New mastitis phenotypes suitable for genomic selection in meat sheep and their genetic relationships with udder conformation and lamb live weights.

A. McLaren[1], K. Kaseja[2], J. Yates[3], S. Mucha[2], N.R. Lambe[2] & J. Conington[2]

[1]*Scotland's Rural College, Future Farming Systems, Hill & Mountain Research Centre, Crianlarich, FK20 8RU, United Kingdom*

[2]*Scotland's Rural College, Animal and Veterinary Services, Easter Bush, Midlothian, EH25 9RG, United Kingdom*

[3]*The British Texel Sheep Society, Stoneleigh Park, Kenilworth, Warwickshire, CV8 2LG, United Kingdom*

Corresponding author: Ann McLaren. Email: Ann.McLaren@sruc.ac.uk

Short title: Breeding for mastitis resistance in meat sheep

Abstract

Mastitis can prove expensive in sheep reared for meat production due to costs associated with treatment methods, poor lamb growth and premature culling of ewes. The most commonly used method to detect mastitis, in dairy systems, is somatic cell counts. However, in many meat-producing sheep flocks ewes are not routinely handled, thus regular milk sampling is not always possible. It is therefore worthwhile to investigate alternative phenotypes, such as those associated with udder conformation and methods of evaluating somatic cell counts in the milk, such as the California Mastitis Test. The main objectives of this study were therefore, a) to estimate genetic parameters of traits relating to mastitis and udder conformation in a meat sheep breed; b) estimate the level of association between somatic cell counts

and the California Mastitis Test and c) assess the relationships between mastitis and both udder conformation and lamb live weights. Data were collected from Texel ewes based on 29 flocks, throughout the UK, during 2015 and 2016. The ewes were scored twice each year, at mid- and late-lactation. Eight different conformation traits, relating to udder and teat characteristics, and milk samples were recorded. The data set comprised of data available for 2 957 ewes. The pedigree file used contained sire and dam information for 31 775 individuals. The animal models used fitted relevant fixed and random effects. Heritability estimates for traits relating to mastitis (somatic cell score and the California Mastitis Test), ranged from 0.08 to 0.11 and 0.07 to 0.11 respectively. High genetic correlations were observed between somatic cell score and the California Mastitis Test (0.76 to 0.98), indicating the California Mastitis Test to be worthwhile for assessing infection levels, particularly at mid-lactation. The strongest correlations observed between the mastitis traits and the udder conformation traits were associated with udder depth (0.61 to 0.75) also at mid-lactation. Moderately negative correlations were also observed between the mastitis traits and teat angle, with those estimated at mid-lactation ranging from -0.41 to -0.55. Negative phenotypic correlations were estimated between mastitis and the weight of lamb reared by the ewe (-0.15 to -0.23), suggesting that lamb weights fell as infection levels rose. Genetic correlations were not significantly different from zero. Reducing mastitis will lead to improvements in flock productivity and the health and welfare of the animals. It will also improve the efficiency of production and the resilience to disease challenge. The economic benefits, therefore, of these results combined could be substantial not only in this breed but also in the overall meat sheep industry.

Keywords: Mastitis, Sheep, Genetic Selection, Somatic Cell Counts, California Mastitis Test

Implications

This research presents the first estimates of the genetic basis to mastitis in sheep reared primarily for meat production, based on somatic cell counts and the California Mastitis Test. It also highlights relationships between a variety conformation traits particularly the depth of the udder and teat shape and angle. The use of the California Mastitis Test tool has been proven to be a good predictor of somatic cell count. Its use as a breeding and management tool for sheep farmers to manage mastitis in their flocks will improve animal health and welfare, improve productivity and increase revenue from lamb sales.

Introduction

Mastitis is often regarded as one of the most important health problems in dairy ruminants, but it can also have a large impact on ruminants reared for meat production. The nature of the disease is complex, involving both genetic and environmental factors. The main causative bacteria associated with the disease, in sheep, are *Staphylococcus aureus* and *Mannheimia* species (Bergonier and Berthelot, 2003; Gelasakis *et al.* 2015). Clinical forms of the disease can result in swelling and pain in the udder, changes to milk appearance and composition, high temperature, lameness on the side affected and in extreme cases even death. Subclinical forms of the disease are often less visible in terms of changes to the

udder or milk, but can be diagnosed using specific tests such as identifying the levels of somatic cells in the milk, as a response to the presence of infection (Fragkou *et al.* 2014).

In addition to animal welfare concerns, the disease can prove expensive in meat producing flocks due to the costs associated with treatment, poor lamb growth and premature culling of ewes (Bergonier and Berthelot, 2003; Gelasakis *et al.* 2015). In dairy sheep, Rupp and Foucras (2010) estimated, based on an assumed 10% incidence of mastitis in EU dairy sheep and goat flocks, the total annual milk production losses could be in the region of €60 million per annum. In a meat producing sheep breed such as the Texel, Conington *et al.* (2008) estimated that a 10% reduction in the risk of contracting mastitis would be worth £8.40 per ewe, or £2.7 million a year to the purebred UK Texel population at that time. The economic and welfare benefits of reducing the impact that mastitis can have on the sheep industry, both in the UK and globally, are therefore likely to be significant.

The genetic basis for mastitis resistance was initially observed in dairy cattle, with a number of studies accumulating evidence based on disease related phenotypes such as the presence of clinical mastitis or somatic cell counts (SCC) in the milk (Mrode and Swanson, 1996). The levels of somatic cells in the milk reflect the degree to which an immune response has begun against infection such as those caused by bacteria associated with mastitis. More recently similar studies in sheep, predominantly relating to dairy sheep, have also observed a genetic component to the disease and the bacterial pathogens relating to the disease (Bergonier and Berthelot, 2003; Rupp *et al.*, 2009; Riggio and Portolano, 2015). The most commonly used method to detect mastitis in dairy animals are SCC as they can be routinely

collected, often have a higher heritability than clinical mastitis and can be an indicator for both clinical and subclinical infections (Tolone *et al*., 2013, Riggio and Portolano, 2015).

However, in many meat-producing flocks the ewes are not routinely handled, thus regular milk sampling is not always practical. It is therefore worthwhile to investigate alternative phenotypes in order to gauge the level of infection. A number of studies, in both dairy cattle and small ruminants, have investigated the associations between mastitis and udder and teat conformation traits (Rupp and Boichard, 2003; Legarra and Ugarte, 2005). Additionally, alternative methods of evaluating cell counts in the milk may prove useful, such as the California Mastitis Test (CMT). This is a quick, simple and inexpensive method of scoring a small sample of milk based upon the reaction there is with a reagent; the level of reaction being proportional to the concentration of somatic cells present (Gonzalez-Rodriguez and Carmenes, 1996; Bergonier and Berthelot, 2003).

The identification of suitable phenotypes also allows the future use of genomic selection. As a disease trait, that is hard to measure but that has such high economic consequences, the use of genomic selection would be beneficial. Provided phenotypes were regularly collected from the reference population, to maintain accuracy, animals under selection in the wider population would not need to be exposed to the disease later in life to determine whether they were susceptible or not. As Rupp *et al.* (2016) discuss, genomic selection can prove useful for traits measured later in life and for those associated with disease. The ability to identify suitable (or non-suitable) animals early in life allows the number of animals that need

to be exposed to the disease to be greatly reduced, thus also leading to considerable welfare and productivity benefits.

