
Femur bone weight (g)	0	0.9	2	+	no
Total leg bone weight (g)	100	1.4	2	ns	no
pH *M. semimembranosus*	94	0.8	2	ns	no
Colour "L*" *M. semimembranosus*	122	0.7	122	ns	no
Colour "a*" *M. semimembranosus*	122	0.8	118	ns	no
Colour "b*" *M. semimembranosus*	112	0.6	110	+	no
Sarcomere length *M. semi* (µm)	64	1.5	64	*	no
Cooking loss *M. semi.* (%)	50	1.2	50	ns	no
Warner Bratzler peak force *M. semi.*	48	0.7	48	+	no
Warner Bratzler yield force *M. semi.*	98	0.6	48	ns	no
pH *M. longissimus*	94	0.7	92	ns	no
Colour "L*" *M. longissimus*	122	0.9	122	*	no
Sarcomere length *M. long.* (µm)	56	0.5	56	ns	no
Colour "a*" *M. longissimus*	122	1.1	122	ns	no
Colour "b*" *M. longissimus*	54	0.9	82	+	no
Cooking loss *M. longissimus* (%)	88	0.7	52	ns	no
Warner Bratzler peak force *M. long.*	36	2.4	42	**	no
Warner Bratzler yield force *M. long.*	30	2.2	54	**	no
Warner Bratzler mean force *M. long.*	36	1.6	48	*	no

[1] Firstly a test for a QTL – Sex Interaction QTL was carried out. then a test for the presence of a sex interaction. In order to report a QTL – Sex interaction, there must be the presence of a significant QTL peak, at which point the interaction is also significant
[2] NS non-significant; + P<0.1; * P<0.05; ** P<0.01; *** P<0.001

8.13.3 Sire 150/96

Table 8.74 Details of non-significant QTL-sex interaction analysis results for sire 150/96 [1]

Trait	QTL - Sex Int., Signif. of Peaks		Sex Int., Maximum Signif.		Sex Int., Signif. at QTL - Sex Int. Peak
	Position	$-\log_{10}P$	Position	P Value[2]	
Birth weight (kg)	66	0.7	122	ns	no
Scan weight (g)	122	1.3	62	+*	no
Slaughter weight (kg)	122	0.4	22	ns	no
Daily gain birth to weaning (g)	122	0.5	22	ns	no
Daily gain weaning to scanning (g)	110	0.7	110	+	no
Ultrasound *M. longissimus* width (mm)	8	0.6	2	ns	no
Ultrasound *M. longissimus* depth (mm)	122	0.8	122	ns	no
Ultrasound *M. longissimus* area (mm^2)	122	0.6	2	ns	no
Ultrasound fat depth C (mm)	6	0.9	2	*	no
Carcass length (cm)	122	0.2	122	ns	no

Trait	QTL - Sex Int., Signif. of Peaks		Sex Int., Maximum Signif.		Sex Int., Signif. at QTL - Sex Int. Peak
	Position	$-\log_{10}P$	Position	P Value[2]	
Width of thorax (cm)	112	1.4	122	+	no
Width of forequarter (cm)	122	0.6	104	ns	no
Gigot width (cm)	122	0.7	6	ns	no
Dressing-out percentage	122	0.7	122	ns	no
Trimmed leg weight (g)	104	0.9	96	ns	no
M. semimembranosus weight (g)	122	0.3	122	ns	no
M. semitendinosus weight (g)	122	0.6	116	ns	no
M. biceps femoris weight (g)	104	0.4	60	ns	no
M. quadriceps weight (g)	98	1.0	92	+	no
M. adductor weight (g)	94	1.5	104	ns	no
M. gluteus medius weight (g)	66	1.2	70	+*	no
Muscle trim weight (g)	100	1.8	96	**	no
Total muscle weight (g)	104	0.9	106	ns	no
Leg muscularity	66	1.5	122	ns	no
Leg muscle percent	116	0.4	118	ns	no
Femur muscle to bone	12	1.0	122	ns	no
Leg subcutaneous fat weight (g)	104	0.2	112	ns	no
Leg intermuscular fat weight (g)	102	0.2	88	ns	no
Leg total fat weight (g)	104	0.3	104	ns	no
Leg fat percentage	104	0.3	104	ns	no
M. longissimus weight (g)	122	0.6	122	ns	no
M. longissimus width (mm)	98	0.9	4	ns	no
M. longissimus width (mm)	26	0.2	4	ns	no
M. longissimus area (mm^2)	26	0.9	4	+	no
GR (mm)	66	0.3	86	ns	no
Fat depth C (mm)	122	0.2	122	ns	no
Loin subcutaneous fat weight (g)	76	0.6	92	ns	no
Pelvic bone weight (g)	0	0.6	122	ns	no
Femur bone weight (g)	66	0.4	66	ns	no
Total leg bone weight (g)	0	0.7	66	ns	no
pH *M. semimembranosus*	120	0.7	22	ns	no
Colour "L*" *M. semimembranosus*	122	0.7	122	+	no
Colour "a*" *M. semimembranosus*	104	0.7	104	ns	no
Colour "b*" *M. semimembranosus*	104	0.7	104	ns	no
Sarcomere length *M. semi* (μm)	66	0.5	116	ns	no
Cooking loss *M. semi.* (%)	66	1.3	66	*	no
Warner Bratzler peak force *M. semi.*	36	1.1	8	*	no
Warner Bratzler yield force *M. semi.*	36	0.6	0	ns	no
pH *M. longissimus*	122	0.1	4	ns	no
Colour "L*" *M. longissimus*	104	0.4	16	ns	no
Sarcomere length *M. long.* (μm)	62	0.7	64	+	no
Colour "a*" *M. longissimus*	0	0.3	122	ns	no
Colour "b*" *M. longissimus*	8	0.5	122	ns	no
Cooking loss *M. longissimus* (%)	122	0.5	122	ns	no
Warner Bratzler peak force *M. long.*	122	1.2	116	+*	no
Warner Bratzler yield force *M. long.*	6	1.0	122	ns	no
Warner Bratzler mean force *M. long.*	6	1.3	108	*	no

[1] Firstly a test for a QTL – Sex Interaction QTL was carried out, then a test for the presence of a sex interaction. In order to report a QTL – Sex interaction, there must be the presence of a significant QTL peak, at which point the interaction is also significant

[2] NS non-significant; + P<0.1; * P<0.05; ** P<0.01; *** P<0.001

8.13.4 Sire 15/98

Table 8.75 Details of non-significant QTL-sex interaction analysis results for sire 15/98 [1]

