Emerging Mastitis Threats on the Dairy

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Introduction

Mastitis is the most frequent and costly disease of dairy cattle. Losses due to mastitis can be attributed to both subclinical and clinical disease. Clinical mastitis losses are generally readily apparent and consist of discarded milk, transient reductions in milk yield and premature culling (Fetrow, 2000). Subclinical mastitis is considered the most economically important type of mastitis because of long term effects of chronic infections. Production losses due to subclinical mastitis on U.S. dairy farms were estimated to cost the dairy industry $1 billion dollars annually (Ott, 1999). Mastitis is a concern of most dairy farmers and their advisors due to these profound economic consequences.

Worldwide, farmers have achieved tremendous success in reducing the incidence of contagious mastitis by adopting the 5 basic principles of mastitis control: post milking teat disinfection, universal dry cow antibiotic therapy, appropriate treatment of clinical cases, culling chronically infected cows and regular milking machine maintenance. The greatest impact of adopting the 5-pt. plan has been on infections caused by contagious bacteria such as *Staphylococcus aureus and Streptococcus agalactia*. It has been estimated that these agents are now responsible for less than one third of all mastitis cases compared with >75% of all cases 20 years ago (Hillerton et al, 1995). Herds that successfully adopt these control strategies generally experience a reduction in subclinical mastitis that is demonstrated by reductions in bulk tank and individual cow somatic cell counts. Many WI dairy farmers have controlled contagious mastitis and consistently produce high quality milk. In 1998, over 1,800 WI dairy farmers had average BTSCC of <130,000 cells/ml and over 4,500 dairy farms obtained BTSCC of <200,000 cells/ml. Wisconsin grade A dairy farmers with BTSCC >400,000 cells/ml were ranked in the bottom 25% of herds (Figure 1).

The success of the 5-pt. plan against clinical and subclinical mastitis caused *by Staph aureus* and *Strep agalactia* has not been demonstrated for clinical mastitis caused by other agents. In a study of 9 Ohio herds that had low SCC, the rate of clinical mastitis varied from 15.6% to 63.7% of cows (Hoblet, et al, 1999). This study reported a mean cost per clinical case of $107 (equivalent to $142 current dollars assuming a 3% rate of inflation). A recent study looked at 65 Ontario dairy herds with above average milk production, low BTSCC and an average herd size of 52 cows (range in herd size of 24 to 216; Sargeant et al, 1998). In this study, clinical mastitis occurred in almost 20% of cows. The microorganisms isolated in this study (Figure 2) indicate that other pathogens have emerged to fill the niche vacated by *Staph aureus* and *Strep agalactia*.

Organisms such as coagulase negative staphylococci (CNS), environmental streptococci, Mycoplasma spp, and Serratia spp are increasingly implicated in mastitis in Wisconsin

dairy herds. The objective of this paper is to discuss diagnosis, treatment and control of these emerging mastitis pathogens.

Coagulase negative Staphylococcus (CNS)

Details. Coagulase-negative Staph (CNS) refer to staphylococcus bacteria that are not *Staph aureus*. The name "coagulase-negative" refers to a laboratory test that differentiates this species of bacteria from the "coagulase-positive" *Staph aureus*. These bacteria are also sometimes referred to as "environmental Staphs." CNS have been isolated from 7-30% of quarters in various herd surveys. They are one of the most frequent organisms isolated from milk samples in herds that have controlled major pathogens (Harmon et al, 1995). CNS live on teat skin and can colonize the teat canal. Anything that decreases the patency of the teat sphincter can allow infections to occur. Both clinical and subclinical mastitis can be caused by infections with CNS. Cows in their first lactation have been consistently shown to have a higher rate of infection with CNS as compared to older animals. This is usually attributed to the effectiveness of dry cow therapy in controlling these organisms. Infections with CNS are usually highest immediately after calving, decline in mid-lactation and increase again in late lactation until the cow receives dry cow therapy.

Effect on SCC and Clinical Mastitis. Somatic cell counts of quarters infected with CNS are generally 2-3 times higher than uninfected quarters, but the magnitude of SCC response is usually less than the magnitude experienced with other pathogens. While the SCC of infected cows can vary, a typical SCC for a cow infected with this pathogen would range between 250,000 – 400,000. While CNS are not a frequent cause of clinical mastitis, surveys in herds that have controlled major pathogens generally attribute 3-10% of clinical cases to CNS.