The main objectives of this study were therefore, a) to estimate genetic parameters of traits relating to mastitis and udder conformation in a meat sheep breed; b) estimate the level of association between somatic cell counts and the CMT method and c) assess the relationships between the mastitis related traits and both udder conformation and lamb live weights.

Material and methods

Data collection

During 2015 and 2016, phenotypic data (relating to the udder, teats and milk quality) were collected from 2 957 purebred Texel ewes based on 29 flocks, located throughout the UK. The ewes were scored twice each year, by trained technicians, at mid-lactation (approximately 4 weeks after lambing) and again at late-lactation/weaning (approximately 11 weeks after lambing). All ewes included in the study were 2 years old or above and had pedigree and performance data available, via *Signet*'s *Sheepbreeder* programme (http://www.signetfbc.co.uk).

The udder and teat traits measured included 4 traits that were linear in form and scored using a 9-point scale, similar to those used by a number of previous studies assessing conformation traits in small ruminants (de la Fuente *et al.* 1996; Manfredi *et al.* 2001; McLaren *et al.* 2016). Udder Drop (UD) is the depth of the udder, scored from the rear, in relation to the hocks of the animal. A score of 5 indicates that the cleft of the udder is at the hock level, whereas scores 1 and 9 were well above or well below the hocks respectively. Udder Attachment (UA), also measured from the rear,

gives an indication of the strength of the attachment based on the perimeter of the insertion to the abdominal wall. Scores of 1 and 9 represent udders with a weak or strong level of attachment respectively. Teat Placement (TP) and Teat Angle (TA) were measured from the rear and the side of the animal respectively. Teat placement gives an indication as to placement of the teats in relation to the medial ligament. Teats pointing straight down, close to each other, were scored 1 whereas those pointing outwards, away from each other, were scored as 9. A score of 5 was given for teats at approximately a 45^{o} angle. Teat angle was measured from the animals left side, and scored from position 1 (approximately 8 o'clock on a clock face) to position 9 (approximately 4 o'clock on a clock face).

The remaining non-linear traits, all of which were measured in centimetres, were; Udder Length (UL), the distance between the udder cleft and the abdominal wall; Udder Width (UW), the measure of the udder width from the front to the rear and both the length (TL) and width (TW) of the teats. The average of both teat measurements, for TL and TW, were used in the final analyses.

Individual milk samples were collected from each ewe and tested by the National Milk Laboratories (http://www.nationalmilklaboratories.co.uk) for Somatic Cell Count (SCC) levels. One sample was collected from each side of the udder at the mid-lactation visit. The average cell count result (from both samples received from the laboratory) for each ewe was then used in the mid-lactation genetic analyses. Milk from both sides was combined into one sample during the late-lactation scoring, on-farm, therefore only one SCC result was received from the laboratory. The SCC values were then log-transformed using the formula $Log_e(SCC)$ similar to the method used by Mrode and Swanson (2003), to produce somatic cell score (SCS) values.

The California Mastitis Test (CMT) was also used to score a sample of milk from each side. The method involves combining an equal sample of milk with a reagent and then mixing for 15-20 seconds. Depending on the reaction that occurs, the samples are scored on a scale of 0 – 4 with score 4 indicating a high level of somatic cells present. Each udder half was awarded an individual CMT score, using the scores as described by Ruegg *et al.* (2005). The two CMT scores were then summed together (cmtSUM) or the maximum score across both halves was used (cmtMAX) in order to gain information on the severity of infection. The range of scores possible were therefore 0 – 8 for cmtSUM and 0 – 4 cmtMAX. Both cmtSUM and cmtMAX were log-transformed in order to normalise the data using the formulae $Log_e(cmtSUM\ value\ +1)$ and $Log_e(cmtMAX\ value\ +1)$ respectively.

Lamb live weights

The weight of lamb reared by each ewe, each year throughout her lifetime, was calculated using performance records available from the *Signet Sheepbreeder* programme. Lambs were weighed at approximately 8 weeks after birth to assess growth rate during this time. Of the 2 957 ewes included in the study, 2 863 also had data available for the 8-week weights of their lambs, up to 2016. In total, there were 4 077 total lamb weight reared records available from 2008 to 2016, of which 2 300 were collected during the two years of the project (2015 and 2016). The weights were used assess the total weight of lamb reared by the ewe (sum of weights of all lambs per ewe) each year and the average weight of lamb reared by the ewe each year (total weight adjusted for litter size and lamb sex).

Genetic Analysis

193 The pedigree file used in the analyses contained sire and dam information for a total
194 of 31 775 individuals. Variance components were estimated using univariate
195 analyses in ASReml (Gilmour *et al.* 2009). The animal models fitted included both
196 direct genetic and permanent environmental random effects. The following fixed
197 effects model was fitted for each trait (random effects in italics):

198 Mastitis/Udder Conformation Trait = ewe parity + (litter size born x litter size reared) +
199 lactation stage + scorer + (farm x lambing month x year) + *direct genetic* +
200 *permanent environment*

201 Where “x” represents an interaction between terms.

202 Ewe parity was the number of times the ewe had given birth and reared a lamb (5
203 levels; 1 to $\geq$ 5), litter size born was the number of lambs the ewe had given birth to
204 in the year of scoring (3 levels; 1 to $\geq$ 3) and litter size reared was the number of
205 lambs the reared during the year of scoring (3 levels; 1 to > 3). There were two
206 different scorers represented in the data. Lactation stage was defined as the number
207 of days between lambing date and scoring date and was fitted as a covariate. The
208 average lactation stage at mid- and late-lactation was 38 and 113 days respectively.
209 The contemporary group formed by the interactions between “farm x lambing month
210 x year” included 29 different farms, 2 different years (2015-2016) and 4 different
211 lambing months (February, March, April, May). Each fixed effect and/or interaction
212 was significant for the majority of traits, although not every fixed effect was significant
213 for each trait (Supplementary Tables S1 and S2). However, to remain consistent, the
214 same models were fitted across the different traits. The only exceptions to this were
215 for the SCS (where scorer was omitted), as these samples were processed by the
216 laboratory. The distributions of the residuals for each trait analyses were checked for

217 non-normality. With the exception of the traits already transformed (SCS, sumCMT
218 and maxCMT) no further trait transformations were required.

219 Univariate analyses for the total and average weight of lambs reared by the ewes, up
220 to 8-weeks old, were also estimated using animal models in ASReml (Gilmour *et al.*
221 2009). The following fixed effects model was fitted for each lamb weight trait (random
222 effects in italics):

223 Total weight of lambs reared by the ewe = lamb age + ewe parity + (farm x lambing
224 month x year) + *direct genetic* + *permanent environment*

225 Average weight of lambs reared by the ewe = lamb age + ewe parity + rearing
226 category + (farm x lambing month x year) + *direct genetic* + *permanent environment*

227 Where “x” represents an interaction between terms.

228 The covariate of age at weighing (in days, average of 66 days), the fixed effect of
229 ewe parity (5 levels; 1 to $\geq$ 5) and the combination of farm x lambing month x year
230 were also fitted in the model used for the total weight of lamb reared by the ewe up to
231 8-weeks. The same model was also fitted for average lamb weight reared by the ewe
232 up to 8-weeks, but also involved adjusting the total weight for ‘rearing category’ (a
233 factor with 6 levels combining the number and sex of the lambs reared; single male,
234 single female, twin males, twin females, twins of mixed sex and triplets, of any sex
235 combination). All effects fitted were significant (P<0.001).

