MASTITIS IN SMALL RUMINANTS

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Abstract

This paper reviews the epidemiology, etiologies, risk factors and preventive management strategies used to minimize mastitis in dairy sheep and dairy goats. Clinical mastitis typically occurs in <5% of lactating does and ewes but subclinical mastitis may occur in up to 15-30% of animals. Somatic cell counts (SCC) of milking ewes can be used to define subclinical mastitis and a threshold of about 200,000 to 400,000 cells/ml will accurately identify most infected ewes. Interpretation of SCC values of milking goats is complicated by the presence of cytoplasmic particles in milk. However, intramammary infection in milking does results in increased SCC values which must be interpreted based on intervening physiological factors such as stage of lactation, parity and estrus. Milking management and dry off treatment are important strategies for producers to adopt to minimize the development of new IMI.

Introduction

In the U.S., dairy products made with milk of small ruminants are considered to be specialty foods that are generally purchased by consumers who have little exposure to the realities of modern agriculture. Consumers assume that they are purchasing high quality, safe dairy products produced by healthy animals and harvested under hygienic conditions. Mastitis is

an important disease of dairy animals because it reduces animal wellbeing and the quantity and quality of the milk that is produced. Mastitis is also important because it reduces production efficiency and farm profitability. Understanding and preventing mastitis is essential to achieving successful management of dairy farms and veterinarians are an important resource for small ruminant dairy producers. The objective of this paper is to review concepts related to mastitis and milk quality in small ruminants that are used for dairy production.

Background Information for Both Species

Definitions

Mastitis is a bacterial disease that occurs in several different forms. *Clinical mastitis* is the term used for bacterial infections of the mammary gland that present with obvious symptoms. Signs of clinical mastitis may include abnormal appearance of milk (presence of clots or serum), swelling, redness or necrosis of one or more half udders, or severe systemic symptoms such as anorexia, fever or agalactia. *Subclinical mastitis* is characterized by inflammation of the udder detected by enumeration of inflammatory cells in the milk. By definition, the appearance of milk obtained from animals with subclinical mastitis is not altered and testing of the milk is required to identify affected animals.

Subclinical mastitis occurs when a mastitis pathogen infects one or more udder halves but does not cause enough disruption of secretory tissue to result in visibly abnormal milk. In these instances, the immune system of the animal responds to the bacterial invasion by sending white blood cells (WBC) to the inflamed mammary gland. The migration of inflammatory cells to the affected gland is in response to bacterial infection but because the inflammatory cells are part of the immune response and are active in engulfing and destroying bacteria, pathogens are not

always present in the milk in detectable quantities. Somatic cell counts (SCC) measure the number of WBC and udder epithelial cells that are present in milk and in dairy sheep and cows are an indication of a healthy immune response to infection. In both dairy sheep and dairy cows, a significant increase in somatic cells occurs almost exclusively in response to bacterial infection of the mammary gland. The SCC response in dairy goats is not as specific to infection and thus different criteria for interpretation are necessary for this species.

Mastitis causing bacteria are often categorized as "*contagious*" if the source is thought to be infected milk that came from a gland infected with subclinical mastitis pathogens or "*environmental*" if the bacteria are considered as opportunistic pathogens that normally reside in the environment of the animals. However, this delineation is not as clear for small ruminants as it is for dairy cattle. For example, in milking ewes the likely source of CNS is skin on the teats or inner legs (this skin often contacts teats) but because many CNS infections become long term chronic infections, it is possible that CNS could be shed in milk from an infected udder and then spread via the milking equipment to other ewes. Thus, the source of mastitis pathogens in small ruminants should not be assumed based simply on behavior of these pathogens in dairy cows.

Regulations

In the U.S., all commercial dairy producers must have state licenses and Grade A dairy products produced from cattle, sheep, goats or buffalos are regulated based on the Pasteurized Milk Ordinance (PMO; www.fda.gov). The PMO requires monthly testing of bulk tank SCC and regulatory action is taken when 2 of 4 monthly bulk tank SCC values exceed the species specific regulatory limit. The dairy license is suspended when the threshold is exceeded for 3 of 5 tests. For milk produced by dairy cows, buffalos and sheep the bulk tank SCC limit is

currently 750,000 cells/ml. As of 2009, the bulk tank SCC limit for goat milk is 1,500,000 cells/ml. For all species, the bacterial count of bulk milk cannot exceed 100,000 cfu/ml.

Impact of Subclinical Mastitis on Product Quality & Yield.

In 2 separate studies, an Israeli research group has compared milk production and milk composition in ewes (Leitner et al., 2004a) and does (Leitner et al., 2004b) with one healthy half udder and one infected half udder (Table 1). All of the subclinical infections were induced by intramammary infusion of coagulase-negative Staphylococci (CNS).

