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Mammary Gland

Herd average milk production for Holstein cows in US corn silage based systems is 65-85lbs per day. At the peak of lactation cows may produce 100-140 lbs of milk per day. During milking up to a quart of milk/minute passes through each teat end.

Anatomy

The cow has one udder and four quarters!

The median suspensory ligament is the most important support structure and supports and separates the two lateral halves of the udder. Front and rear quarters are separated by a thin membrane. There is no crossover of the duct system between quarters. Although the quarters are anatomically separate units, antibiotics infused into one quarter will quickly find their way via the blood stream into the milk in other quarters.

The front teats are usually a little larger than the rear teats. The integrity of the teat end is very important in protection from mastitis. Teat skin condition, teat end condition and the keratin lining of the streak canal are important factors in protection against new intra-mammary infections. With the exception of some *Mycoplasma* mastitis infections, which may be seeded into mammary tissue by hematogenous spread, all mastitis causing bacteria enter through the teat canal (streak canal).

The internal structures of the udder include the secretory parenchyma consisting of grape-like clusters of myo-epithelial alveolar tissue which express milk under the influence of oxytocin, the ducts which lead from the secretory tissue, and the cisterns, including the gland cistern, which opens into the teat cistern. Milk available in the cisterns between milkings amounts to 100-400 ml in each quarter. This milk is available to the milking machine on attachment of the inflations. Note that once the milk in the cistern is exhausted the milking machine will be milking an empty teat unless there has been proper stimulus for milk letdown (see "overmilking").

Udder development

Mammary tissue develops in the udder at an accelerated rate compared to the tissues of the rest of the body between about 3 months of age and puberty (9 - 11 months of age for European type dairy breeds). This is known as the period of **allometric growth**. Underfeeding in this period impairs udder development. More importantly, overfeeding during the allometric growth period can lead to the deposition of excess fat in the udder and reduce milk secretion capacity in adult life. Heifers raised at a rapid growth rate prior to breeding (~15 months of age) have lowered percent of total mammary parenchymal tissue compared to those raised at a more modest rate of growth.

Physiology

Growth hormone is the predominant hormone governing synthesis of milk in cattle (vs. prolactin in non-ruminants), hence the use of rBST for production enhancement in cattle. Milk contains a feedback inhibitor synthesized by mammary cells as they secrete milk. Frequent removal of milk minimizes the effects of feedback inhibition and maintains milk secretion, but this mechanism is also used to "dry off"

cows at the end of lactation. Milk ejection (as opposed to milk synthesis) is under the influence of the pituitary hormone oxytocin. Oxytocin secretion is triggered by stimulation of the mammary gland and teats, and also by other situational triggers, such as entering the milking parlor. Oxytocin travels via the bloodstream and causes contraction of the myoepithelial cells surrounding the alveolus. This results in the ejection of milk into the duct system. The delay between oxytocin secretion from the pituitary gland and ejection of milk in the mammary gland is between 60 and 90 seconds. This is known as the milk let-down time.

Adrenalin is a powerful antidote to oxytocin release from the posterior pituitary gland. In the milking parlor a calm non-threatening environment and a predictable milking routine promote efficient milk letdown.

Udder immunity

The teat canal is the main barrier to infection entering the tissues of the udder. Polymorphonuclear leucocytes (known as somatic cells) constitute the major cellular response to bacterial infection in the udder, but do not trigger immune memory. Despite many years of research, the failure to harness immune mechanisms in the mammary gland in the prevention of mastitis has been a major disappointment. Antibody levels in milk are much lower than those in plasma. Although there is an effective vaccine against coliform mastitis no effective vaccines have been produced against the other major mastitis pathogens including environmental streptococci, *Streptococcus agalactiae*, *Staphylococcus aureus* and *Mycoplasma bovis*.

Mastitis

Mastitis organisms

Contagious

Staphylococcus aureus

Streptococcus agalactiae

Mycoplasma bovis

Environmental

Streptococcus uberis

Streptococcus dysgalactiae

Coliforms (*E.coli*, *Klebsiella*)

Minor pathogens

Corynebacterium bovis

Staphylococcus epidermidis

Staphylococcus hyicus

Contagious Mastitis vs. Environmental Mastitis

Contagious mastitis is transmitted during the milking process as a result of contamination of milking equipment with milk containing mastitis bacteria. Mastitis infections are transferred from teat to teat by **teat-end impacts** (blow-back of milk droplets into the teat end) which result from fluctuations in teat-end vacuum. These fluctuations are most often caused by liner-slips, but also by such problems as

flooding of the milk line. The organisms that cause contagious mastitis are obligate parasites of the tissues of the udder or of the teat skin. They do not live free in the cow's environment.

Environmental mastitis organisms may live in udder tissue or be free in the environment. They enter the udder through the teat end either during the milking process (like contagious mastitis organisms) or because of environmental challenge by large numbers of bacteria in the environment, especially if the teat end is damaged.

