A Practical Look at Contagious Mastitis

Pathogenic microorganisms that most frequently cause mastitis can be divided into two groups based on their source: environmental pathogens and contagious pathogens. The major contagious pathogens are *Streptococcus agalactiae*, *Staphylococcus aureus*, and *Mycoplasma spp.* With the exception of some mycoplasmal infections that may originate in other body sites and spread systemically, these three organisms gain entrance into the mammary gland through the teat canal. Contagious organisms are well adapted to survival and growth in the mammary gland and frequently cause infections lasting weeks, months or years. The infected gland is the main source of these organisms in a dairy herd and transmission of contagious pathogens to uninfected quarters and cows occurs mainly during milking time. This fact sheet will describe the characteristics of the major contagious intra-mammary infections, management efforts and specific control procedures to reduce new infection rates with these organisms, and a step-by-step control program for contagious mastitis.

Organisms

*Streptococcus agalactiae*

*Streptococcus agalactiae* is a common mastitis agent whose eradication from individual herds is practical and cost effective. Most infected cows show few clinical signs of mastitis, such as abnormal milk, but usually have high somatic cell counts (SCC). A decrease in milk production almost always accompanies infection. Mastitis caused by *Streptococcus agalactiae* should be suspected in a herd if cow or bulk tank SCC’s begin to rise and remain high, especially when bulk milk SCC is 1,000,000 cell/ml or higher. Occasionally high bacteria counts in bulk tank milk will occur when infected udders shed high numbers of *Streptococcus agalactiae* in the milk.

*Streptococcus agalactiae* primarily infects the cisterns and the ductal system of the mammary gland. An irritant is produced, causing inflammation of the gland which is mostly subclinical with occasional clinical symptoms. Accumulation of bacterial waste products intensifies the inflammatory response, resulting in destruction of milk producing tissue and reduced milk yield of agalactia. *Streptococcus agalatiae* rarely causes severe illness, but extensive scarring of quarter may render it unproductive in subsequent lactations.
**Staphylococcus aureus**

*Staphylococcus aureus* is more difficult to eradicate than *Streptococcus agalactiae*, but definitely controllable. Infected udders are the most important source of infection. The organism readily colonizes teat skin lesions and the teat canal, and eventually passes into the mammary gland. The organism may also survive at other sites on the cow. Mastitis caused by *Staphylococcus aureus* produces more damage to milk-producing tissues than *Streptococcus agalactiae*, and decreases milk production with reported losses of 45% per quarter and 15% per infected cow. Recurring signs of milk clinical mastitis often causes additional losses. High bacteria counts in bulk milk are generally not seen with *Staphylococcus aureus* mastitis. However, as the number of infected cows increases, the bulk milk SCC increases, resulting in decreased milk quality. Herds with bulk tank milk SCC greater than 300,000 to 500,000 cells/ml often have a high prevalence of *Staphylococcus aureus* infected quarters. The bacteria damage the duct system and establish deep-seated pockets of infection in the milk secreting tissues followed by abscess formation and walling-off of bacteria by scar tissue. This walling-off phenomenon is partially responsible for poor cure rates of *Staphylococcus aureus* infection by antibiotic therapy. During the early stages of infection, damage is minimal and reversible. However, abscesses may release *staphylococci* to start the infection process in other areas of the gland with further abscess formation and irreversible tissue damage. Occasionally, infection by *Staphylococcus aureus* may result in peracute mastitis with gangrene. This gangrenous mastitis is characterized by a patchy blue discoloration and coldness of the affected tissue.

**Mycoplasma species**

*Mycoplasmata* spp. Are highly contagious organisms, are less common than *Streptococcus agalactiae* and *Staphylococcus aureus*, and are generally diagnosed in herds experiencing outbreaks of clinical mastitis that resist therapy. Frequently, the history of affected herds includes the recent introduction of new animals, a previous outbreak of respiratory disease, and/or cattle with swollen joints. Cows of all ages and at any stage of lactation are susceptible, but animals in early lactation seem to suffer more severely because of the occurrence of increased mammary gland edema. *Mycoplasma* spp. should be suspected in herds when multiple cows have clinical mastitis in more than one quarter but continue to eat and have little evidence of systemic disease. Cases are unresponsive to treatment, and generally affected cows show a marked drop in the milk production or cease lactating. However, *Mycoplasma* spp. May be isolated from high-producing cows in herds that do not experience the classic signs. Subclinical cases with intermittent signs of clinical mastitis are not uncommon. Infected cows may have a tissue and produce fibrosis in the udder as well as abscesses with thick fibrous walls, and great enlargement of the supra-mammary lymph nodes.
Management Programs

Transmission of pathogens that cause contagious mastitis from infected cows to uninfected herd mates most generally occurs at milking time. Management factors important in transmitting contagious pathogens include the milking machine, milker’s hands, teat washing materials and treatment procedures. Spread of contagious pathogens can be greatly reduced by good udder hygiene and post-milking teat dipping.