Table 1. Impact of subclinical mastitis caused by CNS on milk yield and milk characteristics.

	Ewes (Leitner, et al., 2004a)		Goats (Leitner et al., 2004b)	
	Healthy Half Udder	Infected Half Udder	Healthy Half Udder	Infected Half Udder
Milk Yield/milking	1.7 lb (0.76 kg)	0.79 lb (0.36 kg)	2.2 lbs (0.98 kg)	1.5 lbs (0.69 kg)
SCC (cells/mL)	311,000	4,999,000	417,000	1,750,000
Fat g/L	64.9	61.7	38.9	38.8
Protein g/L	58.5	53.5	34.2	35.0
Casein (mg/mL)	45.9	40.5	28.1	28.2
Whey (g/L)	11.9	12.8	6.1	6.8
Curd Yield	30.1 g/milking	13.9 g/milking	232 g/L	208 g/L
Clotting time (sec)	413	909	167	295

A large impact of subclinical infection on milk yield was identified and the milk produced in the affected half udders was of much poorer quality and resulted in reduced curd yield. A separate

study investigating the effect of SCC on characteristics of semisoft goat cheese failed to demonstrate differences in milk composition based on high SCC but did indicate lower sensory scores and inferior textures in cheeses made with high SCC milk (Chen et al., 2010).

Species Differences in Cellular Populations of Milk

Subclinical mastitis is generally defined by the migration of neutrophils into the mammary gland in response to bacterial infection. This response occurs in all dairy species but the magnitude of the response and the distribution of cells types in the healthy mammary gland differs considerably (Table 2).

Table 2. Distribution of cell types in milk from healthy and infected mammary glands (adapted from data in Paape et al., 2001; Paape and Capuco, 1997; Leitner, et al., 2000).

	State of Gland	Goat Milk	Sheep Milk	Cow Milk
PMN %	Healthy	45-74%	2-28%	2-30%
	Subclinical Mastitis	71-86%	50-90%	40-90%
Macrophage %	Healthy	15-41%	46-84%	13-88%
	Subclinical Mastitis	8-18%		4-17%
Lymphocyte %	Healthy	9-20%	11-20%	10-27%
	Subclinical Mastitis	5-11%		
Epithelial Cells %	Healthy	1-6%	1-2%	1-2%
SCC (x1,000)	Healthy	270-2,000	185	40-80
	Subclinical Mastitis	650-4,200	1,445	250-3,000

The proportion of neutrophils (PMN) and the number of cytoplasmic particles present in milk are very different in milk produced by goats as compared to milk produced by ewes or cows (Table 2). Part of this difference is generally attributed to different milk secretion mechanisms. Both goats and sheep are thought to produce milk using a largely apocrine process where the apical portion of the secretory cell is excreted into the milk. In spite of similar secretory processes, the number of cytoplasmic particles found in milk obtained from both healthy and infected glands is approximately 10-20 folder greater for goats (about 70,000 – 300,000 cells/ml) as compared to cytoplasmic particles found in sheep milk (about 15,000 cells/ml) (Paape et al. 2001). In contrast, very few cytoplasmic particles are found in cow's milk which is generally thought to be secreted via a merocrine process. The large number of cytoplasmic particles necessitates the use of DNA specific counting mechanisms to accurately enumerate somatic cells in goat milk

Determining the cause of mastitis

There is no way to diagnose the cause of mastitis based on the appearance of the milk, gland or animal. The only way to determine the cause is to submit an aseptically obtained milk sample to a laboratory for microbiological examination. When proper laboratory procedures are used, the recovery of bacteria from milk samples is highly specific for mastitis. However, microbiological examination of milk obtained from glands affected with clinical or subclinical mastitis is not very sensitive. Bacteria are often shed cyclically or in sparsely and it is important to recognize that laboratory methods used for the recovery of mastitis pathogens are not perfect. The failure to recover bacteria from a milk sample obtained from a gland with high SCC does not necessarily mean that bacteria are not the causative agent for mastitis. When a single milk sample is obtained from dairy cattle exhibiting clinical or subclinical mastitis, approximately 35-50% of milk samples will be culture negative (Makovec and Ruegg, 2003) and

it is likely that similar proportion of milk samples obtained from dairy ewes will be falsely negative. If the SCC of an ewe has chronically increased SCC but is culture negative the best strategy is to assume that the udder remains infected. The identification of subclinical mastitis infections in goats is more complex and is discussed later in the paper.