Trait	QTL-Sex Int., Signif of Peaks		Sex Int., Maximum Signif		Sex Int. Signif at QTL-Sex Int. Peak
	Position	$-\log_{10}p$	Position	P Value[2]	
Birth weight (kg)	6	0.4	56	ns	no
Scan weight (g)	122	0.2	110	ns	no
Slaughter weight (kg)	66	0.8	82	ns	no
Daily gain birth to weaning (g)	114	0.9	114	+	no
Daily gain weaning to scanning (g)	66	1.9	66	*	no
Ultrasound *M. longissimus* width (mm)	62	2.8	58	*	yes
Ultrasound *M. longissimus* depth (mm)	54	2.8	56	+	ns
Ultrasound *M. longissimus* area (mm^2)	60	3.6	58	*	yes
Ultrasound fat depth C (mm)	52	1.0	52	*	no
Carcass length (cm)	62	0.3	52	ns	no
Width of thorax (cm)	40	1.4	40	*	no
Width of forequarter (cm)	120	1.0	2	ns	no
Gigot width (cm)	66	2.1	66	ns	no
Dressing-out percentage	74	1.5	50	ns	no
Trimmed leg weight (g)	44	2.4	112	+	no
M. semimembranosus weight (g)	56	3.0	88	ns	ns
M. semitendinosus weight (g)	72	3.6	80	**	yes
M. biceps femoris weight (g)	52	2.3	66	*	no
M. quadriceps weight (g)	52	4.0	112	ns	ns
M. adductor weight (g)	46	2.3	50	+	no
M. gluteus medius weight (g)	66	0.8	74	+	no
Muscle trim weight (g)	52	2.8	112	ns	ns
Total muscle weight (g)	52	5.0	102	+	ns
Leg muscularity	62	2.8	116	+	ns
Leg muscle percent	52	4.1	52	ns	ns
Femur muscle to bone	52	3.0	44	ns	ns
Leg subcutaneous fat weight (g)	54	3.2	54	**	yes
Leg intermuscular fat weight (g)	66	0.9	82	ns	no
Leg total fat weight (g)	68	2.5	54	*	no
Leg fat percentage	54	2.1	54	*	no
M. longissimus weight (g)	66	2.9	54	+	ns
M. longissimus width (mm)	66	1.1	56	ns	no
M. longissimus width (mm)	58	0.5	32	ns	no
M. longissimus area (mm^2)	68	1.6	66	ns	no
GR (mm)	70	0.9	56	+	no
Fat depth C (mm)	54	3.4	54	**	yes
Loin subcutaneous fat weight (g)	104	0.3	112	ns	no
Pelvic bone weight (g)	42	0.6	40	ns	no
Femur bone weight (g)	32	0.4	32	ns	no
Total leg bone weight (g)	40	0.5	42	ns	no
pH *M. semimembranosus*	64	0.7	4	ns	no
Colour "L*" *M. semimembranosus*	66	0.4	84	ns	no
Colour "a*" *M. semimembranosus*	66	0.7	66	ns	no
Colour "b*" *M. semimembranosus*	52	0.4	20	ns	no
Sarcomere length *M. semi* (μm)	2	0.4	54	ns	no
Cooking loss *M. semi.* (%)	122	0.6	120	ns	no
Warner Bratzler peak force *M. semi.*	116	0.6	4	ns	no

Trait	QTL-Sex Int., Signif of Peaks		Sex In., Maximum Signif		Sex Int, Signif at QTL-Sex Int. Peak
	Position	$-\log_{10}p$	Position	P Value[2]	
Warner Bratzler yield force *M. semi.*	110	0.7	2	ns	no
pH *M. longissimus*	110	0.6	112	ns	no
Colour "L*" *M. longissimus*	122	0.2	110	ns	no
Sarcomere length *M. long.* (μm)	32	0.6	112	ns	no
Colour "a*" *M. longissimus*	50	0.4	50	ns	no
Colour "b*" *M. longissimus*	50	0.5	50	ns	no
Cooking loss *M. longissimus* (%)	106	1.4	108	*	no
Warner Bratzler peak force *M. long.*	122	1.2	120	+*	no
Warner Bratzler yield force *M. long.*	122	1.5	122	*	no
Warner Bratzler mean force *M. long.*	122	0.5	116	ns	no

[1] Firstly a test for a QTL – Sex Interaction QTL was carried out, then a test for the presence of a sex interaction. In order to report a QTL – Sex interaction, there must be the presence of a significant QTL peak, at which point the interaction is also significant
[2] NS non-significant; + P<0.1; * P<0.05; ** P<0.01; *** P<0.001

8.13.5 Sire 429/98

Table 8.76 Details of non-significant QTL-sex interaction analysis results for sire 429/98 [1]

Trait	QTL - Sex Int., Signif. of Peaks		Sex Int., Maximum Signif.		Sex Int., Signif. at QTL - Sex Int. Peak
	Position	$-\log_{10}P$	Position	P Value[2]	
Birth weight (kg)	0	0.9	0	+	no
Scan weight (g)	34	1.5	104	ns	no
Slaughter weight (kg)	110	0.7	0	ns	no
Daily gain birth to weaning (g)	46	2.8	106	ns	ns
Daily gain weaning to scanning (g)	50	0.6	40	ns	no
Ultrasound *M. longissimus* width (mm)	82	1.6	82	**	no
Ultrasound *M. longissimus* depth (mm)	80	1.0	80	*	no
Ultrasound *M. longissimus* area (mm^2)	82	1.6	82	**	no
Ultrasound fat depth C (mm)	62	1.2	102	ns	no
Carcass length (cm)	122	1.2	24	ns	no
Width of thorax (cm)	56	0.6	82	ns	no
Width of forequarter (cm)	48	1.1	60	ns	no
Gigot width (cm)	58	0.6	58	ns	no
Dressing-out percentage	116	1.4	122	ns	no
Trimmed leg weight (g)	54	0.7	54	ns	no
M. semimembranosus weight (g)	54	1.6	2	ns	no
M. semitendinosus weight (g)	54	1.5	112	*	no
M. biceps femoris weight (g)	54	1.3	122	*	no
M. quadriceps weight (g)	54	2.2	54	ns	no
M. adductor weight (g)	50	3.5	46	*	no
M. gluteus medius weight (g)	90	0.8	90	+	no
Muscle trim weight (g)	54	1.5	64	ns	no
Total muscle weight (g)	54	3.4	54	ns	ns
Leg muscularity	52	1.8	100	*	no
Leg muscle percent	52	4.2	84	ns	ns
Femur muscle to bone	54	3.7	104	ns	ns
Leg subcutaneous fat weight (g)	54	3.6	82	*	yes
Leg intermuscular fat weight (g)	48	1.1	38	ns	no
Leg total fat weight (g)	54	3.5	82	*	no
Leg fat percentage	52	2.7	82	+	ns

Trait	QTL - Sex Int., Signif. of Peaks		Sex Int., Maximum Signif.		Sex Int., Signif. at QTL - Sex Int. Peak
	Position	$-\log_{10}P$	Position	P Value[2]	
M. longissimus weight (g)	82	0.6	82	+	no
M. longissimus width (mm)	122	0.3	82	ns	no
M. longissimus width (mm)	52	0.5	52	ns	no
M. longissimus area (mm^2)	48	1.8	42	*	no
GR (mm)	56	2.5	104	+	no
Fat depth C (mm)	60	2.5	66	+	no
Loin subcutaneous fat weight (g)	56	3.1	106	*	no
Pelvic bone weight (g)	42	0.4	114	ns	no
Femur bone weight (g)	0	0.6	54	ns	no
Total leg bone weight (g)	8	0.6	54	ns	no
pH *M. semimembranosus*	122	1.6	2	ns	no
Colour "L*" *M. semimembranosus*	56	2.0	0	ns	no
Colour "a*" *M. semimembranosus*	112	2.0	66	ns	no
Colour "b*" *M. semimembranosus*	106	2.3	98	*	no
Sarcomere length *M. semi.* (μm)	70	1.7	68	**	no
Cooking loss *M. semi.* (%)	62	1.2	28	ns	no
Warner Bratzler peak force *M. semi.*	122	1.8	122	ns	no
Warner Bratzler yield force *M. semi.*	122	1.8	122	ns	no
pH *M. longissimus*	122	1.1	60	ns	no
Colour "L*" *M. longissimus*	56	1.4	2	ns	no
Sarcomere length *M. long.* (μm)	104	0.4	66	ns	no
Colour "a*" *M. longissimus*	122	1.8	58	ns	no
Colour "b*" *M. longissimus*	122	1.4	20	ns	no
Cooking loss *M. longissimus* (%)	100	0.7	104	ns	no
Warner Bratzler peak force *M. long.*	0	1.3	2	*	no
Warner Bratzler yield force *M. long.*	0	1.2	0	+*	no
Warner Bratzler mean force *M. long.*	0	1.6	0	**	no

