Factors affecting ewe somatic cell count and its relationship with lamb weaning weight in extensively managed flocks[1]

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INTRODUCTION

Mastitis, an inflammation of the mammary gland caused by bacterial infection, is characterized by palpable lumps in the udder, abnormal milk, and tissue discoloration in its clinical state (Menzies and Ramanoon, 2001). The average incidence of clinical mastitis (**CM**) is relatively low (1.2% to 3%) across flocks sampled around the world (Quinlivan, 1968; Arsenault et al., 2008; Cooper et al., 2016), but can have large variation within flocks (0–37%; Grant et al., 2016). Nevertheless, CM was the primary reason for culling 6.7% of ewes in the United States in 2011 (USDA APHIS, 2012).

Subclinical mastitis (**SCM**) has no visual symptoms but can be diagnosed through bacterial culture and(or) quantifying somatic cell count (**SCC**) in milk. The morbidity rate of SCM in sheep is much greater than CM (12–50%; Watkins et al., 1991; Ahmad et al., 1992; Keisler et al., 1992; Arsenault et al., 2008). Lambs reared by ewes with experimentally (Fthenakis and Jones, 1990) and naturally acquired SCM (Moroni et al., 2007) have reduced growth. However, the direct effect of maternal SCC on lamb growth has been inconsistent (Ahmad et al., 1992) or insignificant (Gross et al., 1978; Keisler et al., 1992) in the reviewed literature. The objectives of the present study were to quantify the relationship of: udder half SCC within and between collection dates; ewe age, breed, litter size (**NLB**), and serum-trace mineral concentration and SCC; and maternal SCC and lamb growth.

MATERIALS AND METHODS

The Montana State University (**MSU**) Agricultural Animal Care and Use Committee and the U.S. Sheep Experiment Station (**USSES**) Institutional Animal Care and Use Committee approved all husbandry practices and experimental procedures used in this study (2017-AA04 and 1803, respectively).

Data Collection

Milk was sampled from ewes free from CM at both MSU and the USSES during the spring of 2017. The same Rambouillet ($n = 26$) and Targhee ($n = 30$) ewes at MSU were sampled twice, the first shortly after parturition (<5 d in milk; early) and the second before turnout to summer grazing (30 to

45 d post-lambing; peak). Milk was collected once from Suffolk ($n = 38$) and crossbred (3/8 Suffolk, 3/8 Columbia, 1/4 Texel; $n = 46$) USSES ewes at peak lactation. At each collection, teat ends were cleaned with 99% isopropyl alcohol and 35 mL of milk was collected from each half, preserved with 8 mg Bronopol and 0.3 mg Natamycin (Microtabs II; D & F Control Systems, Inc.; Dublin, CA), and refrigerated until SCC analysis.

Milk SCC was quantified within 3 d of collection on a LactiCyte HD (Page & Pedersen International, Ltd.; Hopkinton, MA) according to the manufacturer's protocol. Blood was collected from MSU ewes via jugular venipuncture into 13×100 mm royal blue top trace element Vacutainer tubes (Covidien Ltd.; Mansfield, MA) and later centrifuged at 2,700 g and 4 °C for 30 min. The resulting serum was aliquoted and stored at −20 °C until trace mineral analysis (Michigan State University Veterinary Diagnostic Laboratory; Lansing, MI).

Statistical Analyses

Milk SCC was transformed to the $\log_{10}$ scale (**LSCC**) and the CORR procedure of SAS (v. 9.4; SAS Inst. Inc., Cary, NC) was used to estimate Pearson correlation coefficients between udder half LSCC within each collection date. At MSU, additional correlation coefficients were estimated between the same udder half LSCC across each collection date and between composite udder half LSCC (**LSCC$_A$**) at early and peak lactation.

Ewe LSCC$_A$ within collection date and flock was analyzed in the GLM procedure with class effects of ewe age [1 (USSES only), 2, or 3+], breed, and NLB (1 or 2+). A subset of MSU ewes with low LSCC$_A$ (<5.7) at both collections (**LL**; $n = 12$) and low LSCC$_A$ at early and high LSCC$_A$ (≥5.7) at peak lactation (**LH**; $n = 12$) were identified. Serum Se and Zn concentrations were analyzed for this subset in the MIXED procedure with fixed effects of LSCC$_A$ class (LL or LH) and collection date and the random effect of ewe.