Mastitis in Dairy Sheep

Epidemiology of Clinical and Subclinical Mastitis. In North America, most sheep are kept for production of meat and most research literature discusses symptoms of mastitis occurring in ewes that are nursing lambs. In this population, only severe clinical mastitis is likely to be diagnosed. This lack of emphasis on milking ewes has led to an overemphasis on the occurrence of clinical mastitis and a lack of appreciation for subclinical mastitis. While there are no national studies assessing the incidence of clinical mastitis in dairy ewes milked in the U.S., based on research in other regions, clinical mastitis is thought to occur in less than 5% of ewes per year (Bergonier et al., 2003). The experience of the University of Wisconsin milking flock at Spooner is typical. This flock consists of about 250 crossbred milking ewes. Since, 2008, the UW Madison milking flock has experienced clinical mastitis in 1-3% of the ewes each year and in almost all instances, the shepherd has elected to cull (rather than treat) these animals.

Ewes that are affected with subclinical mastitis produce milk that appears visually identical to milk produced from healthy ewes but the milk is produced from glands that have been damaged by bacteria and thus produce less quantities of lower quality milk. While little U.S. data is available to define the prevalence of subclinical mastitis, researchers believe that up to 30% of ewes in some flocks may be affected. Using DHIA testing data, collected during the

lactation periods of 2008, 2009 and 2010, each month about 15-20% of the ewes in the UW flock had SCC >400,000 cells/mL and the prevalence of increased SCC was somewhat influenced by stage of lactation and parity.

Causes of Mastitis in Dairy Ewes. In almost all instances, mastitis is caused by a bacterial infection. The infection occurs when teats are exposed to enough pathogenic bacteria to overwhelm teat end defenses. Almost any bacteria can theoretically cause mastitis but several groups of pathogens are commonly obtained from milk samples of affected ewes. While most bacteria can cause both clinical and subclinical mastitis, *Staphylococcus aureus, Pasteurella hemolytica* and various yeasts and molds are the organisms that have been frequently reported to be recovered from milk samples of ewes affected with clinical symptoms. Bluebag (clinical mastitis with a hard, cold swollen udder) is typically caused by either *Pasteurella hemolytica* or *Staph aureus*. Coagulase-negative staphylococci are considered to be minor pathogens in dairy cows but behave as major pathogens in dairy sheep and have been frequently reported to be the most commonly isolated pathogens recovered from cases of subclinical mastitis of dairy ewes (Fthenakis, 1994; Burriel, 1997; Lafi et al., 1998; Ariznabarreta et al., 2002; Gonzalo et al., 2002; Hariharan et al., 2004). Subclinical infection caused by CNS and other mammary pathogens have been associated with increased SCC (Pengov, 2001; Ariznabarreta et al., 2002). Other pathogens that are typically recovered from subclinical mastitis infections in ewes include *Corynebacterium* spp., Yeast, *Streptococcus* spp., *Enterobacteria* spp. and *Staphylococcus aureus*. Yeast and mold infections in ewes are often associated with non-hygienic administration of intramammary treatments and great care must be taken when these treatments are used (Spanu, et al., 2008).

154 The incidence of intramammary infection in dairy ewes is typically greatest in early
155 lactation and ewes may be subclinically infected in the immediate postpartum period but
156 apparently healthy at later periods (Table 2). However, ewes with subclinical CNS infection are
157 much more likely to remain as chronic subclinical infections as compared to other pathogens
158 (except for yeast infections).

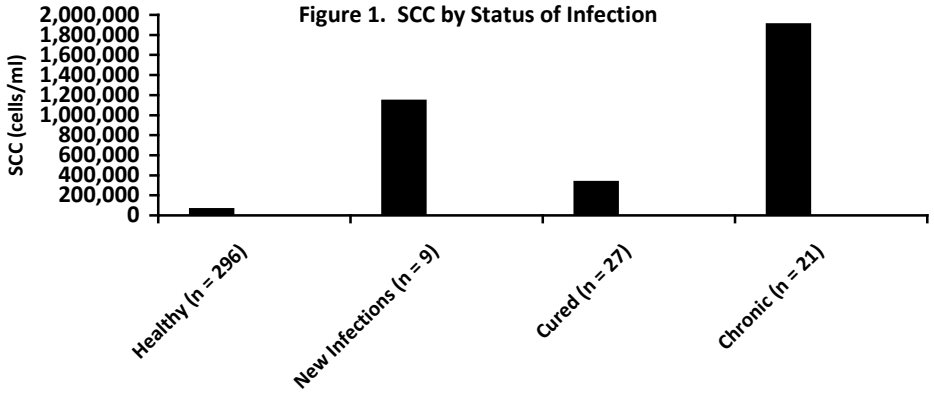
159 Table 2. Outcomes of half udder milk samples (n = 390) obtained in the postpartum period and
160 14-21 days post lambing in the UW Spooner dairy research flock after lambing in 2008.