[1] Firstly a test for a QTL – Sex Interaction QTL was carried out, then a test for the presence of a sex interaction. In order to report a QTL – Sex interaction, there must be the presence of a significant QTL peak, at which point the interaction is also significant
[2] NS non-significant; + P<0.1; * P<0.05; ** P<0.01; *** P<0.001

8.13.6 Sire 535/98

Table 8.77 Details of non-significant QTL-sex interaction analysis results for sire 535/98 [1]

Trait	QTL - Sex Int., Signif. of Peaks		Sex Int., Maximum Signif.		Sex Int., Signif. at QTL - Sex Int. Peak
	Position	$-\log_{10}P$	Position	P Value[2]	
Birth weight (kg)	82	0.8	110	ns	no
Scan weight (g)	8	0.3	16	ns	no
Slaughter weight (kg)	40	0.1	104	ns	no
Daily gain birth to weaning (g)	80	0.3	68	ns	no
Daily gain weaning to scanning (g)	116	0.4	60	ns	no
Ultrasound *M. longissimus* width (mm)	122	0.5	122	ns	no
Ultrasound *M. longissimus* depth (mm)	100	0.5	100	ns	no
Ultrasound *M. longissimus* area (mm^2)	104	0.2	104	ns	no
Ultrasound fat depth C (mm)	58	0.3	122	ns	no
Carcass length (cm)	58	0.9	122	ns	no
Width of thorax (cm)	122	1.5	66	ns	no
Width of forequarter (cm)	34	0.8	34	+	no

Trait	QTL - Sex Int., Signif. Position	Sex Int., of Peaks $-\log_{10}P$	Sex Int., Maximum Signif. Position	Sex Int., Signif. at QTL - Sex Int. Peak P Value[2]
Gigot width (cm)	104	0.5	104	ns no
Dressing-out percentage	98	1.5	98	** no
Trimmed leg weight (g)	34	0.4	2	ns no
M. semimembranosus weight (g)	82	0.2	82	ns no
M. semitendinosus weight (g)	66	0.9	66	* no
M. biceps femoris weight (g)	34	0.4	2	ns no
M. quadriceps weight (g)	34	0.3	2	ns no
M. adductor weight (g)	78	0.9	122	ns no
M. gluteus medius weight (g)	66	0.3	66	ns no
Muscle trim weight (g)	66	0.4	66	ns no
Total muscle weight (g)	34	0.2	66	ns no
Leg muscularity	64	0.2	82	ns no
Leg muscle percent	66	0.6	66	ns no
Femur muscle to bone	122	0.7	122	ns no
Leg subcutaneous fat weight (g)	62	1.6	2	ns no
Leg intermuscular fat weight (g)	122	0.2	122	ns no
Leg total fat weight (g)	62	1.0	2	ns no
Leg fat percentage	66	1.2	62	ns no
M. longissimus weight (g)	34	0.2	6	ns no
M. longissimus width (mm)	82	0.7	82	+ no
M. longissimus width (mm)	90	0.2	12	ns no
M. longissimus area (mm^2)	90	0.6	82	ns no
GR (mm)	94	1.1	122	ns no
Fat depth C (mm)	122	0.9	122	+ no
Loin subcutaneous fat weight (g)	34	0.4	122	ns no
Pelvic bone weight (g)	114	1.2	92	ns no
Femur bone weight (g)	122	0.3	122	ns no
Total leg bone weight (g)	122	0.9	122	ns no
pH *M. semimembranosus*	4	0.6	34	ns no
Colour "L*" *M. semimembranosus*	122	1.3	88	ns no
Colour "a*" *M. semimembranosus*	78	0.3	78	ns no
Colour "b*" *M. semimembranosus*	84	0.3	82	ns no
Sarcomere length *M. semi* (μm)	66	0.2	66	ns no
Cooking loss *M. semi.* (%)	122	0.5	66	ns no
Warner Bratzler peak force *M. semi.*	120	1.1	64	ns no
Warner Bratzler yield force *M. semi.*	122	1.1	64	ns no
pH *M. longissimus*	122	0.4	122	ns no
Colour "L*" *M. longissimus*	114	1.4	14	ns no
Sarcomere length *M. long.* (μm)	82	0.5	82	ns no
Colour "a*" *M. longissimus*	66	0.3	66	ns no
Colour "b*" *M. longissimus*	86	1.2	122	ns no
Cooking loss *M. longissimus* (%)	118	0.8	94	ns no
Warner Bratzler peak force *M. long.*	8	1.1	66	ns no
Warner Bratzler yield force *M. long.*	8	1.1	66	ns no
Warner Bratzler mean force *M. long.*	8	1.1	66	ns no

[1] Firstly a test for a QTL – Sex Interaction QTL was carried out, then a test for the presence of a sex interaction. In order to report a QTL – Sex interaction, there must be the presence of a significant QTL peak, at which point the interaction is also significant

[2] NS non-significant; + P<0.1; * P<0.05; ** P<0.01; *** P<0.001

8.14 Mutations Identified Through On-Line Mendelian Inheritance in Man (OMIM) as Impacting on Muscle or Fat

8.14.1 Muscle Mutations

Table 8.78 Mutations identified in Online Mendelian Inheritance in Man (OMIM) as impacting on muscle, but not specifically fitting the criteria of the candidate gene sought