236 Genetic and phenotypic correlations between all traits, associated with mastitis and
237 udder conformation, were estimated using bivariate analyses in ASReml (Gilmour *et*
238 *al.* 2009), fitting the same models as mentioned above. Multivariate analyses were
239 attempted but could not be completed due to lack of computational power. Genetic

and phenotypic correlations between both SCS and sumCMT and the weight of lamb reared by the ewe were also estimated using bivariate analyses in ASReml (Gilmour *et al.* 2009). The sumCMT trait was selected for these analyses as it provided a more detailed indication of the infection level across both udder halves.

Results

A summary for the traits included in the analyses, for mid-lactation and late-lactation, are given in Table 1. The averages decreased from mid-lactation to late-lactation for all udder traits indicating that the udders had reduced in size between scoring events. The teat trait means were similar across both scoring events, with teat width slightly higher at late-lactation. There was very little difference between the average SCS at both scoring events but the average values observed for the CMT traits fell slightly. The number of records available for the traits associated with the late-lactation scoring event was less than those for the mid-lactation traits. This was due to a combination of factors, predominantly influenced by the fact that a number of ewes were beginning to, or had already, dried off by the second scoring event, therefore samples or measurements could not be collected. CMT records were also removed if the ewe did not have two CMT scores (ie. from both udder halves).

Table 1.

Genetic Parameters

The univariate heritabilities for each trait, at both mid-lactation and late-lactation, are shown in Table 2. The heritabilities estimated across all traits, ranged from 0.08 to 0.35 (mid-lactation) and from 0.07 to 0.33 (late-lactation). The highest estimates were associated with the teat traits (particularly for teat placement and teat length)

whereas the lowest were generally associated with the mastitis traits (SCS, sumCMT and maxCMT).

Table 2

Relationships between somatic cell count (SCC) and California mastitis test

The cell counts associated with each CMT score, awarded to each individual udder half at mid-lactation across both sample years, are shown in Figure 3. Individual cell counts were not collected at late-lactation (as samples from both halves were mixed on-farm before being submitted for laboratory analysis). The medians of each CMT score, as indicated by the thick black lines in Figure 3, were 119 x 10^3 cells/ml; 295 x 10^3 cells/ml; 776 x 10^3; 3,857 x 10^3 and 18,082 x 10^3 somatic cells/ml for scores 0, 1, 2, 3 and 4 respectively. The arithmetic means for the corresponding scores were 189 x 10^3; 467 x 10^3; 1383 x 10^3; 6,403 x 10^3 and 16,139 x 10^3 somatic cells/ml respectively.

Figure 1.

Genetic and phenotypic relationships between all mastitis and udder conformation traits

The genetic and phenotypic correlations estimated between all mastitis traits (SCS, cmtSUM and cmtMAX) and the udder conformation traits, at mid- and late-lactation, are shown in Tables 3 and 4 respectively. The genetic correlations estimated between SCS and both CMT traits were highest at the mid-lactation recording event (0.96 to 0.98) when compared to those observed at late-lactation (0.76 to 0.79). The genetic correlations estimated between the two CMT traits, at each recording event, were both 0.99 therefore indicating that these traits were not significantly different.

Genetic correlations, significantly different from zero ($P<0.05$), observed between the udder depth, length and width and both SCS and CMT traits, at mid-lactation, were all positive ranging from 0.31 to 0.75. The genetic correlations associated with udder attachment and all mastitis traits were not significantly different from zero ($P>0.05$). Both genetic and phenotypic correlations associated with the angle of the teats were negative, where as those associated with the teat length and width measurements were all positive. The genetic correlations associated with teat placement were not significantly different from zero ($P>0.05$).

A range of genetic correlations amongst the udder conformation traits were observed, at mid-lactation, with the highest observed between udder depth and udder length (0.83) and between teat length and teat width (0.81). Moderate correlations were also observed between udder width and both udder depth and udder length (0.63 and 0.58 respectively). Correlations estimated between the udder and teat traits were low to moderate. The relationship between udder depth and both teat length and width were positive (0.34 to 0.38), where as the a negative relationship was observed between udder depth and teat angle (-0.40).

There was no obvious relationship between the mastitis traits and the udder traits at late-lactation, with the majority not significantly different from zero ($P>0.05$). The genetic correlations observed between the mastitis traits the teat traits were all significant ($P<0.05$), with the exception of those associated with teat placement. The correlations associated between the CMT traits and teat angle were in a similar direction to those observed at mid-lactation (-0.48 to -0.50). The genetic correlations observed between the mastitis traits and both teat length and width were all positive in strength (0.20 to 0.44).

As in the mid-lactation analyses, the genetic correlations estimated between udder depth and udder length and between teat length and teat width were moderate to high, ranging from (0.53 to 0.85). The relationships observed between udder depth and teat angle, length and width were also similar to the mid-lactation results, although the strength of the correlations had decreased.

Relationship between mastitis and weight of lamb reared by the ewe

The total weight of lambs reared by the ewes to 8-weeks old, each year, ranged from 7.5kg to 122kg, with an average of 39.9kg (SD 14.79) across 4,077 records (between 2008 and 2016). The relationship between the average total weight of lamb reared by the ewe and each sumCMT score awarded, using data from 2015 and 2016 only, is shown in Figure 4. The slope of trend line shown was estimated as -0.367, therefore indicating that a one point increase in sumCMT score reduced with the total weight of lamb reared by the ewe, on average, by 0.367 Kg.

Figure 4.

The univariate heritability estimate for the total weight of lamb reared by the ewes to 8-weeks old, using the data available between 2008 and 2016, was 0.06 (0.03). Similarly, the univariate heritability estimate for average weight of lambs reared by the ewes to 8-weeks old (total weight adjusted for litter size and lamb sex) was 0.10 (0.03).

The genetic and phenotypic correlations estimated between both SCS and sumCMT with the total and average weight of lamb reared by the ewe, up to 8-weeks old, are shown in Table 5. All genetic correlations estimated were not significantly different to zero ($P>0.05$). Significant negative phenotypic correlations ($P<0.05$) were observed,

ranging from -0.15 to -0.23, indicating that as infection levels rose, the weight of lamb (total and average) reared by the ewe decreased.

Table 5

Discussion

Over recent years the literature available on mastitis in small ruminants has been growing. The majority have concentrated on dairy animals, but the disease is also an important factor to consider in those reared for meat production. To our knowledge, the heritabilities presented for Somatic Cell Scores (SCS) and the California Mastitis Test (CMT) represent some of the first to be estimated in sheep reared primarily for meat production.

The heritabilities estimated for SCS ranged from 0.08 to 0.11, the highest occurring at mid-lactation. In studies where single test-day estimates were considered, in diary ewes, the heritabilities ranged between 0.04 to 0.12 when measured at different points throughout lactation (Barillet *et al.*, 2001 and Rupp *et al.,* 2003). Both authors also observed that the heritabilities rose as the lactation progressed. Psifidi *et al.* (2014) observed a decline in heritabilities in both SCC and CMT, up to week 10 of lactation, after which the estimates began to rise towards the end of lactation. The standard errors associated with the estimates for SCS (and indeed the CMT traits), in the current study, indicate they were not significantly different ($P<0.05$) across both scoring events. It should also be noted that the method used to transform the SCC data in the current study was the method used by Mrode and Swanson (2003), and not the method used by Ali and Shook (1980), used in previous analyses such as those by Barillet *et al.* (2001) and Rupp *et al.* (2003). When the two methods were compared, the distributions were similar. The method adopted in the current study is,

357 at present, used in dairy cattle evaluations across a number of different countries,
358 including Australia, Great Britain and the combined evaluations across Denmark,
359 Sweden and Finland (Interbull, 2017). It was therefore used to maintain consistency
360 with current commercial evaluations.