Treatment and Control. Treatment of cows that experience subclinical infections with CNS during lactation is not recommended. The relationship of CNS infections to milk production losses is unclear and spontaneous cure rates of up to 73% have been reported. Post-milking teat dip is the most effective method of controlling this pathogen. The benefit of pre-dipping to control this organism is unclear (Ruegg et al, 1997). When teat dips are not used (i.e. during the dry period, or during very cold weather) infections with CNS increase. Routine dry cow therapy is effective in decreasing intramammary infection rates. Milking routines, environmental conditions and equipment performance that damage teat ends may result in increased infections.

Environmental Streptococcus species

Details. Environmental streptococci refer to species of streptococcus other than *Strep agalactia* that are isolated from bovine mastitis. These organisms are also referred to as "non-ag" streptococci. The most common mastitis causing environmental streps are *S. uberis* and *S. dysgalactia.* These organisms (especially *S. uberis*) tend to behave somewhat like traditional contagious mastitis organisms because they can cause chronic infections that develop very high SCC. Environmental streptococci differ from traditional contagious mastitis organism because the primary route of exposure is from the environment. Infection rates for environmental streps are highest before calving,

during early lactation and near dry off (Oliver et al, 1998). The beginning and end of the dry period are high-risk times for the development of intramammary infections with these organisms (Figure 3). During the early weeks of the dry period, the udder is many times more susceptible to infection than during the preceding lactation. A recent study reported that cows with environmental pathogens isolated at dry off were 4.5 times more likely to have a new clinical case in their next lactation (Bradley & Green, 1999). In this study, 65% of clinical cases of environmental mastitis during lactation had the same pathogen isolated during the preceding dry period. Environmental streps are ubiquitous in the environment and are frequently isolated from organic bedding sources such as straw. Exposure to these pathogens can occur at anytime but the risk of infection is much higher during the dry period as compared to during lactation. Several studies have shown an increased incidence of infections with environmental streps during winter months.

The occurrence of sporadic high bacterial counts in bulk tank milk have been traced to small numbers of cows infected with S. uberis and S. dysgalactia (Britten, 1998; Mickelson et al, 1998). One processor began isolating the bacteria present in milk sample with >60,000 raw counts. They noted a steady increase in the number of strep species that have been isolated (Figure 4).

Effect on SCC and Clinical Mastitis. Environmental streps can cause both subclinical and clinical mastitis. There are usually many subclinical infections present for every clinical case that is observed. A study of *Strep uberis* intramammary infections demonstrated that the ratio of subclinical to clinical infections increased from 10 subclinical per 1 clinical case in early lactation to 24 subclinical per 1 clinical case in late lactation (Oliver, et al, 1998). In other words, the risk of an infected quarter becoming clinical decreases with stage of lactation.

Treatment and Control. Traditional methods of controlling mastitis (routine dry cow therapy, pre and post dipping) are helpful in controlling these organisms. Additionally, because these organisms thrive in organic bedding, herds that experience problems with environmental streps may need to consider non-organic bedding sources such as sand. Many infections develop during the dry period, so control procedures must focus on the dry cow environment and calving pens. While the spontaneous cure rate for IMI caused by these pathogens may approach 50%, clinical cases of mastitis caused by environmental streps should be treated with approved intramammary antibiotic products. Researchers have documented a shorter duration of infection and fewer relapses in infected cows treated appropriately with approved intramammary antibiotics (Morin et al, 1998). The efficacy and profitability of treating subclinical infections with environmental streptococci is an area of active research.

Mycoplasma species

Details. Mycoplasma refers to a group of bacteria of which over 20 different species have been isolated from cattle. The most important mastitis causing species of this organism is *Mycoplasma bovis*. *M. bovis* lives naturally in the upper respiratory tract of cattle and is an important component of bovine respiratory diseases such as shipping fever and calf pneumonia. Mastitis caused by *M. bovis* has been reported throughout the world. Until recently, mastitis caused by *M. bovis* was considered to be a regional problem of western dairy herds. Prior to 1992, only 2 confirmed cases of mycoplasma mastitis had been reported in Wisconsin (Thomas, 1998). Since 1992 the incidence of mastitis caused by *M. bovis* has increased (Fig 5-data from personal communication with Dr. Chet Thomas, SVM, UW, Madison, 2001 YTD Jan-Jul.).