Abbreviation	Name	Description	Position
AGL	Amylo-1,6-glucosidase	Mutations lead to glycogen storage disease III;	*1q21*
LMNA	Lamin	Muscular dystrophy	*1q21.2*
EDMD2	Emery-Dreifuss Muscular Dystrophy	Muscular dystrophy	*1q21.2*
TNNT2	Troponin T2, cardiac	Mutations cause cardiomyopathy	*1q32*
CAV3	Caveolin 3	Mutations cause rippling muscle	*1q41*
NEM1	Nemaline Myopathy 1	Nemaline myopathy	*1q42.1*
AGT	Angiotensin I	Increased blood pressure	*1q42-q43*
DYSF	Dysferlin	Mutation leads to Muscular Dystrophy, Limb-Girdle, Type 2B	*2p13.3*
DPP4	Dipeptidyl Peptidase IV	Combined immune deficiency	*2q23*
TTN	Titin	Mutations cause cardiomyopathy	*2q24.3*
NDUFS1	NADH-Ubiquinone Oxidoreductase Fe-S Protein 1	Lactic acidosis	*2q33-q34*
DES	Desmin	Myopathy and cardiomyopathy	*2q35*
IRS1	Insulin Receptor Substrate 1	Diabetes mellitus	*2q36*
MYL3	Myosin, light chain 3	Mutations cause cardiomyopathy	*3p*
MYL3	Myosin Light Chain 3; Alkali, Ventricular	Cardiac hypertrophy	*3p*
DAG1	Dystrophin-Associated Glycoprotein 1	Developmental abnormalities	*3p21*
ZLS	Zimmermann-Laband Syndrome	Fibromatosis and splenomegaly	*3p21.2*
SLC25A20	Solute Carrier Family 25 Member 20	Heart problems	*3p21.31*
CAV3	Caveolin	Muscular dystrophy	*3p25*
PPARG	Peroxisome Proliferator-Activated Receptor-Gamma	Promotes adipogenesis; knockouts lead to placental insufficiency	*3p25*
LGMD1C	Muscular Dystrophy, Limb-Girdle, Type 1C	Muscular dystrophy	*3p25*
PROMM	Proximal Myotonic Myopathy	Myopathy	*3q22*
BCL6	B-Cell Lymphoma	Lymphoma	*3q27*
CALNA	Calcineurin A	IGF1 stimulates glycolytic metabolism and skeletal muscle hypertrophy	*4*
PPARGC1	Peroxisome Proliferator-Activated Receptor-Gamma	Lipodystrophy; decreased subcutaneous fat	*4p15.1*
EVC2	Ellis-Van Creveld Syndrome Gene 2	Short-limbed, polydactyly	*4p16*
SGCB	Sarcoglycan Beta	Muscular dystrophy	*4q12*
LGMD2E	Muscular Dystrophy, Limb-Girdle, Type 2E	Muscular dystrophy	*4q12*
UCP1	Uncoupling Protein 1	Deficiency of brown fat	*4q31*
SLC25A4	Solute Carrier Family 25 Member 4	Hypertrophic cardiomyopathy	*4q35*
FSHMD1A	Facioscapulohumeral Muscular Dystrophy 1A	Fascioscapulohumeral muscular dystrophy	*4q35*
	ComplexI, Mitochondrial Respiratory Chain	Myopathy	*5q11.1*
TNXB	Tenascin-XB	Mutation to which lead to Ehler Danlos-Like Syndrome; hypertensible	*6p21.3*
MAPK14	Mitogen-Activated Protein Kinase 14	Differentiation of skeletal; knockout post-natally lethal	*6p21.3*

Abbreviation	Name	Description	Position
TRDN	Triadin	Cardiac hypertrophy	*6q22-q23*
PEX7	Peroxisome Biogenesis Factor 7	Rhizomelic chondrodysplasia	*6q22-q24*
MTPN	Myotrophin	Cardiac hypertrophy	*7q33-q35*
PRKAG2	Protein Kinase, AMP-Activated, Noncatalytic Gamma-2	Cardiomyopathy	*7q36*
PTP4A3	Protein-tyrosine Phosphatase Type 4A	Colorectal cancer	*8q24.3*
FRDA	Friedreich Ataxia 1	Cardiac dysfunction	*9q13*
LMX1B	LIM Homeo Box Transcription Factor 1	Nail-Petella syndrome	*9q34.1*
CHUK	Conserved Helix-Loop-Helix Ubiquitous Kinase	Abnormal limb development	*10q24*
CDKN1C	Cycling-dependent kinase inhibitor 1C	Beckwith-Wiedemann Syndrome	*11p15.5*
TNNT3	Troponin T3, Fast Skeletal	Arthrogryposis	*11p15.5*
MYBPC3	Myosin-binding protein C; cardiac	Mutations cause cardiomyopathy	*11q11.2*
EHD1	EH Domain-Containing 1	Involved with endocytosis of IGF1 receptors	*11q13*
MYL2	Myosin, Light Chain 2, Regulatory, Cardiac	Cardiomyopathy	*12q23*
NOS1	Nitric Oxide Synthase 1	Knock out related to differences in behaviour	*12q24.2-24.31*
IRS2	Insulin Receptor Substrate 2	Knockout leads to diabetes	*13q34*
MYH7	Myosin Heavy Chain 7; Cardiac Muscle Beta	Hypertrophic cardiomyopathy	*14q12*
MYH6	Myosin Heavy Chain 6; Cardiac Muscle Alpha	Hypertrophic cardiomyopathy	*14q12*
AKT1	V-AKT Murine Thymoma Viral Oncogene Homolog 1		*14q32.3*
CMD1A	Cardiomyopathy Dialated 1A	Cardiomyopathy	*15q14*
ACTC	Actin, Alpha, Cardiac Muscle	Cardiomyopathy	*15q14*
TPM1	Tropomyosin 1 (alpha)	Mutations cause cardiomyopathy	*15q22.1*
TPM1	Tropomyosin 1	Hypertrophic cardiomyopathy	*15q22.1*
IGF1R	Insulin-Like Growth Factor 1 Receptor	Knockouts have decreased growth	*15q25*
CTF1	Cardiotrophin 1	Cardiac hypertrophy	*16p11.2*
SLC2A4	Solute Carrier Family 2, Member 4	Stimulation of glucose uptake by insulin in muscle and adipose	*17p13*
FIMG	Myasthenia Gravis, Familial Infantile	Myasthenia gravis	*17p13*
SGCA	Sarcoglycan Alpha	Muscular dystrophy	*17q12*
ACE	Angiotensin 1 - Converting Enzyme	Blood pressure problems	*17q23*
SCN4A	Sodium Channel, Voltage-Gated Type IV Alpha Subunit	Hyperkalemia periodic paralysis and paramyotonia congenita	*17q23.1-q25.3*
HIP	Hyperkalemic Periodic Paralysis	Hyperkalemic Periodic Paralysis	*17q23.1*
GAA	Alpha- 1,4 - Glucosidase Gene	Mutations lead to glycogen storage disease II	*17q25.2-q25.3*
NFATC1	Nuclear Factor of Activated T Cells; Cytoplasmic, Calcineurin-Dependent 1		*18q23*
MAN2B1	Mannosidase, Alpha, Class 2B, Member1	Mental retardation	*19cen-q12*
INSR	Insulin Receptor	Mutations lead to insulin resistance	*19p13.2*
RR1	Ryanodine Receptor 1 Gene	Congenital myopathy	*19q13.1*
LGMD2I	Muscle Dystrophy Limb Girdle Type 2I	Deteriation of muscle tissue; calf muscle hypertrophy	*19q13.3*
FKRP	Fukutin-Related Protein	Congenital muscular dystrophy	*19q13.3*
TNNI3	Troponin I; cardiac	Mutations cause cardiomyopathy	*19q13.4*

Abbreviation	Name	Description	Position
TNNI3	Troponin I; cardiac	Hypertrophic cardiomyopathy	*19q13.4*
SNTA1	Syntrophin, Alpha-1	Muscular dystrophy	*20q11.2*
GNAS	GNAS Complex Locus	Pseudohypoparathyroidism	*20q13.2*
MYLK2	Myosin Light Chain Kinase 2	Cardiomyopathy	*20q13.3*
DSCR1	Down Syndrome Critical Region Gene Faciocutaneoskeletal syndrome	Cardiac hypertrophy Loose skin	*21q22.1* *22q13.1*
OMD	Muscular Dystrophy, Duchenne Type	Muscular dystrophy	*Xp21.2*
SHOX	Short Stature Homeo Box	Growth failure	*Xp22.32*
ITGB1BP2	Integrin, Beta-1, Binding Protein of.	Cardiac hypertrophy	*Xq12-q13*
GLA	Calactosidase Alpha	Cardiac hypertrophy	*Xq22*
LAMP2	Lysosome-Associated Membrane Protein 2	Mutations cause glycogen storage disease	*Xq24*
FMR1	Fragile site mental retardation 1 gene	Mental retardation	*Xq27.3*
MTTI	Transfer RNA, Mitochondrial, Isoleucine	Hypertrophic cardiomyopathy	
MTTG	Transfer RNA, Mitochondrial, Glycine	Hypertrophic cardiomyopathy	
MTTK	Transfer RNA, Mitochondrial, Lysine	Cardiomyopathy	