361 Although Riggio *et al.* (2013) concluded that SCS was the best indirect test of the
362 bacteriological status of the udder, when compared to the CMT, the CMT can be
363 considered as being a very good substitute for use in meat sheep production
364 systems. The median cell count values obtained for each CMT score recorded at
365 mid-lactation in the current study matched reasonably well with the ranges used by
366 Ruegg (2005). MacDougall *et al.* (2001) found that a score 3 (equivalent to a score 4
367 in the current study) was associated with a geometric mean of 8.8×10^6 somatic
368 cells/ml in sheep (and 7.5×10^6 in goats). Lower estimates have also been observed
369 by Kalogridou-Vassiliadou *et al.* (1992) and Gonzalez-Rodriguez and Carmenes
370 (1996). However, these differences could be influenced by various factors such as
371 different methods for calculating somatic cell counts (Kalogridou-Vassiliadou *et al.,*
372 1992); breed differences (Gonzalez-Rodriguez and Carmenes, 1996), or
373 environmental differences, such as production environment conditions (housed or
374 outside) or whether or not ewes were rearing suckling lambs (Arsenault *et al.,* 2008;
375 Waage and Vatn, 2008).

376 The genetic parameters estimated for the CMT traits in the current study, are to our
377 knowledge, the first to be estimated for meat producing sheep. Indeed although
378 Psifidi *et al.* (2014) estimated heritabilities ranging from approximately 0.06 to 0.42
379 throughout lactation in Chios dairy ewes, there are few estimates available in the
380 literature, across all ruminant species. The genetic relationships observed in the

current study between the CMT traits and SCS were very favourable, particularly at mid-lactation, indicating the traits were under a similar genetic influence. Whilst there were differences in the sampling methods at late-lactation, the lower correlations between the CMT traits and SCS at late-lactation could also be influenced by the fact many of these ewes were approaching, or had already achieved, the end of their lactation. Indeed an increase in false-positive results towards the end of the lactation period has been observed elsewhere (Gonzalez-Rodriguez and Carmenes, 1996). These results therefore indicate that selection upon CMT traits, to reduce mastitis levels, would be worthwhile, providing records were collected near mid-lactation rather than later in the lactation period.

Udder and teat conformation scores, and their associations with the mastitis related traits, demonstrated that further progress to reduce mastitis incidence could be achieved. The most commonly scored conformation traits in the literature include udder depth and teat placement. The heritabilities in the current study associated with udder depth and teat placement were in general agreement with previous studies (Legarra and Ugarte, 2005; Marie-Etancelin *et al.,* 2005; and de la Fuente *et al.,* 2011). Other traits with moderate heritability estimates included both teat length and width, which ranged from 0.25 to 0.34. Although measured in Alpine and Saanen dairy goats and not sheep, Manfredi *et al.* (2001) found both of these traits to have higher heritability estimates, ranging from 0.43 to 0.52. In the current study, both teat length and width were metric measurements, but other studies have considered similar traits using a 1-9 scoring system. These included De La Fuente *et al.* (2011), who observed heritabilities for teat length ranging from 0.16 to 0.30. Overall, the range of estimates observed, across both scoring events, indicate that all traits are heritable and therefore genetic improvement could be achieved through selection.

406 The correlations estimated between the udder conformation and mastitis traits were
407 higher at mid-lactation when compared to those observed at late-lactation. The
408 strongest correlations observed at mid-lactation for SCS and both CMT traits were
409 with udder depth (0.61 to 0.75). Similar observations were seen for udder length,
410 which itself was highly correlated with udder depth (0.83). These agree with previous
411 estimates such as those reported by Casu *et al.* (2010) and a number of studies
412 reviewed by Rupp and Boichard (2003). Udder width also proved influential on
413 infection levels, with wider udders associated with higher values associated with the
414 mastitis traits. Overall, these results indicate that longer, wider and therefore fuller
415 udders were more likely to have higher levels of infection.

416 In terms of the teat traits, negative correlations were estimated between the mastitis
417 traits and teat angle, indicating that teats positioned further forward on the udder
418 were at a higher the risk of infection. This may be due to the fact that there is less
419 protection from the elements or that they are more easily accessed by the suckling
420 lambs. The current study also indicates that longer and wider teats were also
421 associated with higher SCS and CMT scores, possibly influenced by the fact that
422 larger teats will contain a larger volume of residual milk increasing the possibility of
423 pathogens multiplying, as suggested by Huntley *et al.* (2012). However, the collection
424 of individual teat measurements is perhaps not appropriate for commercial meat-
425 sheep production systems. An alternative scoring method, such as the 9 point scale
426 for teat length used by De La Fuente (2011), or a trait associated with teat shape,
427 may prove more worthwhile.

428 Antagonistic correlations between SCS and milk yield, have been observed in dairy
429 cattle (Mrode and Swanson, 1996; Rupp and Boichard, 2003) and in some dairy

sheep studies (Rupp *et al.,* 2003). Rupp *et al.* (2003) reported correlations between SCS and milk yield between 0.05 and 0.23 throughout the first lactation in Lacaune sheep. However, other studies have reported opposite findings, such as the correlations of -0.15 and -0.30 observed by El-Saied *et al.* (1999) and Legarra and Ugarte (2005) respectively. There therefore seems to be some inconsistency. Although no information is available in the current study relating to milk yields, the weight of the lambs reared by the ewe is a more suitable indicator of performance for this type of production system. The live weights recorded at 8-weeks are currently used by *Signet*'s *Sheepbreeder* programme both as a direct trait of the lamb but also to assess the maternal ability of the ewe, depending on the breed and selection index used. The selection index currently used for Texel sheep in the UK has a high emphasis on carcass-related characteristics and less on maternal traits. However, the heritabilities estimated in the current study for the total and average weight of lamb reared by the ewe, although low indicate that genetic progress could be achieved if these traits were selected upon in the future. The relationships observed between SCS and sumCMT and both the total and average weight of lambs reared by the ewes indicate that the higher the level of infection in the milk, the lighter the lambs at the 8-week weight. This relationship has been observed in a number of other studies, including that by Huntley *et al.* (2012) and Moroni *et al.* (2007). Therefore a reduction in the infection levels of the ewe's milk will have positive effect on the weight of lambs reared by the ewe and the overall production output of the flock. Indeed, the trend observed between the average total weight of lamb reared and each sumCMT score recorded during 2015 and 2016 (Figure 2) indicated that a one point change in sumCMT score reduced the total weight of lamb reared, on average, by 0.367 grams. This suggests that a ewe with a sumCMT score of 8 would

be rearing a total weight of lamb, on average, 2.936 Kg lighter than a ewe with a sumCMT score of 0. If we consider this relationship in monetary terms, using the current average price per kilo for medium farm assured lambs in GB markets, according to AHDB Beef & Lamb (2017) of £2.00 per kilo live weight, for every one point change in sumCMT score, the value of the lamb reared would reduce by 73p, per ewe. Additionally, the difference between the weight of lamb reared by a ewe scoring 0 and a ewe scoring 8 would be £5.87. If this relationship also observed in sheep systems in other countries as well, the financial implications would be even more substantial. The genetic correlations associated with both mastitis traits and the average weight of lambs reared by the ewe were not significantly different from zero, indicating that these traits are under different genetic control and any future selection to improve the average weight of lambs reared would not be associated with a higher genetic incidence of mastitis.