Characteristics of Mastitis Organisms

Streptococcus agalactiae

This used to be the most prevalent contagious mastitis organism. The Five Point Mastitis Control Program was introduced in the 1960's to control this infection, primarily because of its negative impact on cheese yield. Because *Str. agalactiae* infections do not penetrate deeply into the mammary stroma they

- a) shed large numbers of bacteria in the milk and thus spread easily from cow to cow during the milking process and
- b) are accessible to antibiotic treatment.
- c) antibiotic resistance is uncommon

The Five Point Program, if implemented properly, was effective in controlling *Str. agalactiae*. Because of the nature of the organism most farms were able to eradicate the infection completely.

Recently there has been a resurgence of *Str. agalactiae* infections because of the movement of infected cattle into rapidly expanding herds.

Causes clinical and sub clinical mastitis.

Staphylococcus aureus

Once *Str. agalactiae* was controlled *S. aureus* became the most important cause of contagious mastitis. Control is much more difficult because:

- a) The virulence factor for *S. aureus* is its ability to penetrate into tissue, forming micro-abscesses which the cow walls off.
- b) Shedding of the bacteria is intermittent, so milk cultures do not always show the presence of infection
- c) Established infections are not readily accessible to antibiotics
- d) Chronic infections are common and probably incurable
- e) Some strains of *S. aureus* are resistant to beta-lactam antibiotics

The Five Point Mastitis Control Program

1. Maintain the milking machine
2. Disinfect every teat after every milking (post-dip)
3. Treat all quarters at dry off
4. Treat clinical cases promptly

S. aureus causes subclinical and clinical mastitis. Some strains cause an acute gangrenous mastitis which can result in sloughing of the affected quarter.

Mycoplasma bovis

M. bovis is a growing concern. It has spread from farm to farm through the purchase of infected cattle during herd expansions. *Mycoplasma bovis* mastitis in cows was prevalent in California in the 1970s, in the Midwest by the 1990s, and by 2000 was a problem in Pennsylvania and New York. In 2003, 16 of 23 states involved in a herd survey had at least one dairy with a positive *Mycoplasma* culture from the bulk tank. Once in the herd it behaves as a contagious organism and is spread during the milking process or by contaminated bulk mastitis treatments.

New udder infections can also arise when *Mycoplasma bovis*, which is a common inhabitant of the respiratory tract of cattle, spreads from the respiratory system to the udder. *Mycoplasma* is the only mastitis organism of cattle that spreads hematogenously. A history of respiratory disease or ear infection in calves occasionally precedes outbreaks of mastitis.

The characteristics of *M. bovis* mastitis are:

- a) Multiple quarters infected
- b) Dramatic drop in milk production
- c) Cows appear healthy apart from severe mastitis
- d) Unresponsive to antibiotic treatment
- e) Milk has flakes in it, and the milk is often watery

Control is by culling infected cows. However some cows are clinically normal carriers with very low levels of infection.

When submitting milk samples to the laboratory *Mycoplasma* culture must be specially requested. Bulk tank cultures are effective in identifying infected herds or milking strings. Bulk tank culturing is very sensitive (approximately 1:1000 cows).

Environmental Streptococci

Str. uberis and *dysgalactiae* are the most common pathogens isolated from high SCC cows and clinical cases in herds in which contagious mastitis is under control. They cause a high rate of clinical and subclinical mastitis, particularly in hot weather. New infections occur very commonly in the dry period, just after dry-off and just before calving. Most clinical cases occur in the first few weeks after calving, and many infections self-cure in about 8 days.

The most important virulence factor of *Str. uberis* and *Str. dysgalactiae* are their ability to resist phagocytosis by PMNs.

High levels of environmental streps. are found in bedding, particularly straw. The bacteria also colonize the gut and can often be isolated in high numbers from the teats and underside of the cow.

Most infections respond well to penicillins, pirlimycin, and cephalosporins, but many spontaneously cure in two to 3 milkings with milking out and adjunct oxytocin treatment.

Coliforms

The coliform bacteria that cause mastitis include the gram negative organisms *E. coli*, *Klebsiella spp.* and *Enterobacter* as well as less common pathogens such as *Serratia*, *Pseudomonas* and *Proteus spp.*

Coliform bacteria are opportunist organisms and are ubiquitous in the environment. The vast majority of infections are short-lived and inapparent but coliforms can also cause severe mastitis and toxemia. In herds with very low somatic cell counts most clinical mastitis cases are caused by coliforms.

When acute coliform mastitis occurs the coliform bacteria grow to very large numbers in the mammary gland and then die off releasing large amounts of endotoxin, which is responsible for most of the clinical manifestations of disease. Coliforms rarely establish persistent infections and are not responsible for persistently elevated SCC. New infections occur at dry off and around the time of calving. Most cases of coliform mastitis occur early in lactation.

High levels of *E. coli* and *Klebsiella* have been associated with the use of sawdust and shavings, especially green and wet sawdust. Only use sawdust and shavings from kiln dried lumber.