	Outcome at 14-21 days post lambing			
At Lambing	No Growth Both sampling periods	No bacteria recovered (cured)	Same bacteria recovered (chronic)	Different bacteria recovered (new infection)
No Growth (n = 299; 77%)	289 (97%)	Not applicable (NA)	NA	10 (3%)
CNS (n = 35; 9%)	NA	14 (40%)	20 (57%)	1 (3%)
Corynebacterium spp (n = 12; 3%)	NA	10 (83%)	0	2 (17%)
Other (n = 10; 3%)	NA	10 (100%)	0	0
Enterobacteria (n = 7; 2%)	NA	4 (57%)	1 (14%)	2 (29%)
Mixed (n = 6; 2%)	NA	5 (83%)	0	1 (17%)
Bacillus (n = 5; 1%)	NA	4 (80%)	0	1 (20%)
Yeast (n = 12; 3%)	NA	1 (8%)	11 (92%)	0

161

In rare instances, the lentivirus that causes Ovine Progressive Pneumonia (OPP) has been associated with mastitis in sheep (Deng et al., 1986) but there is no evidence that this virus has influence on SCC of sheep milk (Bergonier et al., 2003). Mammary gland symptoms are associated with lesions in secretory tissue. While it is known that this virus has an affinity for mammary glands, the disease is a slowly progressive disease that results in weight loss, greatly reduced milk production and other symptoms that make it unlikely to become widespread in flocks that are used for dairy production.

Somatic Cell Counts and Subclinical Mastitis. The types of cells and proportions of cells present in sheep milk are more similar to dairy cows rather than goats and standard methods used to count somatic cells in cows' milk are considered accurate for counting somatic cells in ewes' milk. Evaluation of SCC data is considered to be an effective tool for diagnosing intramammary infections in dairy sheep (Gonzalo et al., 1994; Gonzáles-Rodríguez et al., 1995; Pengov, 2001). In an uninfected half-udder, the SCC count is generally lower than 200,000 to 400,000 cells/ml (Bergonier, et al., 2003). Higher counts are almost always associated with bacterial infections and indicate the presence of subclinical mastitis. Many healthy half-udders have SCC values that are less than 100,000 cells/ml (Pengov, 2001). The SCC of half-udder milk samples, by status of intramammary infection (based on microbiological analysis) in early lactation for samples obtained from the UW Spooner Research Flock in spring 2008 is shown in Figure 1. The data demonstrates characteristic responses with SCC values least for uninfected glands, modestly increased SCC values for glands that were responding to previous infections and increased SCC values for glands with either new IMI or chronic infections.



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184 Individual gland SCC values increase in response to IMI in ewes and thus bulk tank SCC
185 values are an indication of the quality of milk and increase when the prevalence of subclinical
186 mastitis increases. Dairy sheep producers should monitor bulk tank SCC and manage the flock
187 to maintain SCC less than 300,000 cells/ml. Ewes with even mild chronic subclinical mastitis
188 infections can be expected to produce about 5% less milk as compared to ewes with healthy
189 udders (Spanu, et al., 2008). The impact of SCC on milk yield was evaluated by comparing
190 monthly SCC data (n = 4402 monthly values) obtained from ewes (n = 495) in the UW Madison
191 milking sheep flock during 2008-2010. After adjusting for parity, month in milk, and year, a
192 significant impact of SCC on milk yield was observed (Ruegg, unpublished). Monthly test day
193 milk yields were 3.4 lbs (1.54 kg) for months when the SCC was <400,000 cells/ml in contrast to
194 3.1 lbs (1.4 kg) for months when the SCC had been increased for 2 consecutive months. Milk
195 yields for ewes with newly increased SCC (SCC < 400,000 cells/mL in previous month) or
196 newly cured SCC (SCC >400,000 cells/mL in previous month) were intermediate (about 3.3 lbs;
197 1.48 kg).

198 Management of milk quality is impossible without knowing how many ewes are affected
199 with subclinical mastitis. Dairy sheep producers should feel confident in using SCC values to

identify ewes with subclinical mastitis. Somatic cell counts in ewes are quite specific for infection. Ewes with a single half-udder infection will normally have high SCC in the infected half udder and low SCC in the healthy half udder. For example, in 39 ewes with intramammary infections in a single half udder, the SCC of the healthy half udders was 195,000 cells/ml as compared to 1,329,820 cells/ml in the infected halves (Ruegg, unpublished data). Using this data, half-udders that were infected were 6 times more likely to have SCC >400,000 cells/ml as compared to half-udders that were healthy. This data indicates that the CMT paddle or other ewe-side SCC tests (such as the PortaSCC or the Direct Cell Counter (DCC, Delaval)) can be used to help producers identify subclinical infections.