Other management factors which may influence susceptibility to mastitis pathogens including those that cause contagious mastitis are:

- **Injury.** Healthy teat skin is the first line of defense against mastitis. Lesions on teat skin frequently harbor bacteria that may cause mastitis. The cause of teat injuries should be quickly identified and eliminated. In cold climates, frostbite and chapped teat skin constitute injury and such injuries have been shown to harbor *Staph aureus*.

- **Nutrition.** Soils in many parts of the United States are deficient. Also, the vitamin A and E content of ensiled forages decreases during storage. Research indicates that diets deficient in vitamin A, E or the trace minerals selenium and copper can lead to increased incidence of mastitis.

- **Milking system.** Machine milking can also influence the rate of new contagious mastitis infection:
  - The role of machines as a carrier of bacteria to uninfected cows can be minimized by segregating and milking cows with known infection or those with high SCC last.
  - Bacteria can be transferred during milking form the infected quarter across the claw-piece to an infected quarter of the same cow. Cross infections may account for up to 40% of new infections in some herds. Properly designed and functioning milking equipment will prevent movement of air and milk droplets from one quarter to another, and may reduce these infections.
  - Abrupt reduction in milking vacuum can cause movement of air toward the teat end, and droplets of milk may strike the teat end (impacts). If the droplets are contaminated with bacteria, the impact may force bacteria into the teat duct and may increase the rate of new infection. Research has shown the high new infection rates were associated with vacuum fluctuations only accompanied by liner slip, a condition known to generate teat end impacts.
  - Although it has been difficult to show experimentally that the milking machine can damage the teat end sufficiently to increase the chance of infection, machine operators must always be aware of this possibility.
Control Procedures

Contagious organisms, for which the primary source is the mammary gland of the cow, are transferred primarily by events associated with milking. Good milking procedures, including cleaning and sanitizing teats before milking and post-milking teat dipping, help reduce the spread of infection from infected to uninfected cows. In mycoplasma infected herds, the use of rubber or plastic gloves when milking is recommended. Ideally, gloved hands should be disinfected between cows and dried off with paper towels. Some research trials have indicated additional control of contagious pathogens by automated disinfection of teat cup clusters (backflushing) or dipping teat cluster in disinfectant between cows. However, this practice in the field has minimal effect in reducing the rate of new infection, especially when compared to what can be achieved when an effective post-milking teat dip is used properly.

Streptococcus agalactiae

Streptococcus agalactiae is an obligate parasite of the mammary gland which means that, in nature, it can only live and reproduce in the gland. Because of this host-parasite relationship, Streptococcus agalactiae can be controlled and eradicated from a herd by identifying and treating infected animals. This can be done by obtaining milk samples for microbiological culture from all cows in the herd, and by treating the Streptococcus agalactiae infected udders with an appropriate intra-mammary infusion product. Streptococcus agalactiae infection responds well to beta-lactam intra-mammary mastitis preparation in both lactating and dry cows. Using other classes of antibiotics often results in poor cure rates. Some chronic infections do not recover. If two regimens of treatment do not eliminate the infection, culling should be considered to prevent infecting other cows.

Once Streptococcus agalactiae has been eliminated from a herd, careful control measures should be maintained to prevent re-infection, including monitoring bulk tank milk by monthly cultures for at least 6 months to assure clearance of infections. A closed herd is required to maintain it free from this pathogen. Breakdowns frequently happen due to the purchase of infected animals or by using contaminated milking equipment at fairs or livestock shows. New arrivals should be sampled before joining the milking herd. Dry cows and heifers also need to be included in Streptococcus agalactiae eradication programs, since they can represent a source of re-introduction of the organism to the milking herd. Calves fed discarded milk containing Strep. Agalactiae can spread the infection by suckling themselves or other pen-mates. Once Streptococcus agalactiae is established within the immature gland, it can persist until first parturition many months later. Therefore, dry cows and heifers should be cultured at calving before joining the milking herd.
**Staphylococcus aureus**

*Staphylococcus aureus* commonly produces long-lasting infections that can persist through the lactation and into subsequent lactations. To prevent *Staphylococcus aureus* intra-mammary infections, it is necessary to limit the spread of this organism from cow to cow and to reduce to a minimum the number of infected cows in a herd. To attain these objectives, milk from infected cows should never come in contact with uninfected cows. *Staphylococcus aureus* infected cows should be identified and milked last, or milked with a separate unit from those used on uninfected cows. Clinical mastitis sometimes occurs following prolonged subclinical infections. Antibiotic therapy during lactation may improve the clinical condition but usually does not eliminate infection. Infected quarters which do not respond to a single regimen of therapy are generally unresponsive to additional lactation treatment, regardless of culture and antimicrobial sensitivity test. Dry cow therapy may give better results than treatment during lactation, but even then, chronic infections can persist into subsequent lactations. *Staphylococcus aureus* infection status of cows should be one of the considerations when culling decisions are made.