Acute cases have a swollen gland, watery milk with small flakes and mild systemic disease.

Peracute cases have rapid onset of severe toxemia, fever, tachycardia and impending shock. Cow may be recumbent (always check the udder of post-partum downer cows for mastitis!). Quarter may or may not be swollen. Milk is usually thin and serous with very small flakes.

Efficacy of antibiotics is uncertain because by the time the clinician is called to the cow the predominant pathology is toxemia, not bacterial infection. Therapy is frequent stripping of the gland to remove toxins, fluid and electrolytes (hypertonic saline i/v is commonly used so that cows rehydrate themselves), NSAIDs.

Control is by improving the environment, substituting sand bedding for organic materials, keeping cows clean, use of a pre-dip in the milking routine, avoid spraying the udder with water, barrier post-dips, J-5 vaccine in the dry period and early lactation.

Minor pathogens

These include coagulase negative staphylococci (particularly *S. epidermidis* and *S. hyicus*) and *Corynebacterium bovis*. They generally behave as contagious pathogens but rarely cause clinical mastitis and are usually only responsible for mild increases in SCC. Their presence (especially *C. bovis*) is generally thought to increase resistance to colonization by the major mastitis pathogens and thus exert a protective effect. The prevalence of *C. bovis* is low in herds using an effective teat dip, good milking hygiene and dry cow therapy.

In herd sampling the presence of *S. epidermidis* and *C. bovis* in a large percentage of samples suggests deficiencies in milking hygiene, particularly in the use of teat dip.

Mastitis case management

Examination of the udder

There are five stages to the examination of the udder.

1. Visual examination for symmetry, externally visible changes, and lesions of the skin e.g papillomata.
2. Palpation - use two hands and compare side to side
 - a. by superficial palpation for signs of inflammation of the udder (i.e. heat, pain and swelling)
 - b. by deep palpation for signs of fibrosis and other changes in the udder parenchyma.
3. Use Strip cup to examine milk for wateriness, flakes and clots.
4. Use California Mastitis Test (CMT) to detect sub-clinical mastitis.
5. Take Milk sample for bacteriological culture and antibiotic sensitivity. (Culture for *Mycoplasma* is usually only done by the lab if requested).

Subclinical mastitis

PMNs are attracted into the mammary gland when bacterial infection occurs. These are known as somatic cells, and the somatic cell count (SCC)/ml of milk is used as an indication of udder infection. In the absence of bacterial infection SCC is <200,000/ml. SCC > 200,000 in the absence of systemic disease (inflamed udder, fever, off feed, large clots in milk) indicates sub-clinical mastitis. Somatic cells are detected by the California Mastitis Test, or by cell counting methods. Farmers can opt to have the DHIA labs test samples monthly from individual cows, and the dairy co-ops that buy the milk test the bulk tank SCC (BTSCC) every time they pick up milk from a farm.

In the US the Pasteurized Milk Ordinance (PMO) says that the maximum BTSCC for milk to be sold as Grade A milk (i.e. for fluid consumption) is 750,000/ml of milk. (The European Union has adopted a standard of 400,000 SCC/ml.)

Most sub-clinical mastitis is caused by persistent infection with organisms such as *S. agalactiae*, *Staph aureus* or the environmental streptococci. Sub-clinical mastitis results in replacement of secretory tissue by scar tissue and abscesses, and the consequent loss of production is the major cause of economic loss from mastitis infections.

Bacteriological cure rates for mastitis organisms in lactating cows are low (60% or less). Treatment of lactating cows with subclinical mastitis is usually not an economical proposition unless the farm is going to lose its Grade A permit to ship milk for having somatic cell counts persistently over 750,000/ml. The

only exception appears to be in cows which acquired a new infection with an environmental *Streptococcus* (e.g. *Streptococcus uberis* or *Streptococcus dysgalactiae*) during the dry period. In this case, identifying cows with new infections using the CMT test in the first three days of lactation and treating with a cephalosporin or pirlimycin is probably justifiable because you can recover the costs throughout the lactation. In addition many veterinarians prescribe an extra-label extended treatment for these early lactation cows to try and improve cure rates (e.g. infuse at every milking for 3 days).

Clinical mastitis may result from flare-ups of subclinical mastitis but may also be new infections. In herds with low SCC <100,000 most cases of clinical mastitis are due to coliform bacteria.

Acute mastitis. If there is changed milk (clots or flakes) or the udder is inflamed, but the cow is not systemically ill, use a propriety intramammary antibiotic treatment according to label directions. Systemic antibiotics are not required.

More severe cases with systemic involvement (febrile, off feed) may be treated with systemic antibiotics as well as intramammary antibiotics, but most cases will respond to intramammary antibiotics and NSAIDs.

Summer mastitis

This is most often seen in heifers and dry cows at summer pasture and is due to infection with *Arcanobacter pyogenes* and *Str. dysgalactiae*. The quarter is usually abscessed and is lost. Treatment is usually not effective.