8.14.2 Fat Mutations

Table 8.79 Mutations identified in Online Mendelian Inheritance in Man (OMIM) as impacting on fat, but not specifically fitting the criteria of the candidate gene sought

Abbreviation	Name	Description	Position
FPLD	Familial Partial Lipodystrophy	Mutations lead to fat redistribution	*1q21.2*
NCOA1	Nuclear Receptor Coactivator 1	Knock-out mice are obese	*2p22.3*
PPARG	Peroxisome Proliferator - Activated Receptor Gamma	Mutations associated with obesity	*3p25*
APM1	Adipose most abundant gene transcript1	Mutations associated with obesity	*3q27*
PPARGC1	Peroxisome Proliferator-Activated Receptor-Gamma, Coactivator 1	Mutations associated with obesity	*4p15.1*
UCP1	Uncoupling Protein 1	Limited effects, made up for by UCP2	*4q31*
ESR1	Estrogen Receptor 1	Knock-out mice have hyperplasia and hypertrophy of adipose	*6q13.1*
DGAT1	Diacylglycerol O-Acyltransferase 1	Mutations lead to changes in milk fat content	*6q25.1*
PAI1	Plasminogen Activator Inhibitor 1	Mutations associated with obesity	*7q21.3*
ADRB2	Beta-2-Adrenergic Receptor	Mutations lead to obesity	*7q22*
LEP	Leptin	Mutations associated with obesity	*7q31.3*
ADRB3	Beta-3-Adrenergic Receptor	Knock-out mice are obese	*8p12*
LPL	Lipoprotein lipase	Mutations lead to increased circulating triglycerides	*8p22*
DBH	Dopamine Beta-Hydroxylase, Plasma	Knock-out mice have inability to produce brown fat	*9q34*
	Leprechaunism	Mutations lead to decreased fat and muscle	*19p13.2*
CEBPA	CCAAT/Enhancer-Binding Protein, Alpha	Mutations lead to defective development of fat	*19q13.1*
AHO	Albright Hereditary Osteodystrophy	Mutations lead to resistance to parathyroid hormone, and obesity	*20q13.2*
PCK1	Phosphoenolypyruvate Carboxykinase 1	Is the main target for regulation of gluconeogenesis	*20q13.31*

8.15 Details of Genes Identified within Region of Interest on Human Chromosome 2

Table 8.80 List of genes identified using USCS Genome Bioinformatics (June 2002) that map to human Chromosome 2, 160 –190 mega base pairs

Name	Abs.[1]	Appr.[2]	Full Name	Other Names[3]				
TANK	*347*	appr.	TRAF- family member-associated NFKB activator	I-TRAF	ITRAF	TRAF2		
POH1	*8*	not ap.	26S proteasome-associated pad1 homolog	PAD1				
TBR1	*38*	appr.	T-Box, Brain, 1	T-box, Brain,1	Transcriptional regulator, T-brain-1			
SLC4A10	*1*	appr.	Solute Carrier Family 4, Sodium Bicarbonate Transporter-Like, Member 10					
GCG	*382*	appr.	glucagon					
FAP	*35*	appr.	Fibroblast Activation Protein, Alpha	FAPA	Seprase			
MDA5	*1*	not ap.	Melanoma Differentiation Associated Protein-5					
GCA	*17*	appr.	grancalcin, EF-hand calcium binding protein	GCL				
KCNH7		appr.	Potassium Voltage-Gated Channel, Subfamily H (eag-related), Member 7	HERG3	KV11.3			
FIGN	*3*	appr.	Fidgetin					
GRB14	*21*	appr.	growth factor receptor-bound protein 14					
KIAA0877		not ap.						
SCN3A	*6*	appr.	Sodium Channel, Voltage-gated, type III, alpha polypeptide	NAC3				
SCN2A2	*1*	appr.	Sodium Channel, Voltage-gated, type II, alpha 2 polypeptide	HBSCII				
	8	appr.	UDP-N-Acetyl-Alpha-D-Galactosamine: polypeptide N-	GalNAc-T3				
FLJ11457		not ap.						
SCN1A	*39*	appr.	Sodium Channel, Voltage-gated, type1, alpha polypeptide	GEFSP2	NAC1	SCN1		
SCN9A	*3*	appr.	Sodium Channel, Voltage-gated, type IX, alpha polypeptide	NE-NA	PN1			
SCN7A	*2*	appr.	Sodium Channel, Voltage-gated, type VII, alpha polypeptide	SCN61				
B3GALT1	*2*	appr.	UDP-Gal:betaGlcNAc beta, 1,3-galactosyltransferase, polypeptide 1	BETA3GAL-T1				
STK39	*6*	appr.	Serine theronine kinase 39	DCHT	SPAK			
MGC20702		not ap.						
AD024		not ap.						
IGRP	*5*	not ap.	Islet-specific glucose-6-phosphate catalytic subunit-related protein					
ABCB11	*113*	appr.	ATP-binding cassette, sub-family b (MDR/TAP), member 11	ABC16	BSEP	PFIC2	PFIC-2	PGY4
RDHL	*15*	not ap.	NADP-dependent retinol dehydrogenase/reductase					
LRP2	*4*	appr.	Low density lipoprotein-related protein 2					
SARCOSIN	*8*	not ap.	Sarcomeric muscle protein					
FLJ21901		not ap.						
PPIG	*3*	appr.	Peptidyl-prolyl isomerase G (cyclophilin G)	CARS-Cyp	Cyp	SRCyp		

Name	Abs.[1]	Appr.[2]	Full Name	Other Names[3]			
MGC22679		not ap.					
SSB	*204*	appr.	Sjogren syndrome antigen B (autoantigen La)				
HSPC133		not ap.	Myelin associated glycoprotein				
MYO3B	*1*	appr.	Myosin III B				
GAD1	*19*	appr.	Glutamate Decarboyxlase 1	GAD			
GORASP2	*8*	appr.	Golgi reassembly stacking protein 2	GOLPH6	GRASP55	GRS2	
TLK1	*2*	appr.	Tousled-like kinase 1	KIAA0137	PKU-Beta		
PRO2037		not ap.					
FLJ13984		not ap.					
FLJ13096		not ap.					
FLJ23462		not ap.					
SLC25A12	*3*	appr.	Solute carrier family 25 (mitochondrial carrier, Aralar), member 12	ARALAR	ARALAR1		
HAT1	*46*	appr.	Histone acetyltransferase1				
DLX2	*9*	appr.	Distal-less homeo box 2	TES-1	TES1		
ITGA6	*8*	appr.	Integrin, alpha 6				
PDK1	*15*	appr.	Pyruvate dehydrogenase kinase, isoenzyme1				
CAMP-GEFII	*6*						
ZAK	*6*	not ap.	sterile alpha motif and leucine zipper containing kinase AZK				
CDCA7	*1*	appr.	cell division cycle associated 7	FLJ14736	JPO1		
PTD004	*4*	not ap.					
CIR	*4*	not ap.	CBF1 interacting co-repressor				
FLJ23142		not ap.					
WASPIP	*20*	appr.	Wiskott-Aldrich syndrome protein interacting protein	WIP			
CHRNA1	*4*	appr.	cholinergic receptor, nicotinic, alpha polypeptide 1	ACHRA	CHNRA	CHRNA	
CHN1		appr.	chermerin	ARHGAP2	CHN	RHOGAP2	n-chirmerin
ATF2	*183*	appr.	activating transcription factor 2	CRE-BP1	BREB2	CREBP1	TREB7
ATP5G3	*2*	appr.	ATP synthase, H+ transporting, mitochondrial F0 complex, subunit c				
HOXD13	*62*	appr.	Homeobox D13	HOX4I	SPD		
HOXD12	*11*	appr.	Homeobox D12	HOX4H			
HOXD11	*21*	appr.	Homeobox D11	HOX4	HOX4F		
HOXD10	*39*	appr.	Homeobox D10	HOX4	HOX4D		
HOXD9	*29*	appr.	Homeobox D9	HOX4	HOX4C		
HOXD8	*18*	appr.	Homeobox D8	HOX4	HOX4E		
HOXD4	*33*	appr.	Homeobox D4	HOX4	HOX4B		