Dairy shepherds should consider monitoring production and SCC of each ewe on a monthly basis using a DHIA service. If DHIA is not available, producers should use a monthly individual ewe SCC test such as CMT, PortaSCC or DCC to assess udder health each month. Monthly SCC data can be used to select ewes that should have milk submitted for culturing or to identify chronically infected ewes for interventions such as treatment or culling, target specific ewes for intramammary dry off therapy or identify risk factors for mastitis such as stage of lactation, housing or milking management. When using individual ewe or half-udder SCC values, a threshold of 200,000-400,000 cells/ml should be used to identify ewes that have subclinical mastitis. Care must be taken to accurately use the CMT to identify ewes with subclinical mastitis. The CMT is scored using a 5 point scale (negative, trace, 1,2,3). Milk containing 200,000-400,000 cells/ml would result in CMT scores of “trace.” Trace CMT scores are difficult to read and the expected appearance of the CMT reaction is defined as: “slight precipitate, best seen by tipping, disappears with continued movement.”

Risk Factors for Mastitis in Ewes

Risk factors for subclinical mastitis are not well defined for intensively managed milking sheep in North America. European research in Mediterranean countries has indicated that most of the variation in mastitis is associated with differences in herd management (Gonzalo et al, 2005). In the same study, higher producing breeds were at greater risk of mastitis and the use of dry off treatment resulted in less mastitis (Gonzalo et al., 2005). Mastitis in milking sheep is usually caused by bacteria that live on skin (such as CNS), and it is sensible to conclude that practices that reduce exposure of teat ends to bacteria should result in reduced prevalence of mastitis. Udders, inner legs and tails (if left long) should be as clean as possible. Pastures and other housing for ewes should be managed to provide a clean and dry place for all ewes to rest. Milking equipment should be clean, well maintained and provide stable teat end vacuum. Teat cup liners should be observed for wear and replaced in accordance with the manufacturers recommendations. Practices that improve udder hygiene and reduce teat exposure to bacteria are likely to result in less mastitis. For example, all teats of milking ewes should be disinfected post-milking using a commercially available teat dip product. Mastitis can spread from infected ewes to healthy ewes if bacteria present in milk from a subclinically infected half udder are allowed to contact healthy teats. It is important to identify chronically infected ewes and either cull or milk them last to reduce the risk of infecting healthy ewes. It may also be important to review nutritional management. While there is no research data examining the effect of selenium or vitamin E deficiency on the incidence of mastitis in sheep, these nutrients are known to be important in ensuring immune function and deficiencies have been associated with increased mastitis in dairy cattle goats (Sánchez et al., 2007).

Treatment & Prevention of Mastitis

Ewes that develop clinical mastitis are often seriously ill and should be treated immediately according to protocols that have been developed in consultation with the flock veterinarian. Most treatments for severe clinical mastitis are administered systemically and the ewe may require supportive therapy. There are no antibiotic compounds that are approved for treatment or prevention of mastitis in milking sheep. Drugs that are used for these purposes are considered by the FDA to be administered in an "extralabel" manner and this usage must be prescribed and supervised by a licensed veterinarian. The administration of a drug that is approved for treatment of another sheep disease (such as the use of ceftiofur for treatment of pneumonia) to treat mastitis is also considered as extralabel usage. It is important to recognize that systemic administration of ceftiofur will not achieve effective inhibitory levels in the mammary gland of cows, sheep or goats.

There is virtually no research literature that describes efficacy or economics of treatment during the lactation period of ewes affected with subclinical mastitis. Most subclinical mastitis in dairy sheep is caused by CNS and the behavior of CNS in sheep is uniquely different than the behavior of CNS in dairy cows. Thus, extrapolation of recommendations developed for CNS infections in dairy cows is probably not appropriate. Clinical trials are needed to determine if intramammary treatments result in economically beneficial outcomes in subclinically affected lactating dairy sheep. The use of intramammary dry off treatment has been shown to positively influence milk yield and SCC in the subsequent lactation and is recommended (Gonzalo, et al., 2004; Spanu, et al., 2011). However, administration of intramammary treatments does increase the risk of mastitis caused by yeast bacteria and selective dry off treatment can be recommended

in flocks that have a relatively low prevalence of subclinically affected ewes. Milk samples obtained from ewes with 3 or more monthly somatic cell counts ≥ 400,000 cells/mL in the previous lactation were 6 to 8 times more likely to be positive for mastitis pathogens in the next lactation as compared to milk samples obtained from ewes with SCC below that threshold and that threshold may be appropriate to identify ewes that should receive dry off treatment (Spanu, 2009).