Antibiotic Choice

Most intramammary antibiotics available in the US are aimed at gram positive bacteria, though a broad spectrum antibiotic, (ceftiofur {Spectramast LC} “for the treatment of clinical mastitis in lactating dairy cattle associated with coagulase-negative staphylococci, *Streptococcus dysgalactiae*, and *Escherichia coli*”) has been added to the line-up.

Common lactating cow intramammary antibiotic preparations:

Amoxicillin trihydrate (Amoximast)
Sodium cloxacillin (Dariclox)
Hetacillin potassium (Hetacin-K)
Penicillin and Novobiocin (Albacillin)
Cephaprin sodium (Today, Cefalak)
Ceftiofur hydrochloride(*Spectramast LC)

Common antibiotics for dry-off treatment

Benzathine Cloxacillin (Dryclox, Orbenin Dry Cow)
Cephaprin benzathine (Tomorrow, Cefadry)
Penicillin and Novobiocin (Albadry Plus)

Penicillin and Streptomycin (*Quartermaster)

Ceftiofur hydrochloride (*Spectramast DC)

*prescription antibiotics

Injectable antibiotics in mastitis

Injectable antibiotics have often been used as adjunct mastitis therapy or as sole treatment. Systemic antibiotics should not be used unless the cow is systemically sick. Common antibiotics used are Procaine Penicillin G (4 day milk withdrawal), Ampicillin (e.g. Polyflex – 48 hour milk withdrawal). Parenteral ceftiofur is not an effective mastitis treatment (see below).

Some cautions about antibiotic use in mastitis:

- Homemade and bulk mastitis preparations are dangerous – they are common causes of yeast mastitis and mastitis caused by unusual pathogens such as *Pseudomonas aeruginosa*. They are also a means of transferring *Mycoplasma* infections from cow to cow.
- Ceftiofur (Naxcel, Excenel) given systemically has no milk withdrawal but also does not have bacteriocidal activity in the udder.
- Penicillin crosses the blood/milk barrier very inefficiently except in the presence of acute inflammation
- Gentamycin should never be used in cattle – although the recommended milk withdrawal is 10 days, there is no defined withdrawal period for meat. It may bind to kidney tissue for 6 – 18 months.
- Tilmicosin (Mycotil) has been used extra label to treat chronic *S.aureus* cases at dry off. Note that if used in lactating cattle the milk withdrawal time for Mycotil is at least 15 days.

Technique of udder infusion

e.g. Dry Cow Treatment

1. **Confirm and segregate dry cows.** After cows have been separated from the milking herd, review their pregnancy records to ensure the right cows have been selected for dry treatment.
2. **Prepare materials.** Place gauze pads in a container of 70% isopropyl alcohol for cleaning teat ends, or find the pre-packaged pads in the carton of dry cow tubes.
3. **Milk the quarters**
4. **Sanitize teats.** Apply teat dip and allow to stand for at least 30 seconds.
5. **Scrub teat ends.** Clean the teats furthest away from you first so that you do not re-contaminate the teats by accidentally touching them. Clean each teat with fresh gauze.

6. **Administer dry cow therapy.** Start with the teats closest to you. Use the partial insertion method.
7. **Re-dip teats immediately.**
8. **Clearly mark all animals as dry.** Use two methods on each cow e.g. leg band + paint stick.

Udder edema

Udder edema can be a problem in the peri-parturient cow, particularly in heifers when they have their first calf. It manifests as a pitting swelling of the teats, base of the udder and sometimes the ventral abdomen. It can interfere with machine milking, and can lead to chapping of teats. In severe cold weather (below 10F) it can lead to freezing of teats, resulting in gangrene.

Etiology of udder edema

Various theories have been advanced, including excessive dietary sodium, potassium or energy intake, genetic predisposition and oxidative stress as a result of e.g. mycotoxins in the ration, or excess iron or molybdenum.

Management of udder edema

Eliminate free-choice salt and buffers from the diet and limit dietary intake of sodium and potassium in the ration. Avoid over-conditioning of pregnant cows, particularly the heifers. Provide adequate antioxidants by feeding 1,000 IU/day vit E and adequate selenium.

Treat severe udder edema by massage, diuretics (furosemide) or corticosteroids such as dexamethasone. Watch out for excessive urinary losses of calcium when giving repeated doses of furosemide to the older cow around the time of calving, as this may precipitate clinical hypocalcemia.

Vaccines and mastitis

Common core antigen vaccines

The *Enterobacteriaceae* share a common core antigen. Core antigen bacterins derived from *Salmonella* strains have been used to make vaccines against coliform mastitis (J-Vac, J-5, Endovac Bovi). These vaccines decrease the incidence and severity of clinical disease although the vaccines seem unable to prevent new coliform intramammary infections. The vaccines are given as two or three doses during each dry period.