Name	Abs.[1]	Appr.[2]	Full Name	Other Names[3]				
HOXD3	*20*	appr.	Homeobox D3	HOX4	HOX4A			
HOXD1	*16*	appr.	Homeobox D1	HOX4	HOX4G			
MTX2	*14*	appr.	metaxin2					
NFE2L2	*2*	appr.	nuclear factor (erythroid-derived 2)- like 2	NRF2				
AGPS		appr.	alkylglycerone phosphate synthase	ADAP-S	ADAPS	ADHAP-PEN	ADHAP	ADAS
PDE11A	*11*	appr.	phosphodiesterase 11A					
OSBPL6	*2*	appr.	oxysterol binding protein like-6	ORP-6	ORP6			
PRKRA	*16*	appr.	protein kinase, interferon-inducible double stranded RNA dependent activator	PACT	RAX			
PLEKHA3	*1*	appr.	pleckstrin homology domain containing, family A (phosphoinositide binding	FAPP1				
TTN	*10*	appr.	Titin	CMD1G				
UBE2E3	*6*	appr.	ubiquitin-conjuugatin enzyme E2E3	UBCH9				
ITGA4	*141*	appr.	integrin, alpha 4 (antigen CD49D, alpha 4 subunit of VLA-4 receptor)	CD49D				
NEUROD1	*70*	appr.	neurogenic differentiation 1	BETA2	BHF-1	NEUROD	NIDDM	
SSFA2	*6*	appr.	sperm specific antigen 2	CS-1	CS1	KIAA1927	SPAG13	
PDE1A	*14*	appr.	phosphodiesterase 1A, calmodulin-dependent					
FRZB	*65*	appr.	frizzled-related protein	FRE	FRP-3	FRZB-PEN	FRZB1	FRITZ
NCKAP1	*48*	appr.	NCK-associated protein 1	HEM2	KIAA0587	NAP1		
SKRP1		not ap.						
HT010		not ap.						
ITGAV	*2*	appr.	integrin, alpha V (vitronectin receptor, alpha polypeptide, antigen CD51)					
CALCRL	*94*	appr.	calcitonin receptor-like	CGRPR	CGRPR1	CGRPR1-PEN	CRLR	
TFPI	*735*	appr.	tissue factor pathway inhibitor (lipoprotein-associated coagulation inhibitor)	LAC1	TFPI1			
CED-6	*13*	not ap.						
DIRC1	*1*	appr.	disrupted in renal carcinoma 1					
COL3A1	*116*	appr.	collagen, type III, alpha 1 (Ehlers-Danlos syndrome type IV, autosomal					
SLC11A3	*77*	appr.	solute carrier family 11 (proton coupled divalent metal ion transporters),	FPN1	IREG1	MTP1		
PMS1	*161*	appr.	postmeiotic segregation increased 1 (S. cerevisiae)	HNPCC3	PMSL1			
GDF8	*29*	appr.	growth differentiation factor 8	MSTN				
HIBCH		appr.	3-hydroxyisobutyryl-coenzyme A hydrolase					
INPP1	*8*	appr.	inositol polyphosphate-1-phosphatase					
NAB1	*17*	appr.	NGFI-A binding protein (EGR1 binding protein 1)					
GLS	*4*	appr.	glutaminase	KIAA0838				

[1] The number of abstracts identified relating to this gene, using PubMed (Jan 2003)
[2] Whether or not the name of the gene has been approved by the UCL/HGNC/HUGO Human Gene Nomenclature committee
[3] Alternative name for the gene identified in GeneCards™ website

Table 8.81 List of genes identified using USCS Genome Bioinformatics (June 2002) that map to human Chromosome 2, 160 –190 mega base pairs

Name	Cyto.[1]	Position (Base Pairs)[1]	Description[2]
TANK	*2q24.2*	*160,047,187-160,146,403*	Acts as a regulator of TRAF function by maintaining them in a latent state. Over-expression inhibits TRAF2-Mediated NF-Kappa-B activation signalled by CD40, TNFR1 and TNFR2. Blocks TRAF2 binding to LMP1 and inhibits LMP1-mediated NF-Kappa-B activation. Interacts with TRAF1, TRAF2, and TRAF3 by binding to their TRAF-C domains. Interacts more strongly with TRAF1 and TRAF2 than TRAF3
POH1	*2q24.2*	*160,218,270-160,321,947*	Component of the 26S proteasome, a multiprotein complex that degrades proteins targeted for destruction by the ubiquitin pathway
TBR1	*2q24.2*	*160,326,619-160,335,294*	Probable transcriptional regulator involved in developmental processes. TBR1 is required for normal brain development
SLC4A10	*2q24.2*	*160,534,667-160,895,482*	-
GCG	*2q24.2*	*161,053,109-161,062,478*	Glucagon promotes hydrolysis of glycogen and lipids, and raises the blood sugar level. GLP2 stimulates intestinal growth and unregulated villus height in the small intestine, concomitant with increased crypt cell proliferation and decreased enterocyte apoptosis. Produced in the A cells of the islets of Langerhans in response to a drop in blood sugar concentration.
FAP	*2q24.2*	*151,080,916-161,153,766*	May have a role in tissue remodelling during development and wound healing, may contribute to invasiveness in malignant cancers. Degrades gelatine and heat-denatured type I and type IV collagen but not native type I or type IV collagen. Does not cleave laminin, fibronectin, fibrin or casein.
MDA5	*2q24.2*	*161,177,312-161,228,706*	MDA5 is a putative RNA helicase that is upregulated in response to treatment with IFNB or IFNB and MEZ
GCA	*2q24.2*	*161,254,369-161,271,350*	May play a role in granule-membrane fusion and degranulation
KCNH7	*2q24.2*	*161,281,643-151,750,340*	Belongs to the ERG subfamily of voltage-gated potassium channels
FIGN	*2q24.3*	*162,519,327-162,523,183*	Is a AAA proteins are molecular chaperones that facilitate a variety of functions, including membrane fusion, proteolysis, peroxisome biogenesis, endosome sorting, and meiotic spindle formation
GRB14	*2q24.3*	*163,404,527-163,533,552*	Interacts with the cytoplasmic domain of the autophosphorlyated insulin receptor which is then inhibited. The interaction is mediated by the SH2 domain (by similarity)
SCN3A	*2q24.3*	*163,999,234-153,115,747*	This protein mediates the voltage-dependent sodium ion permeability of excitable membranes. Assuming opened or closed conformations in response to the voltage differences across the membrane, the protein forms a sodium-selective channel through which NA+ ions may pass in accordance with their electrochemical gradient.
SCN2A2	*2q24.3*	*164,151,149-164,301,733*	-
GALNT3	*2q24.3*	*164,658,516-164,682,402*	Is one of several enzymes that catalyze a reaction that's initiates O-glycosylation of serine and threonine residues on an array of glycoproteins
SCN1A	*2q24.3*	*163,900,863-164,985,341*	This protein mediates the voltage-dependent sodium ion permeability of excitable membranes. Assuming opened or closed conformations in response to the voltage differences across the membrane, the protein forms a sodium-selective channel through which NA+ ions may pass in accordance with their electrochemical gradient.