Lamb weaning weights were adjusted to 120 d and summed within ewe (litter weaning weight; **LWW**), and a zero was recorded in the event that all lambs in a litter did not survive to weaning. Total LWW at MSU was analyzed in two separate models in the GLM procedure that fit either early or peak LSCC$_A$ as a linear covariate and the fixed-class effects of ewe breed and age. The same model was used to analyze LWW at USSES, but only peak LSCC$_A$ was available for these ewes.

RESULTS AND DISCUSSION

Relationship of SCC Within and Between Collection Dates

The Pearson correlation coefficient between udder half LSCC in early lactation was moderate (0.45; $P < 0.01$) at MSU. At peak lactation, the correlation between udder half LSCC was moderate at USSES (0.35; $P = 0.001$) and strong at MSU (0.92; $P < 0.001$). The correlation coefficients between left (0.03) and right (0.15) udder half LSCC and LSCC$_A$ (0.04) across collection dates were not significantly different from zero ($P \geq 0.33$) at MSU. Ahmad et al. (1992) reported that the spontaneous cure rate of intramammary infection present at lambing was 59%. Therefore, SCC is not constant throughout lactation which likely warrants multiple collections.

Production and Biological Factors Influencing Ewe SCC

Ewe age, breed, and NLB did not affect ($P \geq 0.13$) any ewe LSCC$_A$ measure at MSU or USSES. Waage and Vatn (2008) reported that ewes rearing three or more lambs were 6.7 times more likely to develop CM than ewes rearing a single lamb. Additionally, Gross et al. (1978), Ahmad et al. (1992), and Arsenault et al. (2008) reported that SCC increased with ewe age. Data in the present study are part of an ongoing project and it is possible that ewe production factors that influence SCM may be identified in future analyses.

Table 1. Least-squares means (±SE) for the main effects of collection date and udder half composite $\log_{10}$ somatic cell count (LSCC$_A$) class on Montana State University ewe serum Se and Zn concentrations

Effect	Level	Serum trace mineral	
		Se, ng • mL^{-1}	Zn, µg • mL^{-1}
Collection date*	Early	118.7 ± 4.71[b]	1.24 ± 0.12
	Peak	142.1 ± 4.63[a]	1.42 ± 0.11
LSCC$_A$ class[†]	LL	127.0 ± 5.59	1.53 ± 0.11[a]
	LH	133.9 ± 5.67	1.13 ± 0.12[b]

*Early, peak = serum-trace mineral concentration quantified at early (<5 d) or peak (30 to 45 d) lactation, respectively.

[†]LL = ewes with low LSCC$_A$ (<5.7) at both early and peak lactation; LH = ewes with low LSCC$_A$ at early and high LSCC$_A$ (≥5.7) at peak lactation.

[a,b]Means within an effect and column with no superscript in common are different ($P < 0.05$).

Table 2. Least-squares means (±SE) for the main effects of ewe age and breed and solutions for udder half composite log_{10} somatic cell count ($LSCC_A$) on total LWW at MSU and the USSES

Effect	Level	LWW MSU, kg	LWW USSES, kg
Ewe age, yr	1	—	32.8 ± 5.44[b]
	2	35.0 ± 2.08[b]	50.6 ± 4.76[a]
	3+	41.7 ± 2.08[a]	59.6 ± 2.71[a]
MSU/USSES breed*	Rambouillet/Crossbred	38.6 ± 2.26	53.8 ± 3.26[a]
	Targhee/Suffolk	38.1 ± 1.93	41.5 ± 3.39[b]
$LSCC_A$[†]	Early	−12.8 ± 4.00*	—
	Peak	ns[‡]	⁻14.8 ± 5.22*

*Rambouillet and Targhee ewes were sampled at MSU, Crossbred (3/8 Suffolk, 3/8 Columbia, 1/4 Texel) and Suffolk ewes were sampled at USSES.