Staph. aureus bacterins (such as Lysygin) have been available for many years but their efficacy has been questionable, particularly when used in cattle already sub-clinically infected with *Staph. aureus*. There has been some success in reducing the number of new *Staph aureus* intramammary infections when three doses are given to heifers before their first calving.

Troubleshooting Somatic Cell Count Problems

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Introduction

High somatic cell counts result in poor milk quality and economic loss to the producer. Laboratory investigation is required to determine the type of bacteria responsible. A management strategy may then be put in place to reduce somatic cell counts and prevent new infections from occurring. The exact strategy depends on whether the bacteria recovered from milk cultures are primarily environmental, or are contagious organisms transmitted cow to cow during milking. Strategies for dealing with environmental bacteria center on environment and cow comfort, pre-milking udder preparation, teat-end condition and dry cow treatment. Strategies for contagious bacteria are aimed at improving milking hygiene, post-milking teat dipping, correcting faults in milking machine function, and dry cow treatment.

Increased somatic cell counts (SCC) have become a matter of increasing concern for dairy producers as the economic costs of poor milk quality to both the producer and the industry have been more clearly recognized. Some states have taken the initiative to reduce limits on somatic cell counts to 500,000/ml, but the United States still has lax milk quality standards compared to other nations competing for the global market for dairy products. This puts US producers at a competitive disadvantage in the world marketplace. Indeed, the National Conference on Interstate Milk Shipments recently failed to take the action needed to reduce the legal limit on SCC, and the limit for US milk remains at 750,000/ml. The major economic cost to producers caused by elevated SCC is in reduced milk revenue from lost production through damage to the milk secreting tissues in the udder, particularly as a result of chronic infections, and through lost premiums. Chronic infections result in replacement of milk secreting tissue by scar tissue, which also reduces longevity of cows in the herd. Some developments in the dairy industry have exacerbated the impact of udder infections. For instance, milk flow rates have increased over the years from 1.76 – 3.52 lbs/min, and this has been accompanied by a 12-fold increase in mastitis susceptibility (Grindall and Hillerton, 1991).

Records

Increases in somatic cell counts are first evident in processing plant tickets delivered to the farm. Examination of herd records and individual cow somatic cell counts give important information about the nature of the increase in somatic cell counts.

High somatic cell counts reflect the fact that the udder is infected with a mastitis-causing agent. Quarters free of any infectious agent have a cell count ranging from less than 50,000 to 150,000. Quarters infected with a mastitis causing bacteria generally have a somatic cell count greater than 250,000.

Linear Scores vs Weighted Average Somatic Cell Counts

The bulk tank SCC sent out by the dairy is important in letting the producer know if the tank is of sufficient quality to attract a premium and provides a crude average of the SCC of individual cows. Linear scores derived from individual cow somatic cell counts provide information which average somatic cell counts do not provide. Particularly in small herds the presence of a few cows with high somatic cell counts can cause a dramatic increase in SCC, but the linear score may not elevate because it does not represent a change in the overall character of the herd with respect to somatic cell counts. Changes in SCC accompanied by an elevation of linear score indicate elevation of cell counts in a much larger number of cows. The other advantage of linear score is that it relates in a linear fashion to loss of production and allows the producer to estimate rather accurately the loss of income caused by udder infections.

An exceptionally useful report is one similar to the DHI 520 report, which ranks cows by their contribution to the bulk tank count. In some herds two or three cows may be responsible for 30 – 40% of the bulk tank SCC. Examination of the report allows appropriate decisions to be made on individual cows and can be instrumental in preserving premiums while appropriate investigation of the causes of new cases of sub-clinical mastitis is done.

Investigating High Somatic Cell Counts – Step by Step

High SCC is indicative of inflammation in the udder, most commonly because of bacterial infections. In order to develop a strategy for management changes that will lead to a reduction in the number of infected quarters it is necessary to learn whether the predominant bacteria responsible for the high SCC are contagious or environmental bacteria.

Contagious

Strep. agalactiae
Staph. aureus
Mycoplasma species

Environmental

Strep. uberis
Strep. dysgalactiae
Coliforms (e.g. E. coli, Klebsiella)

Many veterinary practices, milk processors, state and private laboratories can culture milk for the presence of the common organisms, especially the Streps., Staphs., and coliforms. Growth of Mycoplasma requires special conditions, and culturing for this organism must be specifically requested. It will not show up during routine bacterial cultures. Since Mycoplasma infections are at present uncommon in this part of the country it is not usual to routinely culture for them.

Coliforms generally cause short-lived infections and self-limiting infections in the udder. Coliform infections range from undetectable to fatal toxic mastitis, but because they are short-lived infections they contribute little to a prolonged elevation of the bulk tank SCC. Investigations of high SCC, as opposed to investigations aimed at determining the causes of clinical mastitis, rarely focus on the coliforms. Indeed, since the infections are usually so transient, it is unusual to find many infections when doing bacterial culture surveys. The presence of large numbers of coliforms when culturing milk from a high SCC herd usually indicates that the samples have become contaminated because of poor collection technique.