Conclusions

The results presented have improved our knowledge in terms of meat producing sheep, for a number of different aspects associated with mastitis. First of all, the validation that the CMT method is highly correlated with SCS and is therefore a good indicator for mastitis is notable, given the relatively few recent studies to date that have investigated this method. Secondly, the fact that both SCS and, perhaps of more significant interest the CMT traits, have been proven to have a genetic component in this breed. These are the first known estimates to be produced for meat sheep in the UK and will allow future genetic selection upon these traits to be explored, particularly relevant in the era of genomic selection. The ability to identify animals suitable for further breeding, at an early stage before they have been

exposed to the disease, will not only improve the rates of genetic improvement, but also have a positive impact on flock productivity and overall health and welfare. The relationship between both SCS and sumCMT traits and the weight of lambs reared by the ewe is also of significant interest with improvements in lamb weights possible if infection levels in the milk are reduced. The overall economic benefits therefore, of these results combined, could be substantial not only in this breed but also in the meat sheep industry as a whole.

Acknowledgements

This paper is part of an 18-month project co-funded by Innovate UK. The authors gratefully acknowledge the cooperation with all the Texel Sheep Society members who provided us with their time and animals throughout the study period. Thanks also to Will Sawday for his involvement in the data collection and to Laura Nicoll, Claire Anderson and William Conington for their technical assistance.

References

AHDB Beef & Lamb. 2017. Agriculture & Horticulture Development Board (AHDB) Beef & Lamb weekly GB regional averages. Retrieved on 8th August 2017. http://beefandlamb.ahdb.org.uk/markets/auction-market-reports/weekly-gb-regional-averages/

Ali AKA and Shook GE. 1980. An optimum transformation for somatic cell concentration in milk. Journal of Dairy Science 63, 487-490.

Arsenault J, Dubreuil P, Higgins R and Belanger D. 2008. Risk factors and impacts of clinical and subclinical mastitis in commercial meat-producing sheep. Preventive Veterinary Medicine 87, 373-393.

503 Barillet F, Rupp R, Mignon-Grasteau S, Astruc J-M and Jacquin M. 2001. Genetic analysis
504 for mastitis resistance and milk somatic cell score in French Lacaune dairy sheep.
505 Genetic Selection Evolution 33, 397-415.
506 Bergonier D and Berthelot X. 2003. New advances in epizootiology and control of ewe
507 mastitis. Livestock Production Science 79, 1-16.
508 Conington J, Cao G, Stott A and Bunger L. 2008. Breeding for resistance to mastitis in
509 United Kingdom sheep, a review and economic appraisal. Veterinary Record 162, 369-
510 376.
511 Contreras A, Sierra D, Corrales JC, Sanchez A and Marco J. 1996. Physiological threshold
512 of somatic cell count and California Mastitis Test for diagnosis of caprine subclinical
513 mastitis. Small Ruminant Research 21, 259-264.
514 De la Fuente LF, Fernandez G and San Primitivo F. 1996. A linear evaluation system for
515 udder traits of dairy ewes. Livestock Production Science 45, 171-178.
516 De la Fuente LF, Gonzalo C, Sanchez JP, Rodriguez R, Carriedo JA and San Primitivo F.
517 2011. Genetic parameters of the linear body conformation traits and genetic
518 correlations with udder traits, milk yield and composition, and somatic cell count in
519 dairy ewes. Canadian Journal of Animal Science 91, 585-591.
520 El-Saied UM, Carriedo JA, De La Fuente LF and San Primitivo F. 1999. Genetic Parameters
521 of Lactation Cell Counts and Milk and Protein Yields in Dairy Ewes. Journal of Dairy
522 Science 82, 639-644.
523 Fragkou IA, Boscos CM and Fthenakis GC. 2014. Diagnosis of clinical or subclinical mastitis
524 in ewes. Small Ruminant Research 118, 86-92.
525 Gelasakis AI, Mavrogianni VS, Petridis IG, Vasileiou NGC and Fthenakis GC. 2015. Mastitis
526 in sheep – The last 10 years and the future of research. Veterinary Microbiology 81,
527 136-146.
528 Gilmour AR, Gogel B, Cullis BR and Thompson R. 2009. ASReml User Guide Release 3.0.
529 VSN International Ltd. Hemel Hempstead, HP1 1ES, UK.

Gonzalez-Rodriguez MC and Carmenes P. 1996. Evaluation of the California mastitis test as a discriminant method to detect subclinical mastitis in ewes. Small Ruminant Research 21, 245-250.

Huntley SJ, Cooper S, Bradley AJ and Green LE. 2012. A cohort study of the assocations between udder conformation, milk somatic cell count, and lamb weight in suckler ewes. Journal of Dairy Science 95. 5001-5010.

Interbull. 2017. National genetic evaluation forms provided by countries. Retrieved on 18th December 2017. http://www.interbull.org/ib/geforms

Kalogridou-Vassiliadou D, Manolkidis K and Tsigoida A. 1992. Somatic cell counts in relation to infection status of the goat udder. Journal of Dairy Research 59, 21-28.

Legarra A. and Ugarte E. 2005. Genetics parameters of Udder Traits, Somatic Cell Score, and Milk Yield in Latxa Sheep. Journal of Dairy Science 88, 2238-2245.

Marie-Etancelin C, Astruc JM, Porte D, Larroque H and Robert-Granie D. 2005. Multiple-trait genetic parameters and genetic evaluation of udder-type traits in Lacaune dairy ewes. Livestock Production Science 97, 211-218.

Manfredi E, Piacere A, Lahaye P and Ducrocq V. 2001. Genetic parameters of type appraisal in Saanen and Alpine goats. Livestock Production Science 70, 183-189.

McDougall S, Murdough P, Pankey W, Delaney C, Barlow J and Scruton D. 2001. Relationships among somatic cell count, California mastitis test, impedance and bacteriological status of milk in goats and sheep in early lactation. Small Ruminant Research 40, 245-254.

McLaren A, Mucha S, Mrode R, Coffey M and Conington J. 2016. Genetic parameters of linear conformation type traits and their relationship with milk yield throughout lactation in mixed-breed dairy goats. Journal of Dairy Science 99, 5516-5525.

Moroni P, Pisoni G, Varisco G and Boettcher P. 2007. Effect of intramammary infection in Bergamasca meat sheep on milk parameters and lamb growth. The Journal of Dairy Research 74, 340-344.

Mrode RA and Swanson GJT. 1996. Genetic and statistical properties of somatic cell count and its suitability as an indirect means of reducing the incidence of mastitis in dairy cattle. Animal Breeding Abstracts 64, 847-857.

Mrode RA and Swanson GJT. 2003. Estimation of genetic parameters for somatic cell count in the first three lactations using random regression. Livestock Production Science 79, 239-247.

Psifidi A, Bramis G, Arsenos G and Banos G. 2014. Genetic Parameters and Genomic Markers Associated with Mastitis Resistance in Dairy Sheep. Proceedings of the 10th World Congress of Genetics Applied to Livestock Production, 17th-22nd August 2014, Vancouver, Canada, p. 521.