Additional management strategies that may be helpful to control subclinical mastitis include the use of post-milking teat disinfection, culling of chronically infected ewes (identified by several months of SCC >400,000 cells/ml) and in some instances the use of pre-milking teat disinfection.

Mastitis in Dairy Goats

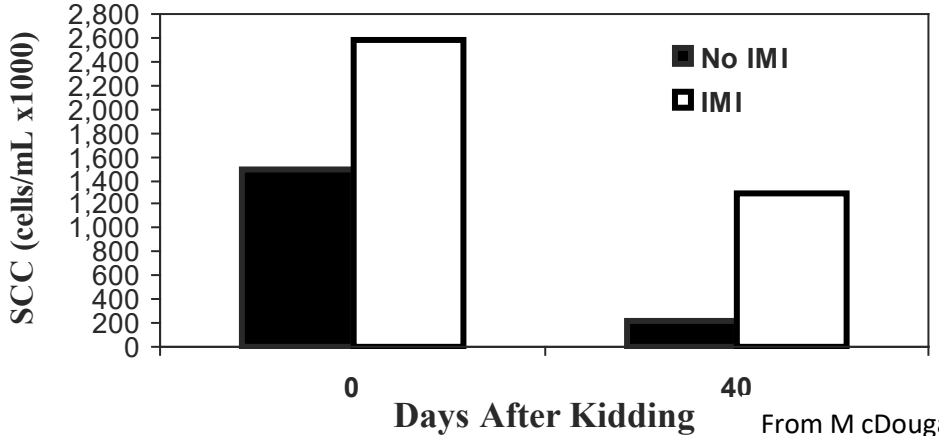
Epidemiology of Clinical and Subclinical Mastitis. Similar to dairy ewes, the incidence of clinical mastitis is generally reported to be <5% of lactating does per year (Bergonier et al., 2003). A recent study that surveyed about 90% of all goat dairy farms in Holland (about 300 farms), reported that the annual incidence of clinical mastitis was 2% per year and about two-thirds of the farms culled the majority of affected does (rather than treat them) (Koop et al., 2009). Of 19 goat dairy farms visited as part of an observational study in Wisconsin in 2009, farmers reported 1.4 cases of clinical mastitis had occurred in the previous 60 days (1% incidence) and of that 66% were treated (Ruegg, unpublished). One interesting study conducted in Spain, linked the incidence of clinical mastitis to selenium deficiency (Sánchez et al., 2007). Spanish researchers reported that for does consuming a deficient diet, the incidence of clinical

mastitis was 3.8% and 15.4% for does that had been treated with slow release barium selenite or were enrolled in a non-supplemented control group, respectively.

There are neither national surveys nor comprehensive reviews that describe the prevalence of subclinical mastitis in dairy goats in the US or Canada. Review of existing data about the prevalence of subclinical infection is further complicated by the lack of a uniform SCC threshold and the influence of intervening factors (such as estrus) on SCC. When recovery of bacteria from milk samples is used as the gold standard to identify subclinical mastitis, several studies have indicated that half-udder prevalence of subclinical mastitis varies between about 15-40% (adapted from Table 1, Koop et al., 2011). When using SCC threshold of 500,000 cells/ml as a threshold for defining subclinical mastitis, researchers have estimated sensitivity (probability of recovery of pathogen when the SCC is > threshold) as ranging from 0.69-0.90 and specificity (probability of not recovering pathogen when the SCC is < threshold) of about 0.35-0.77 (from Table 1, Koop et.al., 2011). Equivalent values for dairy cows, using a SCC threshold of 200,000 cells/ml have been estimated to be 0.75 and 0.9% for SE and SP, respectively (Schepers et al., 1997). In 2009, the distribution of individual doe SCC for 5 WI dairy goat farms (n = 1,011 goats) sampled in mid-summer was: 25% (<200,000 cells/mL), 48% (201,000-800,000); 15% (801,000 – 1,600,000) and 12% (>1,600,000). In an analysis of 29,045 test day milk samples obtained from >6,000 does located in 38 US states, 50% of the samples were <400,000 cells/mL, 31% of samples exceeded 750,000 cells/mL and 24% of the samples exceeded 1,000,000 cells/mL (Zhang et al., available online: http://www.luresext.edu). While some of these high SCC values are likely associated with physiological changes, some reflect IMI and it is likely that the prevalence of subclinical mastitis in many goat herds is somewhere around 20-30%.