Bulk Tank Cultures

On the face of it, routine bulk tank cultures would seem to be an economical method of detecting the presence of new infectious agents in the herd and monitoring levels of infection within the herd. The factors which affect the ability of bulk tank cultures to provide this information are the efficiency with which bulk tank cultures detect infections in the herd (high for *Str. agalactiae*, low for *S. aureus*), and the prevalence of infection within the herd. An Ontario study showed that with direct culture *S. aureus* infection could be demonstrated in 57% of bulk tanks, whereas reincubation and reculturing led to the identification of *S. aureus* infection in 98% of the same tanks (Kelton et al., 1999). Thus it can be assumed that if appropriate culture methods are used virtually all herds will be found to be infected. On the other hand it has been shown that it is difficult to distinguish between low and high levels of infection with *S. aureus* because of the variable shedding of the bacteria into the milk. These two observations indicate that bulk tank culturing for monitoring *S. aureus* infection rates is of limited value. In the case of *Str. agalactiae*, however, bulk tank culturing will efficiently identify the introduction of infected cows in to the herd. This is important, because with increasing numbers of herd expansions and the consequent increase in the movement of cattle, a resurgence of *Str. agalactiae* infections is occurring. Bulk tank culturing, as a biosecurity device for controlling the introduction of *Str. agalactiae*, is an excellent back up to the testing of purchased animals before they enter the milking string.

Bulk tank cultures as a diagnostic tool or monitor for *Mycoplasma* infection is sensitive and accurate. Bulk tank cultures for *Mycoplasma* can detect up to one infected cow in 1,000. Identification of individual cows can be done by narrowing down the groups tested and then testing individual cows. *Mycoplasma* culture must be requested specifically as it is not included in standard aerobic culture.

Individual cow sampling

It is important to collect excellent quality, uncontaminated milk samples in order to have a high probability of understanding from the culture results which are the important bacteria that contribute to the SCC problem. The growth of contaminating bacteria from the teat skin or environment may complicate interpretation of the culture results, or make their interpretation impossible.

Selection of cows to sample

When going to the trouble of culturing cows it is tempting to select those chronic problem cows that have been treated and not responded. They may not be representative of the cause of the herd's high somatic cell count, though they will clearly contribute to it. Our technique is to check cows in the order in which they enter the parlor, using the California Mastitis Test (CMT). This causes the least disruption of milking and allows us to collect samples in the early part of milking and watch the milking process, time take-offs, look at teat-ends during the remainder of the milking period. Any cow that has a positive quarter in the CMT test is selected for sampling, but only one CMT positive quarter is selected from each cow. We do not select animals just because they have a very high SCC. We find that culturing 16 CMT positive quarters generally gives us enough positive cultures so that we can say whether we are dealing with predominantly contagious bacteria, environmental bacteria, or a mixture of the two.

If *Str. agalactiae* is present it is easy to culture and we usually get a high number of positive samples. Because of the intermittent shedding of *S. aureus*, even from high somatic cell count cows, we often find only two or three positive cultures from the 16 samples in a *Staph.* herd. It probably requires 3 cultures on separate occasions to be 90% sure of detecting a *S. aureus* infected quarter. Thus our interpretation is that 2 or 3 positive samples represent a significant *S. aureus* problem. The environmental *Streps.* are generally easy to grow. We also often find significant numbers of other bacteria which cause low grade infections (often other *Staphylococci* and *Corynebacterium*) and whose presence may be significant if recovered from a sufficient number of quarters.

Sample collection

Pre-dip and forestrip all four quarters to observe any clumps or flakes in the milk. Dry the teats well with an individual towel. Carry out the California Mastitis Test on all four quarters and select a CMT positive quarter to sample. Swab the end of the teat with an alcohol soaked cotton ball (rubbing alcohol is fine). If you don't use too much alcohol the teat-end will dry rapidly, and the alcohol will not contaminate the sample. Use a sterile screw top tube available from your veterinarian to collect the sample. The first squirt of milk should go on to the parlor deck and the second squirt into the tube. One squirt of milk is enough. If you collect more you simply increase the chance of the sample being contaminated and less than one ml is required for the needed for the test. Collecting composite samples (all four quarters) into a WhirlPak bag or other container is frequently done, but it is difficult to take a sample without contaminating it. Composite samples may be taken at a later date if identification of all *S. aureus* cows is needed for the control program. Similarly samples taken from the sampling port on the milking machine are often contaminated with bacteria that did not originate in the milk (including coliforms and environmental *Streps.*). Growth of *Str. agalactiae* on the culture plates is always significant because it can only have come from an udder infection, and this is mostly true for *S. aureus*.

The milk samples should be put on ice immediately and sent to the laboratory. Both culture and antibiotic sensitivity can be requested. The antibiotic sensitivity test can be useful in helping to select an antibiotic for dry cow treatment. In most herds the culture results from 16 samples will give a clear pattern of infection, and will determine whether management needs to be modified to improve control of environmental bacteria, contagious bacteria, or both.