[†]Early, Peak = $LSCC_A$ quantified at early (< 5 d) and peak (30 to 45 d) lactation.

[‡]ns = coefficient is not different from zero ($P = 0.87$).

[a,b]Means within an effect and column with no superscript in common are different ($P < 0.05$).

Least-squares means of the main effects of collection date and $LSCC_A$ group on MSU ewe serum Se and Zn concentrations are displayed in Table 1. Serum Se was 23.4 ng • mL⁻¹ greater ($P < 0.01$) in peak lactation, but no difference in serum Zn was detected ($P = 0.29$) between collection dates. Early and peak lactation $LSCC_A$ class had no effect ($P = 0.39$) on serum Se concentration. However, LL ewes had 0.40 µg • mL⁻¹ greater ($P = 0.02$) serum Zn concentration than LH ewes. Zinc is involved in the production and maintenance of keratinized tissues (O'Rourke, 2009) and Saianda et al. (2007) reported that bacterial adherence to the mammary epithelium was greatly reduced in dairy ewes supplemented with additional Zn.

Maternal SCC and Lamb Growth

Least-squares means for the main effects of ewe age and breed and solutions for $LSCC_A$ on LWW are displayed in Table 2. Not surprisingly, LWW was 6.7–26.8 kg greater ($P ≤ 0.03$) in multiparous ewes than primiparous ewes at MSU at USSES. Rambouillet and Targhee ewes had similar ($P = 0.88$) LWW at MSU but USSES crossbred ewes weaned 12.3 kg heavier litters ($P < 0.01$) than Suffolk ewes. At MSU, LWW was negatively affected by $LSCC_A$ in early (−12.8 kg; $P < 0.01$) but not peak lactation ($P = 0.87$). However, peak $LSCC_A$ reduced LWW (−14.8 kg; $P < 0.01$) at USSES. According to these estimates, a ewe with a SCC of 1,000,000 cells mL⁻¹ ($LSCC_A = 6.0$) is expected to wean 12.8 and 14.8 kg less total lamb than a ewe with a SCC of 100,000 cells mL⁻¹ ($LSCC_A = 5.0$) at MSU (early $LSCC_A$) and USSES (peak $LSCC_A$), respectively.

Gross et al. (1978) and Keisler et al. (1992) reported that ewe SCC class had no effect on lamb weaning weights. Dam SCM reduced lamb ADG in only 1 of a 3 yr in Ahmad et al. (1992), during which lambs reared by healthy ewes gained 10.7% more than those raised by infected ewes. These observational reports could not account for non-milk sources of lamb intake. In a controlled experiment, Fthenakis and Jones (1990) found that ewes induced with SCM produced 37% less milk than healthy ewes and their lambs weighed 8.8% less and consumed 25% more supplemental feed.

IMPLICATIONS

Clinical mastitis contributes to increased culling rates and lost revenue in Western sheep production, but the importance of ovine SCM is less clear. The SCC threshold which diagnoses SCM in sheep is not well-defined, with suggested cutoffs varying from 300,000 to 1,000,000 cells • mL⁻¹ (Fthenakis et al., 1991; González-Rodríguez et al., 1995). The percentage of ewes with SCC > 500,000 cells • mL⁻¹ was 19% and 17% at MSU and USSES, respectively. On average, such ewes weaned 6.4 and 22.2 kg less lamb, a lost revenue of $31 and $106 per ewe ($4.77 kg⁻¹ feeder lamb; USDA, 2018). While mean serum Zn concentration of LH ewes was considered adequate (Herdt et al., 2000), reliance upon Zn deficient forages in the months leading up to parturition and after weaning may predispose ewes to intramammary infection. Strategic trace mineral supplementation and other management strategies to reduce the prevalence of ewe mastitis in Western rangeland management systems warrants further investigation.

LITERATURE CITED

Ahmad, G., L. L. Timms, D. G. Morrical, and P. O. Brackelsberg. 1992. Dynamics and significance of ovine subclinical intramammary infections and their effects on lamb performance. Sheep Res. J. 8(1):25–29.