Causes of Mastitis in Goats. Similar to dairy sheep, researchers have consistently reported that CNS are responsible for the greatest proportion of subclinical mastitis infections occurring in this species (Bergonier et al., 2003; McDougall et al., 2002, White and Hinckley, 1999). Infection with CNS are especially prevalent in goats at parturition with recovery of CNS from up to 17% of goats, reported (McDougall et al., 2002). Similar to ewes, the early lactation spontaneous cure rate is only about 50% for IMI caused by CNS and up to 25% of does may remain infected, 6 weeks after parturition (McDougall et al., 2002). Researchers have noted that SCC values of infected udder halves were always significantly greater than SCC values of healthy udders (Figure 2; McDougall et al., 2002). Other pathogens that are frequently recovered from goats with subclinical mastitis include Corynebacteria spp., Streptococci spp. and *Staphylococcus aureus*. The relationship between lentiviral infections (CAEV) and SCC has been reviewed (Bergonier et al., 2003; Paape et al., 2003) and herds with greater prevalence of seropositive does have been shown to have greater SCC values. However, this relationship is considered weak and may have been a result of immunosuppression caused by CAEV infection.

Figure 2. SCC for Formilk of Goats

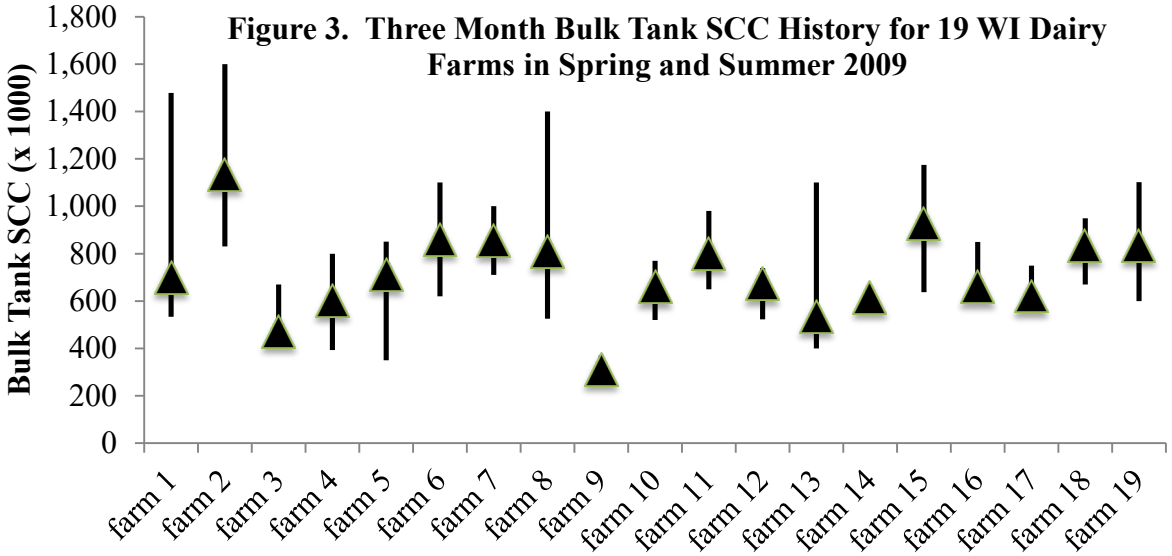


From M cDougall et al., 2002

Clinical mastitis in goats is often associated with infection by *Staphylococcus aureus*, Streptococci spp. or miscellaneous pathogens such as yeast. In many regions of the world, IMI

are associated with infection by a variety of Mycoplasma spp. and milk samples obtained from goats with chronically increased SCC should be submitted for Mycoplasma culture.

Factors influencing SCC in Goats. Bulk milk SCC values vary considerably among goat herds (Figure 3, Ruegg unpublished) and while factors other than mastitis influence SCC of goats, the prevalence of subclinical mastitis is an important determinant of bulk tank values.



Enumeration of SCC in goat milk must be performed using DNA specific methods such as fluoro-optical electronic cell counters such as Fossomatic cell counters used in DHIA centers or the Direct Cell Counter (Delaval) used for individual animal samples. When direct microscopic counts are performed as a gold standard, the slides must be stained with Pyronin Y-methyl green stain (Paape et al., 2001). The CMT test is based on reaction of the detergent with DNA in cells and is also considered accurate as are other individual animal's tests such as the PortaSCC (PortaCheck).