Management strategies for reducing high SCC caused by environmental bacteria

The key areas for attention are the cow environment, udder preparation procedures and the health of the teat-end.

Environment

Streptococcus uberis is the most frequently recovered of the environmental *Streps.* followed by *Str. dysgalactiae*. In herds where there are high levels of *Str. uberis* infections the bacteria colonize the gut and large numbers of bacteria are ejected into the environment. Particularly high numbers of bacteria can be recovered from the underside of the cow in this situation, particularly if the cows are dirty. The bacteria seem to thrive in a warm wet environment, so this can be a

particular problem of badly ventilated freestall barns in the summer months. The solution is to provide dry comfortable free stalls and adequate ventilation. The opening of barn walls to provide adequate airflow at the level of the freestalls, and the provision of an adequate ridge opening, are of particular importance.

Udder preparation

When the inflation is attached to the udder there is always a short period of liner slip as the inflation is attached to the teats. When liner slips occur bacteria that are on the outside of the teat are sucked in to the milking cluster by the vacuum and propelled into the teat end of one of the other teats. Thus excellent sanitation of the teat before attachment is essential. Insist on the use of a dip tested in National Mastitis Protocols. This is the only guarantee of the efficacy of the dip. Teat dips require twenty to thirty seconds to kill the bacteria on the teat skin, so this time must be built into the udder preparation routine. Fore stripping increases the effectiveness of the dip by working it into the teat skin and allowing more efficient killing of the bacteria. It is therefore a particularly valuable addition to the milking routine if environmental bacteria are a problem. It is also the most powerful stimulus to proper milk let down and thus helps to ensure adequate milk-out.

If possible it is best to avoid the use of water in udder preparation. Water remaining on the base of the udder after udder preparation, or on the teats, carries large numbers of bacteria into the inflation. If water must be used to remove caked manure from the teats, it should be used sparingly. Teats and the base of the udder must then be dried with a single use paper or cloth towel.

The most important defense mechanism that the udder has against the introduction of infection is the integrity of the teat canal at the end of the teat. This short tube, the streak canal, is lined with an extension of the skin surface, which secretes a waxy substance called keratin. This substance helps to seal the streak canal after milking. As the cow is milked some of this material is lost. Prolonged milking time leads to excessive loss of keratin and increased susceptibility to mastitis. The milk flowing through the streak canal acts as a lubricant which minimizes the loss of keratin. If milking continues after milk flow has ceased, or begins before let down occurs, the loss of keratin is excessive. Increased congestion and bruising of the teat end as a result of over milking also leads to the production a condition known as “hyperkeratosis”, the familiar donut like ring around the teat end. This also occurs if vacuum level, pulsation rates or pulsation ratios are not correctly adjusted. In severe cases there is prolapse of the teat end. Damaged teat ends of this type are less likely to seal after the cow leaves the milking parlor and may lead to increased levels of environmental mastitis.

Over-milking, leading to damaged teat ends, is a very common fault. It has various causes. Insufficient udder stimulation, insufficient time for milk letdown to occur, discomfort or fear, all interfere with the milk letdown response and lead to poor milk-out and a prolonged milking time. The mistaken impression that moderate amounts of milk left in the udder at the end of milking leads to an increased probability of mastitis also results in over-milking. Poorly adjusted automatic detachers are another common cause. The timing of detachment of the inflation is always a matter of compromise, because different quarters often take substantially different

periods of time to milk out. But as a rule of thumb, take-offs should detach when milk flow falls to one lb./minute, otherwise over-milking occurs.

Provision of water in the exit alley and fresh feed in the feed bunk will tend to keep cows occupied after they come out of the parlor and give teat ends time to seal before they lie down in the freestalls. However, if cows have been kept for an excessive period of time in the holding pen before milking they are likely to by-pass the food and water and lie down immediately.

The use of barrier dips (again tested in NMC protocols) is also recommended as an aid to controlling high SCC caused by environmental mastitis, but should not be regarded as a panacea. It is essential that, for barrier dips to have a reasonable chance of success, the housing conditions, udder preparation, milking machine function and milking time be optimized at the same time..

It is generally uneconomic to treat individual cows for high SCC (as opposed to clinical mastitis) during lactation. Dry cow treatment with an effective antibiotic is the method of choice for terminating existing infections, yet less than 75% of producers treat all cows with a dry cow antibiotic (Jayarao and Cassel, 1999). Also, in the absence of dry cow treatment, many new infections with environmental Streps. occur during the first two weeks of the dry period, and thus an effective dry cow program is doubly important in controlling environmental bacteria.

Management strategies for dealing with elevated SCC due to contagious bacteria.

Str. agalactiae and *S. aureus* have to live in the udder tissue or on the skin. Cows will not pick them up from the environment. Transmission of these bacteria from quarter to quarter, or cow to cow, has to occur during the milking process. If the testing process has identified a substantial *S. aureus* problem, then individual sampling is called for, to identify all infected cows. Testing options are to carry out bacterial cultures on milk pooled from all four quarters of each cow (composite sample) or to use a test such as Pro-Staph, which is an antibody test which can be performed on the same milk samples as are collected for routine DHIA testing.