Riggio V, Pesce LL, Morreale S and Portolano B. 2013. Receiver-operating characteristic curves for somatic cell scores and California mastitis test in Valle del Belice dairy sheep. The Veterinary Journal 196, 528-532.

Riggio V and Portolano B. 2015. Genetic selection for reduced somatic cell counts in sheep milk: A review. Small Ruminant Research 126, 33-42.

Ruegg P 2005. California Mastitis Test (CMT) Fact Sheet 1. Retrieved on 1st February 2016 from http://milkquality.wisc.edu/wp-content/uploads/2011/09/california-mastitis-test-fact-sheet.pdf

Rupp R and Boichard D. 2003. Genetics of resistance to mastitis in dairy cattle. Veterinary Research 34, 671-688.

Rupp R, Lagriffoul G, Astruc JM and Barillet F. 2003b. Genetic Parameters for Milk Somatic Cell Scores and Relationships with Production Traits in French Lacaune Dairy Sheep. Journal of Dairy Science 86, 1476-1481.

Rupp R, Bergonier D, Dion S, Hygonenq MC, Aurel MR, Robert-Granie C and Foucras G. 2009. Response to somatic cell count-based selection for mastitis resistance in a divergent selection experiment in sheep. Journal of Dairy Science 92, 1203-1219.

583 Rupp RA and Foucras GI 2010. “Genetics of Mastitis in Dairy Ruminants”. In Breeding for
584 Disease Resistance in Farm Animals (3rd edition) (eds. SC Bishop, RFE Axford, FW
585 Nicholas and JB Owens), pp. 183-212. CAB International.
586 Rupp R, Mucha S, Larroque H, McEwan J and Conington J. 2016. Genomic application in
587 sheep and goat breeding. Animal Frontiers 6, 39-44.
588 Tolone M, Riggio V and Portolano B. 2013. Estimation of genetic and phenotypic
589 parameters for bacteriological status of the udder, somatic cell score, and milk yield in
590 dairy sheep using a threshold animal model. Livestock Science 151, 134-139.
591 Waage S and Vatn S. 2008. Individual animal risk factors for clinical mastitis in meat sheep
592 in Norway. Preventive Veterinary Medicine 87, 229-243.
593

Table 1 Summary of traits included in the analyses

	Mid-Lactation					Late-Lactation				
Trait	Count	Min.	Max.	Mean	s.d.	Count	Min.	Max.	Mean	s.d.
Udder Depth (UD)	3591	1	8	3.32	1.07	3083	1	8	2.92	1.06
Udder Attach. (UA)	3589	1	9	7.49	1.21	3082	2	9	7.16	1.21
Udder Length (UL) (cm)	3589	4.00	30.50	13.48	2.67	3082	3.80	25.70	11.34	2.63
Udder Width (UW) (cm)	3588	5.00	29.40	15.49	2.92	3080	4.00	25.80	13.57	2.68
Av.[1] Teat Length (TL) (cm)	3590	1.30	4.90	2.58	0.42	3085	1.20	4.70	2.48	0.36
Av.[1] Teat Width (TW) (cm)	3590	0.80	4.40	1.61	0.27	3085	0.70	5.80	2.28	0.87
Teat Placement (TP)	3590	1	9	5.93	1.39	3080	1	9	6.5	1.48
Teat Angle (TA)	3590	1	8	3.60	1.03	3081	1	8	3.27	1.06
Av.[1] SCS (SCS)[2]	3410	6.91	17.22	12.88	1.65	2628	6.91	17.39	12.82	2.13
Sum CMT[3] (cmtSUM)[5]	3539	0	2.20	1.07	1.11	2337	0	2.20	0.79	0.81
Max CMT[4] (cmtMAX)[5]	3529	0	1.61	0.87	0.86	2337	0	1.61	0.67	0.67

[1]Average of samples collected from both udder halves

[2]Somatic cell scores (SCS) calculated by transforming somatic cell counts (SCC) using equation Log_e(SCC)

[3]Sum of California Mastitis Test (CMT) scores awarded across both udder halves

[4]Maximum California Mastitis Test (CMT) score awarded across both udder halves

[5]Data transformed using the equation Log_e(CMT score + 1)

Table 2 Univariate heritabilities (h^2), permanent environment variance (*pe*) and phenotypic variances (σ^2_p) for traits scored at mid- and late-lactation (SE in parentheses).

	Mid-Lactation			Late-Lactation		
Trait	h^2	*pe*	σ^2_p	h^2	*pe*	σ^2_p
Udder Depth (UD)	0.20 (0.05)	0.27 (0.05)	0.81 (0.02)	0.21 (0.05)	0.26 (0.06)	0.89 (0.03)
Udder Attach. (UA)	0.14 (0.04)	0.13 (0.06)	1.04 (0.03)	0.14 (0.05)	0.12 (0.06)	1.17 (0.04)
Udder Length (UL)	0.20 (0.05)	0.26 (0.05)	4.77 (0.15)	0.16 (0.05)	0.33 (0.06)	5.23 (0.17)
Udder Width (UW)	0.14 (0.04)	0.09 (0.05)	4.67 (0.14)	0.17 (0.05)	0.07 (0.06)	4.99 (0.16)
Teat Placement (TP)	0.35 (0.05)	0.27 (0.05)	1.86 (0.06)	0.27 (0.06)	0.27 (0.06)	2.08 (0.07)
Teat Angle (TA)	0.19 (0.05)	0.19 (0.05)	0.93 (0.03)	0.25 (0.06)	0.13 (0.06)	0.96 (0.03)
Av.[1] Teat Length (TL)	0.34 (0.05)	0.24 (0.05)	0.14 (0.005)	0.33 (0.06)	0.25 (0.06)	0.11 (0.004)
Av.[1] Teat Width (TW)	0.28 (0.05)	0.28 (0.05)	0.06 (0.002)	0.25 (0.06)	0.24 (0.06)	0.10 (0.003)
Av.[1] SCS (SCS)[2]	0.11 (0.04)	0.15 (0.05)	2.45 (0.07)	0.08 (0.05)	0.16 (0.07)	3.65 (0.12)
Sum CMT[3] (cmtSUM)[5]	0.09 (0.04)	0.15 (0.05)	0.50 (0.01)	0.11 (0.06)	0.12 (0.07)	0.55 (0.02)
Max CMT[4] (cmtMAX)[5]	0.08 (0.04)	0.14 (0.05)	0.36 (0.01)	0.07 (0.05)	0.14 (0.07)	0.39 (0.01)

[1]Average of samples collected from both udder halves

[2]Somatic cell scores (SCS) calculated by transforming somatic cell counts (SCC) using equation Log_e(SCC)

[3]Sum of California mastitis test (CMT) scores awarded across both udder halves

[4]Maximum California mastitis test (CMT) score awarded across both udder halves

[5]Data transformed using the equation Log_e(CMT score + 1

Table 3 Genetic (above diagonal) and phenotypic (below diagonal) correlations (SE in parentheses) between all mastitis traits (somatic cell score and California Mastitis Test) and udder conformation traits, measured at mid-lactation.