When enumeration of SCC in goat milk is properly performed, intramammary infection is a well-known cause of increased SCC but the threshold used to determine infection must be

determined relative to stage of lactation (Bergonier et al., 2003). Milk samples obtained from infected udder halves generally exhibit SCC values >500,000 cells/mL (first 90 DIM) and >1,000,000 cells/mL (later stages of lactation). Important factors that must be considered when evaluating SCC of goats include: parity, stage of lactation, breed and estrus (Bergonier et al., 2003; McDougall and Voermans, 2002; Paape et al., 2007). Paape et al., (2007) indicated that parity is an important determinant of SCC in goats and reported SCC values at 15 DIM of about 200,000 cells/mL (1st parity) and 250,000 cells/ml for 1st lactation and 5th lactation does, respectively. Paape et al., (2207) indicated that larger differences were observed in later lactation and reported SCC values at 285 DIM of about 500,000 cells/mL (1st parity) and 1,150,000 cells/ml for 1st lactation and 5th lactation does, respectively. Several researchers have reported that SCC values vary by breed with milk samples obtained from Toggenburgs recording the greatest values (Paape et al., 2007). Reasons for the effect of breed are unknown and may be related to either physiological differences or perhaps to differences in resistance to mastitis. Many goat producers have indicated that SCC values increased after does are exposed to bucks so a relationship between estrus and increased SCC has long been postulated. The ability of estrus to stimulate increased SCC in the absence of IMI has been demonstrated in a controlled study using induced estrus (McDougall and Voermans, 2002). In one part of the trial, the day after inducing estrus, SCC values were 1,778,000 cells/mL for does in estrus versus 363,000 cells/mL for does in the control group (both values have been converted from the reported log values). These physiological increases were not associated with IMI or with decreased milk production but the mechanism behind the increase was not elucidated. Overall, while several non-infectious causes for increased SCC are observed in goats, intramammary infection remains an important cause of increased SCC. While it is more complex to use SCC values to investigate

mastitis problems in goats, the large variation observed among herds indicates that control of mastitis can result in lower bulk tank SCC and producers should work to understand the factors that influence SCC in their herd.

Risk Factors for Mastitis in Dairy Goats. Most research related to dairy goat mastitis has focused on defining SCC thresholds and there is very little research that has been conducted to elucidate risk factors for the development of mastitis in dairy goats. For most herds, Staphylococci spp. cause the greatest amount of mastitis. When CNS is the prevalent mastitis organism, control procedures should be focused on premilking hygiene, use of best management practices for milking and maintaining healthy teat ends. In one preliminary study that involved 16 goat farms in mid-lactation, (Ruegg, unpublished) teat condition was scored on a 4 –pt scale (1=smooth; 4 = very rough) and considerable variation was found among farms. Of 16 farms where teats were observed, no does with teat scores of 4 where found on 4 farms whereas >20% of does were observed to have very rough teats on another 4 farms. A linear relationship between the amount of time that the milking unit was attached and the percent of teats with rough teat ends was observed. While the study was too small to be able to determine causal factors, intriguing relationships between teat score and milking characteristics (such as pulsation rate and ratio, the liner type and the use of a claw milking unit) deserve more research. Herds that are experiencing mastitis problems caused by *Staphylococcus aureus* should focus on reducing the prevalence of infected animals and identifying and segregating infected animals.

Treatment and Prevention of Mastitis in Dairy Goats. As all mastitis treatments involve extralabel drug usage, treatment of clinical mastitis should be performed using protocols developed by the veterinary practitioner who has a valid veterinary client patient relationship.

Treatment of systemically ill animals should be focused on supportive care and appropriate antimicrobial therapy. Treatment of animals with local signs of clinical mastitis generally involve administration of commercial intramammary products and should be accompanied by microbiological assessments of at least some cases. Treatment of subclinical mastitis is unlikely to be pursued by most farms and aggressive culling of affected animals has been shown to be associated with herds that have lower bulk tank SCC (Koop et al., 2009). At least one study has demonstrated that treatment of subclinical mastitis in early lactation based on CMT resulted in increased bacteriological cure but was not economically beneficial (McDougall, et al., 2010). Thus, treatment of subclinical infections during lactation is not currently recommended. However, the use of dry off therapy has been shown to effectively cure CNS infections and result in lower SCC in early lactation (Poutrel, et al., 1997). As with sheep, producers should be taught to use extreme care when disinfecting teat ends to prevent the iatrogenic development of IMI caused by yeast.

As in all dairy species, exposure of the teat end to bacteria is the mechanism for development of mastitis and control programs are based on principles that improve hygiene and reduce exposure to potential pathogens. The prevalence of subclinical mastitis has been shown to be decreased for goat herds that practice good teat dipping and premilking teat sanitation (Contreras, et al., 1999).

Conclusions

Mastitis is an important disease of small ruminants used in dairy production and the prevalence of mastitis varies depending on management. Most mastitis occurs in a subclinical form and producers who do not routinely measure individual animal SCC will not be able to

412 determine the impact of subclinical mastitis on production and milk quality. Most subclinical
413 mastitis in small ruminants is caused by CNS which should be considered as major mastitis
414 pathogens in these species. Prevention of infection is the key to control of mastitis and good
415 hygienic housing and milking practices are a necessity to minimize the impact of this disease.

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