Liner slips

Liner slips (squawks) are the most important method by which quarters acquire new infections with contagious bacteria. When liner slips occur milk particles, which may be contaminated with the mastitis causing bacteria, impact the end of one of the other teats. This blow-back of milk particles may occur from as far back as the claw and thus carry bacteria from milk originating in another quarter or from another cow. The occurrence of liner slips in the parlor should be monitored. All milkers should understand that this is a parlor emergency and that the inflation needs to be re-positioned as soon as possible. Dealing with it should over-ride all other tasks. If liner slips are frequent then the cause needs to be determined. Common causes are poor positioning of the inflation under the udder, and low vacuum.

Teat dipping

Post-milking teat dipping is the management technique that shows greatest reduction in the number of new infections. It is important to understand what is being accomplished by post-milking teat dipping. The purpose of teat dipping is to kill the bacteria that are on the surface of

the teat when the inflation is removed and that are capable of being drawn into the milking machine at the next milking. This means that all of the teat that enters the liner must be covered by teat dip. Mastitis control is not accomplished by only dipping the end of the teat and adequate coverage of the teat skin by dip cannot be accomplished with most hand held or parlor fitted sprayers. Dipping with a dip cup with a non return reservoir is the only method which we recommend for post dipping. Sprayers are adequate for pre-dip, particularly if the dip is massaged into the teat skin.

Dry cow treatment

After teat dipping, dry cow treatment is the second most important technique for the control of contagious mastitis. All quarters of all cows should be treated with an effective intramammary antibiotic. Cephalosporins, cloxacillins and penicillins are in wide use. Some strains of *S. aureus* are resistant to penicillin, and, unless it is known that the strain affecting the herd is penicillin sensitive, it is best to use another antibiotic. Although some antibiotic resistance is beginning to appear to *Str. agalactiae*, all dry cow intramammary preparations will give good control. The same cannot be said for *S. aureus*. This bacterium is much more invasive than *Str. agalactiae* and walls itself off in micro-abscesses in the udder tissue. This explains why shedding of the bacteria into the milk may be intermittent, and thus not always easy to detect. It also explains why antibiotic cure rates, even at dry cow treatment, are relatively low.

Culling

S. aureus infected cows that have not been cleared by treatment during the dry period are very difficult to cure and should be culled. One of the reasons *S. aureus* is such a problem in good herds is that the longer cows stay in the herd, the greater their risk of becoming infected. Herds that have striven for good cow longevity are paradoxically some of the herds in which *S. aureus* infections are a great problem. The best cows, which have often been in the herd longest, are the ones that are most likely to become chronically infected, and also the ones that the producer is most reluctant to cull. In small herds these chronically infected, high producing cows are often substantial contributors to total milk production and the culling decision can be a difficult one. However, maintaining chronically infected cows within the herd increases the risk of infection for the remainder of the herd, and in the long term leads to a deteriorating and intractable SCC problem. In herds undergoing expansion there is an especially marked tendency to keep cows which would otherwise be culled, in order to keep up cow numbers and maintain cash flow.

Segregation

Creating a group of *S. aureus* infected cows that can be milked as the last group through the milking parlor is a short term strategy that has been successfully applied on some farms. This removes the cows from the main herd and allows them to be culled after they pass peak milk production. Some farms have created this group for a short period during a herd expansion, while facilities are underutilized, and thus improved overall udder health in the enlarged herd. There is now good evidence that cows at the time of calving have weakened immune systems (Kherli et al., 1999) and cows are particularly susceptible to infections with coliform mastitis in the period around calving. Segregation of these cows into a fresh cow group, housed and milked separately, has many advantages from the point of view of general health, adaptation to the milking herd, and udder health.

Vaccination

Commercial vaccines for *S. aureus* have been on the market for a long time, and autogenous vaccines can be made from the farm strain of the bacteria. Generally vaccination has not resulted in elimination of existing infections. However evidence suggests that vaccination of heifers before they enter the herd may be an effective approach (Sears et. al, 1990) for reducing the rate of new infections.

Implementation

Effective and practical management techniques are available to control elevated SCC caused by both contagious and environmental bacteria. Once the bacterial cause of high SCC is established through testing, a somatic cell count reduction plan should be put in place. This is a situation where the team approach is essential (Kenyon, 1998). It works best if the package of management responses is agreed between the producer, veterinarian, milking equipment technician, dairy company field person, extension agent, nutritionist (for cow grouping advice) and others involved in helping make decisions about the farm operation. It is essential that the milking crew understands what is to be accomplished, and why, and that an effective monitoring system is installed. A management review after two to three months will help to fine tune the program, and provide positive reinforcement for the dairy crew to maintain the milk quality effort.

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