The most important risk factor for mycoplasma mastitis is purchase of an infected carrier cow but outbreaks of this organism have occurred without a history of purchasing cows. Heifers that have been raised in commingled specialized calf-raising facilities may also be a source of infection. Many WI herds that experienced mycoplasma mastitis, report that an outbreak of respiratory disease preceded the mastitis outbreak .

Mycoplasma mastitis is classified as a contagious mastitis pathogen because the reservoir for the infection is other infected cattle, including calves. In contrast to other forms of contagious mastitis, mycoplasma infection can spread from the respiratory system to the udder. The spread can occur due to transmission through the air or through the blood stream. A history of respiratory disease or ear infections in calves occasionally precedes outbreaks of mycoplasma mastitis. A common source of infection is the purchase of cows subclinically infected with mycoplasma mastitis. Non-lactating animals are at risk as they can be subclinically infected prior to freshening. After calving, these animals may never develop clinical mastitis but may shed high levels of mycoplasma organisms in their milk (Blackmer, 1998). Transmission between cows can occur during the milking process or through contamination of cow contact areas in the environment.

Effect on SCC and Clinical Mastitis. There is considerable variation in the appearance of milk from glands infected with mycoplasma. The classic symptoms of mycoplasma mastitis have been described (Blackmer, 1998):

- Multiple quarters involved
- Dramatically decreased milk production
- Cows appear otherwise healthy but have severe mastitis
- Milk has sandy or flaky sediments in watery or serous fluid

It is well recognized that cows can develop subclinical infections with mycoplasma and have normal appearing milk. These subclinically infected cows may have intermittent periods of abnormal milk or their milk may continually appear normal. Somatic cell counts of subclinically infected cows will be increased. While there are some cows that appear to spontaneously recover from intramammary infections with M bovis, cows that

have had mycoplasma cultured from their milk should be considered to be permanently infected regardless of the visual appearance of their milk. Asymptomatic quarters that are infected can shed Mycoplasma organisms. Mycoplasma mastitis should be suspected when mastitis that does not respond to conventional mastitis treatment occurs in multiple quarters.

Treatment and Control. Bacteriologic culture of milk is required for the diagnosis of mycoplasma mastitis. Milk samples from infected quarters, composite milk samples from infected cows or bulk tank samples can be submitted for culturing. Not every mastitis laboratory performs cultures for mycoplasma because special techniques must be used to grow this organism. The Wisconsin Animal Health Laboratory is one Wisconsin laboratory that performs mycoplasma cultures. Even at laboratories that offer mycoplasma culture, the culture is not performed unless it is specifically requested. To detect mycoplasma, milk is plated on different media and incubated for 7 days in a special incubator. In milk samples obtained from individual cows, a negative mycoplasma culture usually means that the organism is not present. However, intermittent shedding of the organism has been reported, so false negative cultures may occasionally occur (Jasper, 1981). Bulk tank culturing is a good way to monitor a herd for the introduction of mycoplasma mastitis. Detection of as few as one infected cow in bulk tank milk from a 1000 cow dairy has been reported. Like individual cow milk cultures, periodic shedding patterns may lead to an occasional false negative bulk tank sample in a herd that contains infected cattle.

The first step in controlling mycoplasma mastitis is recognizing that the disease is present in Wisconsin dairy herds. A strong association between the introduction of new cattle and outbreaks of mycoplasma mastitis has been reported. Mastitis biosecurity programs can be used to decrease the risk of purchasing infected cattle. Bulk tank cultures from the herd of origin should be requested for non-lactating purchased cows and somatic cell counts and composite milk samples from individual cows should be reviewed prior to purchasing lactating cows. Cows that calve after purchase should be isolated until a negative composite milk sample is obtained. Herds that are routinely purchasing cattle should submit bulk tank milk for mycoplasma culture twice monthly.

The management of sick and fresh cows contribute to the spread of this organism. Fresh cows should not be housed in the same pens or milked with the same equipment as sick cows. The feeding of waste milk to calves is another source of transmission of this disease throughout the herd. Calves fed infected milk may develop pneumonia, joint infections and head tilts related to ear infections.

When mycoplasma is found in a bulk tank or individual cow culture, the number of infected cows must be determined. Depending upon herd size, there are several strategies that can be considered. If resources allow or the herd is small, composite samples from all cows should be submitted for culture. In larger herds, group milk samples can be

submitted by sequentially culturing the bulk tank during milking. Individual milk samples can be obtained from cows only in the infected groups.