Maintaining a *Staphylococcus aureus* –free herd is possible but more difficult than maintaining a *Streptococcus agalatia* –free herd and *Staphylococcus aureus* may reappear even in a closed herd. To achieve a “*Staphylococcus aureus* –free” status, every infected cow must be identified and handled as described in the preceding paragraph. The “uninfected” herd should be closely monitored by individual SCC and milk culturing. Teat injuries and chapped teat skin during cold weather should be minimized because they predispose cows to *Staphylococcus aureus* intra-mammary infections.

*Staphylococcus aureus* has also been implicated in intra-mammary infections in claves, breeding age heifers, and heifers at calving. The source of the *Staphylococcus aureus* to infect these you animals is not known but may be contaminated bedding, feeding milk from *Staphylococcus aureus* infected cows, cross suckling or exposure to high fly populations. Pregnant heifers should not be housed together with dry cows, when a significant number of cows in the herd are known to be infected with *Staphylococcus aureus*.

**Mycoplasma species**

There is no effective treatment for mycoplasmal mastitis, but the disease can be controlled by identifying infected animals by sampling and culturing milk samples from all cows in the herd, followed by segregation and/or culling the infected animals. If *Mycoplasma* spp. infected cows remain in the herd, they should be milked last or with a separate unit from those used on remain in the herd, they should be milked last or with a separate unit from those used on uninfected cows. Improper intra-mammary treatment of lactating or dry cows for other mastitis pathogens provides a good opportunity for spreading mycoplasmal infection from cow to cow, and even from herd to herd. Rigid sanitary precautions must be followed including the use of only single-
use commercial treatment products. Multi-dose vials and intra-mammary infusion products have been implicated in herd outbreaks of mycoplasmal mastitis.

Great care should be used when purchasing replacements. Many herds become newly infected by adding cows with *Mycoplasma* spp. infected udders. Before commingling with the herd, milk should be cultured from all replacement cows and heifers at calving for *Mycoplasma* spp. as well as for *Streptococcus agalactiae* and *Staphylococcus aureus*. When herds are purchased, it is a good policy to culture all suspected mastitic cows as well as bulk tank milk.

Sometimes, the disease may suddenly appear in previously uninfected herds without the introduction of replacements. Mycoplama is widely found as a resident of the bovine respiratory tract of apparently normal cows, and transfer of the microorganisms form the lungs to the mammary gland can occur. Mycoplasmal mastitis outbreaks have been associated with respiratory problems in calves, heifers, and cows in poorly ventilated barns. Young calves fed milk from cows with *Mycoplasma* spp. Infected mammary glands are prone to have respiratory infections, which may persist for several months.

A herd suspected of having mycoplasmal mastitis, based on history and clinical signs, should be cultured in order to establish the nature of the infection. Mycoplasmal infections may be complicated by common bacterial infections which occur concurrently.
Summary – Controlling Contagious Mastitis

1. **Prepare teats properly prior to milking.** Udders should be dry, and teats should be cleaned and dried prior to machine attachment using single-service paper towels or individual cloth towels which have been laundered and dried after each milking.

2. **Use adequately sized, properly functioning milking equipment.** Use milking machines in a proper manner on properly prepared cows. Avoid unnecessary air admission into the teat cups during attachment, machine stripping and unit take-off that can cause irregular vacuum fluctuations.

3. **Disinfect teats. Use an effective product after every milking.** Post-milking teat disinfection is the single most effective practice to reduce the rate of new intra-mammary infection by contagious pathogens.

4. **Assess clinical cases for treatment decisions.** Most cases of clinical mastitis other than those caused by *Streptococcus agalactiae*, are only minimally affected by antibiotic therapy during lactation. Work together with the herd veterinarian to design a management protocol for milk, moderate and severe cases of clinical mastitis.

5. **Use dry cow therapy.** Treat each quarter of every cow at drying off with a single dose of a commercially formulated, FDA-approved dry cow treatment product.

6. **Consider culling chronically infected cows.** Cows which are infected with *Streptococcus agalactia*, *Staphylococcus aureus*, or *Mycoplasma spp.* present a risk to non-infected cows in the herd.

7. **Maintain a closed herd.** If a new animals are purchased, culture milk from them before adding them to the herd.

8. **Establish an active milk quality program with the herd veterinarian.** Achievable goals for controlling contagious mastitis include: 0% cows infected with *Streptococcus agalactia, and Mycoplasma spp.*; and less than 5% of cows infected with *Staphylococcus aureus.*