Mid-Lactation	SCS	cmtSUM	cmtMAX	UD	UA	UL	UW	TA	TP	TL	TW
Somatic Cell Score (SCS)		**0.96 (0.04)**	**0.98 (0.04)**	**0.61 (0.11)**	**ns[4]**	**0.53 (0.14)**	**0.31 (0.13)**	**-0.41 (0.13)**	**ns[4]**	**0.26 (0.09)**	**0.44 (0.09)**
Sum of CMT (cmtSUM)[1]	0.73 (0.01)		**0.99 (0.01)**	**0.75 (0.18)**	**ns[4]**	**0.53 (0.16)**	**0.44 (0.18)**	**-0.55 (0.17)**	**ns[4]**	**0.37 (0.10)**	**0.50 (0.11)**
Max. CMT (cmtMAX)	0.73 (0.01)	0.97 (0.001)		**0.71 (0.16)**	**ns[4]**	**0.49 (0.15)**	**0.33 (0.16)**	**-0.54 (0.16)**	**ns[4]**	**0.38 (0.11)**	**0.51 (0.11)**
Udder Depth (UD)	0.15 (0.02)	0.13 (0.02)	0.14 (0.02)		**ns[4]**	**0.83 (0.04)**	**0.63 (0.06**	**-0.40 (0.09)**	**0.18 (0.07)**	**0.34 (0.06)**	**0.38 (0.06)**
Udder Attach. (UA)	-0.05 (0.02)	-0.07 (0.02)	-0.07 (0.02)	0.10 (0.02)		**ns[4]**	**0.21 (0.10)**	**ns[4]**	**-0.18 (0.08)**	**ns[4]**	**ns[4]**
Udder Length (UL)	0.08 (0.02)	ns[4]	0.05 (0.02)	0.63 (0.01)	0.12 (0.02)		**0.58 (0.07)**	**ns[4]**	**0.16 (0.07)**	**0.28 (0.06)**	**0.33 (0.07)**
Udder Width (UW)	0.06 (0.02)	0.07 (0.02)	0.08 (0.02)	0.45 (0.02)	0.29 (0.02)	0.39 (0.02)		**ns[4]**	**0.29 (0.08)**	**0.24 (0.07)**	**0.33 (0.08)**
Teat Angle (TA)	-0.09 (0.02)	-0.11 (0.02)	-0.11 (0.02)	-0.13 (0.02)	ns[4]	ns[4]	ns[4]		**-0.26 (0.08)**	**-0.23 (0.07)**	**-0.31 (0.07)**
Teat Placement (TP)	ns[4]	ns[4]	ns[4]	ns[4]	-0.10 (0.02)	ns[4]	ns[4]	-0.09 (0.02)		**-0.40 (0.05)**	**-0.39 (0.06)**
Teat Length (TL)[3]	0.12 (0.02)	0.13 (0.02)	0.14 (0.02)	0.18 (0.02)	ns[4]	0.16 (0.02)	0.13 (0.02)	-0.10 (0.02)	-0.21 (0.02)		**0.81 (0.04)**
Teat Width (TW)[3]	0.14 (0.02)	0.16 (0.02)	0.17 (0.02)	0.24 (0.02)	0.08 (0.02)	0.20 0.02)	0.22 (0.02)	-0.09 (0.02)	-0.21 (0.02)	0.56 (0.01)	

[1] Sum of California Mastitis Test (CMT) scores awarded across both udder halves

[2] Maximum California Mastitis Test (CMT) score awarded across both udder halves

[3] Average of teat measurements across both udder halves

[4] Correlations not significantly (ns) different to zero (P>0.05)

Table 4 Genetic (above diagonal) and phenotypic (below diagonal) correlations (SE in parentheses) between all mastitis traits (somatic cell score and California Mastitis Test) and udder conformation traits, measured at late-lactation

Late-Lactation	SCS	cmtSUM	cmtMAX	UD	UA	UL	UW	TA	TP	TL	TW
Somatic Cell Score (SCS)		**0.76 (0.09)**	**0.79 (0.09)**	**ns[4]**	**ns[4]**	**ns[4]**	**ns[4]**	**ns[4])**	**ns[4]**	**0.26 (0.10)**	**0.20 (0.09)[5]**
Sum of CMT (cmtSUM)[1]	0.65 (0.01)		**0.99 (0.01)**	**ns[4]**	**ns[4]**	**ns[4]**	**ns[4]**	**-0.48 (0.15)**	**ns[4]**	**0.41 (0.11)**	**0.39 (0.10)[5]**
Maximum CMT (cmtMAX)[2]	0.65 (0.01)	0.97 (0.001)		**0.40 (0.19)**	**ns[4]**	**ns[4]**	**ns[4]**	**-0.50 (0.17)**	**ns[4]**	**0.44 (0.12)**	**0.39 (0.10)**
Udder Depth (UD)	ns[4]	ns[4]	ns[4]		**ns[4]**	**0.85 (0.03)**	**0.53 (0.08)**	**-0.21 (0.09)**	**ns[4]**	**0.19 (0.07)**	**0.23 (0.06)[5]**
Udder Attach. (UA)	-0.15 (0.02)	-0.21 (0.02)	-0.19 (0.02)	0.27 (0.02)		**ns[4]**	**0.37 (0.14)**	**0.26 (0.12)**	**ns[4]**	**ns[4]**	**ns[4]**
Udder Length (UL)	-0.05 (0.02)	-0.07 (0.02)	ns[4]	0.67 (0.01)	ns[4]		**0.53 (0.09)**	**ns[4]**	**ns[4]**	**0.15 (0.07)**	**ns[4]**
Udder Width (UW)	-0.06 (0.02)	-0.11 (0.02)	-0.08 (0.02)	0.47 (0.02)	0.42 (0.02)	0.44 (0.02)		**ns[4]**	**ns[4]**	**ns[4]**	**0.23 (0.08)[5]**
Teat Angle (TA)	-0.10 (0.02)	-0.19 (0.02)	-0.17 (0.02)	ns[4]	0.08 (0.02)	0.10 (0.02)	0.13 (0.02)		**ns[4]**	**-0.32 (0.07)**	**-0.18 (0.06)[5]**
Teat Placement (TP)	ns[4]	ns[4]	-0.01 (0.02)	-0.07 (0.02)	-0.12 (0.02)	-0.08 (0.02)	-0.06 (0.02)	-0.07 (0.02)		**-0.29 (0.06)**	**ns[4]**
Teat Length (TL)[3]	0.11 (0.02)	0.10 (0.02)	0.11 (0.02)	0.14 (0.02)	0.03 (0.02)	0.09 (0.02)	0.13 (0.02)	-0.12 (0.02)	-0.18 (0.02)		**0.53 (0.05)**
Teat Width (TW)[3]	0.05 (0.02)[4]	0.09 (0.03)[4]	0.10 (0.03)	0.15 (0.02)[4]	0.07 (0.02)[4]	0.09 (0.02)[4]	0.16 (0.02)[4]	-0.08 (0.02)[4]	-0.11 (0.02)[4]	0.41 (0.02)	

[1] Sum of California Mastitis Test (CMT) scores awarded across both udder halves

[2] Maximum California Mastitis Test (CMT) score awarded across both udder halves

[3] Average of teat measurements across both udder halves

[4] Correlations not significantly (ns) different to zero ($P>0.05$)

[5] No permanent environment effect fitted

Table 5 Genetic (r_g) and phenotypic (r_p) correlations (SE in parentheses) between mastitis traits (somatic cell score and California Mastitis Test) recorded at mid-lactation and the weight of lamb reared by the ewe up to 8-weeks old.