Name	Cyto.[1]	Position (Base Pairs)[1]	Description[2]
SCN9A	*2q24.3*	*165,109,999-165,223,506*	Voltage-dependent sodium channels are responsible for the initial membrane depolarization that occurs during generation of action potentials in most electrically excitable cells
SCN7A	*2q24.3*	*165,317,081-165,398,673*	Voltage-dependent sodium channels are responsible for the initial membrane depolarization that occurs during generation of action potentials in most electrically excitable cells
B3GALT1	*2q24.3*	*166,727,261-166,779,445*	-
STK39	*2q24.3*	*166,862,610-167,156,257*	May act as a mediator of stress-activated signals
IGRP	*2q31.1*	*167,809,907-167,816,654*	Similar to insulin and islet amyloid polypeptide, it is expressed in a highly pancreatic beta-cell manner
ABCB11	*2q31.1*	*167,831,513-167,939,902*	Involved in the ATP-dependent secretion of bile salts into the canaliculus of hepatocytes
RDHL	*2q31.1*	*167,990,154-168,004,746*	
LRP2	*2q31.1*	*168,036,929-168,271,082*	Receptor-mediated uptake of polybasic drugs such as aprotinin, aminoglycosides and polymyxinb. May participate in regulation of parathyroid hormone related protein release
SARCOSIN	*2q31.1*	*168,418,281-168,434,834*	-
PPIG	*2q31.1*	*168,492,850-168,546,151*	-
SSB	*2q31.1*	*168,707,286-168,720,472*	LA protein plays a role in the transcription of RNA polymerase III. It is most probably a transcription termination factor. Binds to the 3' termini of virtually all nascent polymerase III transcripts. It is associated with precursor forms of RNA polymerase II transcripts including TRNA and 4.5S, 5S,7S and 7-2RNAs
MYO3B	*2q31.1*	*169,086,551-169,562,886*	-
GAD1	*2q31.1*	*169,724,967-169,769,556*	Catalyses the production of GABA
GORASP2	*2q31.1*	*169,837,646-169,875,533*	-
TLK1	*2q31.1*	*169,900,465-170,068,986*	-
SLC25A12	*2q31.1*	*170,692,762-170,802,627*	Calcium-dependent mitochondrial solute carried, may have a function in the urea cycle. High levels in heart and skeletal muscle, low in brain and very low in kidney. Binds calcium
HAT1	*2q31.1*	*170,830,759-170,901,081*	May play a role in telomeric silencing. Acetylates soluble but not nucleosomal H4 at LYS-5 and LYS-12 and acetylates histone H2a at LYS-5. HAT1 has intrinsic substrate specificity that modifies lysine in recognition sequence GXGKXG.
DLX2	*2q31.1*	*171,016,710-171,019,809*	Likely to play a regulatory role in the development of the ventral forebrain. May play a role in craniofacial patterning and morphogenesis. Belongs to the distal-less homeobox family
ITGA6	*2q31.1*	*171,344,860-171,423,553*	Integrin alpha-6/beta1 is a receptor for laminin on platelets. Integrin alpha-6/beta-4 is a receptor for laminin in epithelial cells and it plays a critical structural role in the hemidesmosome.
PDK1	*2q31.1*	*171,473,371-171,513,381*	Inhibits the mitochondrial pyruvate dehydrogenase complex by phosphorylation of the E1 alpha subunit, thus contributing to the regulation of glucose metabolism

Name	Cyto.[1]	Position (Base Pairs)[1]	Description[2]
CDCA7	*2q31.1*	*172,272,123-172,286,262*	-
CIR	*2q31.1*	*173,265,421-173,312,940*	Member of the CSL family of DNA-binding factors, which mediate transcriptional activation or repression
WASPIP	*2q31.1*	*173,479,452-173,551,850*	May have direct activity on the actin cytoskeleton. Induces actin polymerisation and redistribution
CHRNA1	*2q31.1*	*173,665,149-173,681,726*	After binding acetylcholine, the ACHR responds by an extensive change in conformation that affects all subunits and lead to opening of an ion-conducting channel across the plasma membrane
CHN1	*2q31.1*	*173,991,775-174,085,442*	A brain GTPase-activating protein (GAP) for the RAS-related p21
ATF2	*2q31.1*	*173,991,775-174,085,442*	This protein binds the camp response element. A sequence present in many viral and cellular promoters
ATP5G3	*2q31.1*	*174,095,418-174,098,907*	This protein is one of the chains of the nonenzymatic membrane component of mitochondrial ATPase
HOXD13	*2q31.1*	*175,010,069-175,012,023*	Sequence-specific transcription factor which is part of a developmental regulatory system that provides cells with specific positional identifies on the anterior-posterior axis
HOXD12	*2q31.1*	*175,017,067-175,018,025*	Sequence-specific transcription factor which is part of a developmental regulatory system that provides cells with specific positional identifies on the anterior-posterior axis
HOXD11	*2q31.1*	*175,024,621-175,026,407*	Sequence-specific transcription factor which is part of a developmental regulatory system that provides cells with specific positional identifies on the anterior-posterior axis
HOXD10	*2q31.1*	*175,034,067-175,036,565*	Sequence-specific transcription factor which is part of a developmental regulatory system that provides cells with specific positional identifies on the anterior-posterior axis
HOXD9	*2q31.1*	*175,040,028-175,041,509*	Sequence-specific transcription factor which is part of a developmental regulatory system that provides cells with specific positional identifies on the anterior-posterior axis
HOXD8	*2q31.1*	*175,047,632-175,048,877*	Sequence-specific transcription factor which is part of a developmental regulatory system that provides cells with specific positional identifies on the anterior-posterior axis
HOXD4	*2q31.1*	*175,067,655-175,073,335*	Sequence-specific transcription factor which is part of a developmental regulatory system that provides cells with specific positional identifies on the anterior-posterior axis
HOXD3	*2q31.1*	*175,081,342-175,090,363*	Sequence-specific transcription factor which is part of a developmental regulatory system that provides cells with specific positional identifies on the anterior-posterior axis
HOXD1	*2q31.1*	*172,105,844-175,108,172*	Sequence-specific transcription factor which is part of a developmental regulatory system that provides cells with specific positional identifies on the anterior-posterior axis
MTX2	*2q31.1*	*175,196,700-175,262,205*	Involved in the transport of proteins into the mitochondrion
NFE2L2	*2q31.2*	*176,157,573-176,191,954*	May be involved in the transcriptional activation of genes of the beta-globin cluster
AGPS	*2q31.2*	*176,320,040-176,465,452*	Associated with ether lipid biosynthesis