Arsenault, J., P. Dubreuil, R. Higgins, and D. Bélanger. 2008. Risk factors and impacts of clinical and subclinical mastitis in commercial meat-producing sheep flocks in Quebec, Canada. Prev. Vet. Med. 87:373–393. doi:10.1016/j.prevetmed.2008.05.006.

Cooper, S., S. J. Huntley, R. Crump, F. Lovatt, and L. E. Green. 2016. A cross-sectional study of 329 farms in England to identify risk factors for ovine clinical mastitis. Prev. Vet. Med. 125:89–98. doi:10.1016/j.prevetmed.2016.01.012.

Fthenakis, G. C., E. T. el-Masannat, J. M. Booth, and J. E. Jones. 1991. Somatic cell counts of ewes' milk. Br. Vet. J. 147:575–581. doi:10.1016/0007-1935(91)90029-M.

Fthenakis, G. C., and J. E. Jones. 1990. The effect of experimentally induced subclinical mastitis on milk yield of ewes and on the growth of lambs. Br. Vet. J. 146:43–49. doi:10.1016/0007-1935(90)90075-E.

González-Rodríguez, M. C., C. Gonzalo, F. San Primitivo, and P. Cármenes. 1995. Relationship between somatic cell count and intramammary infection of the half udder in dairy ewes. J. Dairy Sci. 78:2753–2759. doi:10.3168/jds.S0022-0302(95)76906-5

Grant, C., E. M. Smith, and L. E. Green. 2016. A longitudinal study of factors associated with acute and chronic mastitis and their impact on lamb growth rate in 10 suckler sheep flocks in Great Britain. Prev. Vet. Med. 127:27–36. doi:10.1016/j.prevetmed.2016.03.002.

Gross, S. J., E. J. Pollak, J. G. Anderson, and D. T. Torell. 1978. Incidence and importance of subclinical mastitis in sheep. J. Anim. Sci. 46:1–8. doi:10.2527/jas1978.4611

Herdt, T. H., W. Rumbeiha, and W. E. Braselton. 2000. The use of blood analyses to evaluate mineral status in livestock. Vet. Clin. North Am. Food Anim. Pract. 16:423–444. doi:10.1016/S0749-0720(15)30078-5

Keisler, D. H., M. L. Andrews, and R. J. Moffatt. 1992. Subclinical mastitis in ewes and its effect on lamb performance. J. Anim. Sci. 70:1677–1681. doi:10.2527/1992.7061677x

Menzies, P. I., and S. Z. Ramanoon. 2001. Mastitis of sheep and goats. Vet. Clin. North Am. Food Anim. Pract. 17:333–358. doi:10.1016/S0749-0720(15)30032-3

Moroni, P., G. Pisoni, G. Varisco, and P. Boettcher. 2007. Effect of intramammary infection in bergamasca meat sheep on milk parameters and lamb growth. J. Dairy Res. 74:340–344. doi:10.1017/S0022029907002506.

O'Rourke, D. 2009. Nutrition and udder health in dairy cows: a review. Irish Vet. J. 62:15–20. doi:10.1186/2046-0481-62-S4-S15

Quinlivan, T. D. 1968. Survey observations on ovine mastitis in New Zealand stud Romney flocks. 1. The incidence of ovine mastitis. N. Z. Vet. J. 16:149–153. doi:10.1080/00480169.1968.33765.

Saianda, I. M., C. M. V. Bettencourt, M. C. Queiroga, G. Ferreira-Dias, and C. L. Vilela. 2007. Effect of dietary zinc upon in vitro bacterial adherence to ovine mammary epithelium. Rev. Med. Vet. 158(7):362–366.

USDA. 2018. Weekly national lamb market summary. Vol. 25 No. 10.

USDA APHIS. 2012. Sheep 2011 Part II: reference of marketing and death loss on U.S. sheep operations.

Waage, S., and S. Vatn. 2008. Individual animal risk factors for clinical mastitis in meat sheep in Norway. Prev. Vet. Med. 87:229–243. doi:10.1016/j.prevetmed.2008.04.002

Watkins, G. H., A. R. Burriel, and J. E. Jones. 1991. A field investigation of subclinical mastitis in sheep in southern England. Br. Vet. J. 147:413–420. doi:10.1016/0007-1935(91)90083-Y