	Somatic Cell Score (SCS)		sumCMT[1]	
	r_g	r_p	r_g	r_p
Total weight of lamb reared	-0.39 (0.19)	-0.23 (0.02)	-0.20 (0.21)	-0.20 (0.02)
Average weight of lamb reared	-0.03 (0.18)	-0.16 (0.02)	-0.09 (0.18)	-0.15 (0.02)

[1] Sum of California Mastitis Test (CMT) scores awarded across both udder halves

Figure Captions

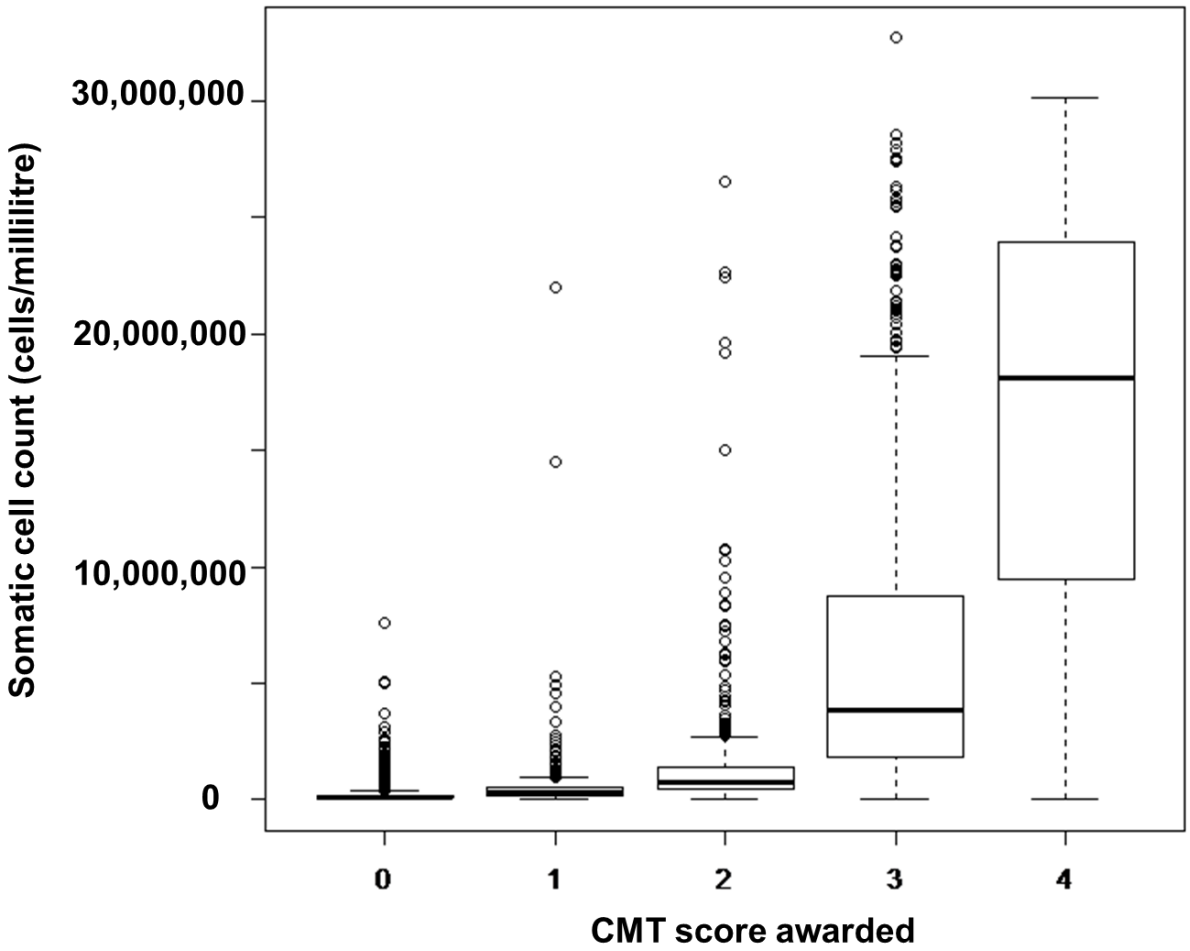
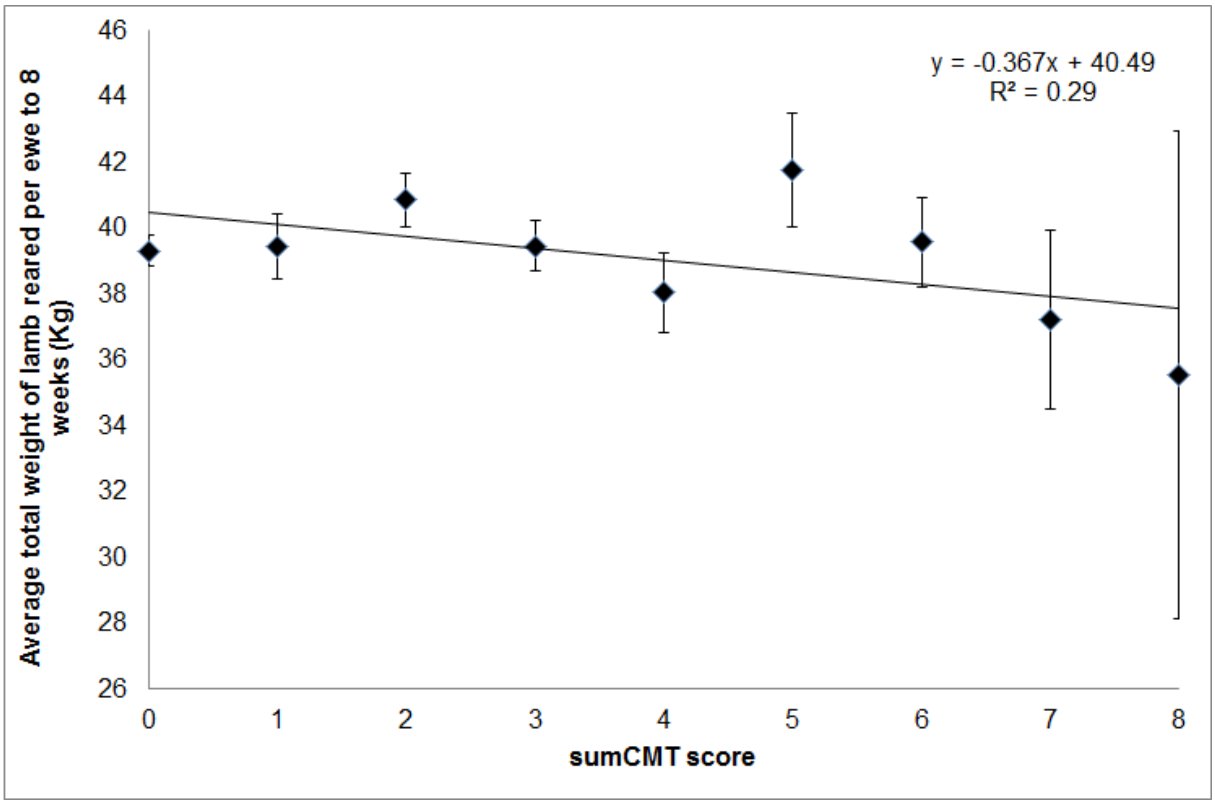


Figure 1. Boxplot of somatic cell counts associated with each California Mastitis Test (CMT) score



649

650 **Figure 2.** Average total weight of lamb reared by the ewes, at 8 weeks old,
651 associated with each sumCMT (sum of California Mastitis Test) score.
652
653