Name	Cyto.[1]	Position (Base Pairs)[1]	Description[2]
PDE11A	*2q31.2*	*176,055,349-177,031,727*	Part of a superfamily of enzymes that catalyze the hydrolysis of 3-prime,5-prime-cyclic nucleotides
OSBPL6	*2q31.2*	*177,121,913-177,322,984*	From family of intracellular lipid receptors
PRKRA		*177,358,687-177,378,401*	Mediator of the effects of interferon
PLEKHA3	*2q31.2*	*177,407,897-177,432,326*	-
TTN	*2q31.2*	*177,453,253-177,734,685*	An abundant protein of striated muscle
UBE2E3	*2q31.3*	*180,033,949-180,135,688*	Involved with Ubiquitination of a protein substrate
ITGA4	*2q31.3*	*180,528,751-180,608,454*	Is a cell surface receptors for extracellular matrix components, as well as a receptor for aspects of leukocyte adhesion
NEUROD1	*2q31.3*	*180,748,735-180,752,794*	Acts as a differentiation factor during neurogenesis. Transcriptional activator. Binds to the insulin gene e-box
SSFA2	*2q32.1*	*180,990,798-181,002,463*	Sperm surface antigen involved in some step or early cleavage of the fertilized oocyte
PDE1A	*2q32.1*	*181,214,553-181,594,515*	Has a higher affinity for CGMP than for CAMP
FRZB	*2q32.1*	*181,906,497-181,938,695*	May be involved in morphogenesis of skeleton. May also act as a soluble WNT-binding protein
NCKAP1	*2q32.1*	*181,996,827-182,110,309*	Associates preferentially with the first SH3 domain of NCK
SKRP1			-
HT010			-
ITGAV	*2q32.1*	*185,662,236-185,751,759*	The alpha-V integrins are receptors for vitronectin, cytotactin, fibronectin, fibrinogen, laminin, matrix metalloproteinases-2, osteopontin, osteomodulin, prothrombin, thromobospoin and von Willebrand factor.
CALCRL	*2q32.1*	*186,419,844-186,522,990*	This is a receptor for calcitonin gene-related peptide type 1.
TFPI	*2q32.1*	*186,541,675-186,629,182*	Inhibits factor X (X(A)) directly and, in a XA-dependent way, inhibits VII(A)/Tissue factor activity
CED-6	*2q32.2*	*187,569,884-187,596,606*	An evolutionarily conserved adaptor protein required for efficient engulfment of apoptotic cells by phagocytes
DIRC1	*2q32.2*	*187,734,413-187,790,779*	Mutations associated with familial clear cell renal cancer
COL3A1	*2q32.2*	*187,975,046-188,013,419*	Collagen Type III occurs in most soft connective tissues along with Type I Collagen.
SLC11A3	*2q32.2*	*188,599,557-188,619,733*	Involved with iron absorption
PMS1	*2q32.2*	*188,823,509-188,926,589*	Probably involved in the repair of mismatches in DNA
GDF8	*2q32.2*	*189,114,672-189,121,704*	Acts specifically as a negative regulator of skeletal muscle growth
HIBCH	*2q32.2*	*189,263,993-189,378,797*	-
INPP1	*2q32.2*	*189,402,684-189,430,556*	-
NAB1	*2q32.2*	*189,718,131-189,751,739*	Acts as a transcriptional repressor for zinc finger transcription factors EGR1 and EGR2
GLS	*2q32.2*	*189,939,808-190,024,519*	Catalyses the first reaction in the primary pathway for renal catabolism of glutamine

[1] Positions were taken from USCS Genome Bioinformatic Website
[2] Descriptions were taken from GeneCards™ Website and OMIM; - indicates that no description was available from theses sources

8.16 Details of New Genes Mapped to Region of Interest Since Initial Search Carried Out

Table 8.82 List of new genes identified using USCS Genome Bioinformatics (July 2003) that map to human Chromosome 2, 160 –190 mega base pairs

Name	Abs.[1]	Appr.[2]	Full Name	Other Names[3]					
DPP4	13	appr.	Dipeptidylpeptidase 4	ADABP	ADCP2	CD26 DPPIV			
TAIP-2	0	not ap.	TGF-beta induced apotosis protein 2						
CMYA3	0	appr.	cardiomyopathy associated 3						
NOSTRIN	1	appr.	nitric oxide synthase trafficker						
CYBRD1	1	appr.	cytochrome b reductase 1	DCYTB					
DNCI2	1	appr.	dynein, cytoplasmic, intermediate polypeptide 2	IC2					
DLX1	43	appr.	Distal-less homeobox 1						
SP3	1004	appr.	Sp3 transcription factor	SPR-2					
LNP	1	not ap.	Lunapark						
FKPB7	1	appr.	FK506 binding protein 7						
ERDJ5	1	not ap.	ER-resident protein ERdj5						
COL5A2	33	appr.	Collagen, type-V, Alpha2	AB collagen					
SLC40A1	2	appr.	Solute carrier 40 (iron regulated transporter), member 1	FPN1	HFE3	IREG1	MTP1	SLC11AE	FERROPORTIN1
NS3TP1	0	not ap.	HCV NS3-transactivated protein 1						
OSGEPL1	0	appr.	O-sialoglycoprotein endopeptidase like 1	O-sialoglycoprotein endopeptidase like 1			Putative sialoglycoprotease type 2		

[1] The number of abstracts identified relating to this gene, using PubMed (Jan 2003)
[2] Whether or not the name of the gene has been approved by the UCL/HGNC/HUGO Human Gene Nomenclature committee
[3] Alternative name for the gene identified in GeneCards™ website

Table 8.83 List of new genes identified using USCS Genome Bioinformatics (July 2003) that map to human Chromosome 2, 160 –190 mega base pairs

Name	Cyto.[1]	Position (Base Pairs)[1]	Description[2]
DPP4	2q24.3	162,812,783-162,895,075	Involved in endocrine and immune functions
TAIP-2	2q24.3	166,392,873-166,501,241	May play a role in apoptosis (programmed cell death)
CMYA3	2q31.1	167,723,999 - 168,080,289	Involved with cardio myopathy
NOSTRIN	2q31.1	169,607,073 - 169,685,588	Contributes to the protein network controlling activity, trafficking, and targeting of eNOS
CYBRD1	2q31.1	172,342,882 - 172,378,663	Encodes key iron transport proteins,
DNC12	2q31.2	172507967- 172,568,950	Mediates movement of many intracellular organelles
DLX1	2q32	172,913,493 - 172,918,429	Likely to play a developmental role in the development of the ventral forebrain
SP3	2q31	174,737,282 - 174,793,276	Transcription factor activator
LNP	2q31.1	176,754,434 - 176,754,434	Shares limb and CNS expression specificities with both Hoxd gene
FKPB7	2q31.3	179,294,045 - 179,307,346	Accelerates the folding of proteins during protein synthesis
ERDJ5	2q32.1	183,544,821 - 183,608,355	Roles for ERdj5 in protein folding and translocation across the ER membrane.
COL5A2	2q32.2	189,861,263 - 190,008,511	It is a minor connective tissue component of nearly ubiquitous distribution. Type V Collagen
SLC40A1	2q32	190,389,339 - 190,409,636	Main Export Iron Protein
NS3TP1	2q32.2	190,490,169 - 190,500,084	-
OSGEPL1	2q32.3	190,575,409 - 190,591,976	-

[1] Positions were taken from USCS Genome Bioinformatic Website
[2] Descriptions were taken from GeneCards™ Website