There is no treatment for cows that develop mycoplasma mastitis. Antibiotics are totally ineffective for this organism. No existing vaccines have demonstrated sufficient efficacy to be recommended. Cows that are infected with mycoplasma should always be considered as infectious, regardless of their production level, appearance of their milk or subsequent negative milk culture. In most cases, infected cows should be promptly culled. The only exception to this rule is when a culling is financially unacceptable because a large proportion of a herd is infected. In this case a herd specific strict segregation plan should be developed. Cows that are segregated should be milked last and great care taken to minimize transmission between cows. Weekly bulk tank cultures should be performed for one year after isolation of mycoplasma from a mastitis case.

Serratia spp.

Details. *Serratia spp.* are common inhabitants of soil and water. Two species (*Serratia marcescens* and *Serratia liquifaciens*) have been implicated in a number of outbreaks of mastitis in dairy cows. Originally, mastitis caused by this organism was associated with contaminated cholorhexidine teat dips but more recently a number of outbreaks associated with unknown or environmental sources have been reported (Ruegg et al, 1992). Susceptibility to infections caused by *Serratia spp*. is highest during the dry period and several outbreaks have been related to adverse weather conditions.

Effect on SCC and Clinical Mastitis. The majority of intramammary infections caused by *Serratia spp.* are subclinical in nature. These infections tend to become chronic and persist for a considerable amount of time (>60 days). SCC's of cows infected with Serratia are increased. Up to 50% of cows infected with Serratia may exhibit mild clinical signs of mastitis (abnormal milk without appearing ill).

Treatment and Control. Spontaneous cure rates exceed the reported cure rates of infected cows treated with intramammary antibiotics, so antibiotic therapy during lactation is not recommended. Control measures for herds experiencing outbreaks of mastitis caused by Serratia should include strict attention to milking hygiene, pre and post dipping with appropriate iodine based dips and routine dry cow therapy.

Conclusion

Tremendous progress has been made in the control of contagious mastitis pathogens. The adoption of accepted methods of mastitis control have greatly decreased the amount of mastitis caused by *Staph aureus* and *Strep agalactia* in many herds. While BTSCC levels have dropped in response to these measures, the rate of clinical mastitis (and milk discard) remains unacceptably high on many dairy farms. Mastitis causing pathogens such as CNS, environmental Streps, Mycoplasma spp. and others have emerged to fill the niche vacated by control of the major pathogens. BTSCC levels do not necessarily reflect clinical mastitis rates caused by these emerging pathogens. It is imperative to have systems in place to identify outbreaks of these organisms. To continue to make progress in mastitis control, surveillance methods must shift from strict reliance upon

decreasing BTSCC to routine recording of clinical mastitis and regular bulk tank culturing.

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Figure 1. Percentiles of Bulk Tank SCC, in Wisconsin Grade A Dairy Herds, 1998

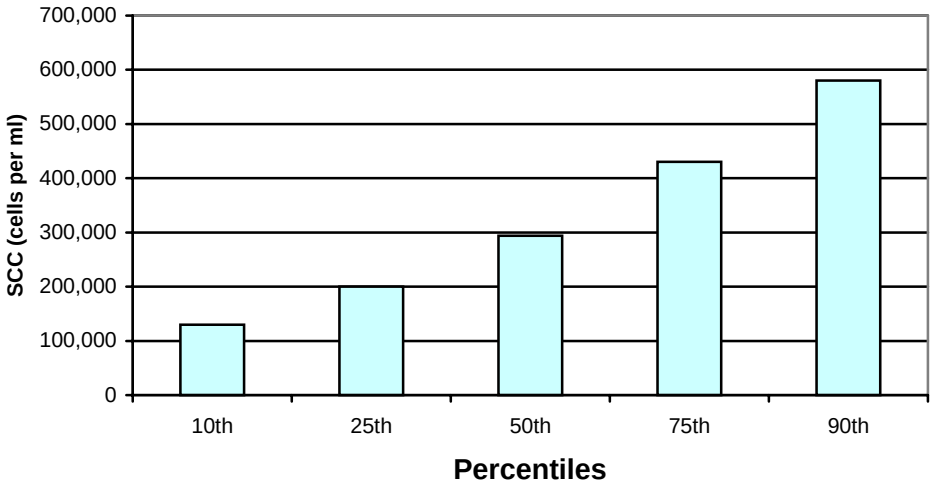

Figure 2. Frequency of Isolation from Clinical Mastitis

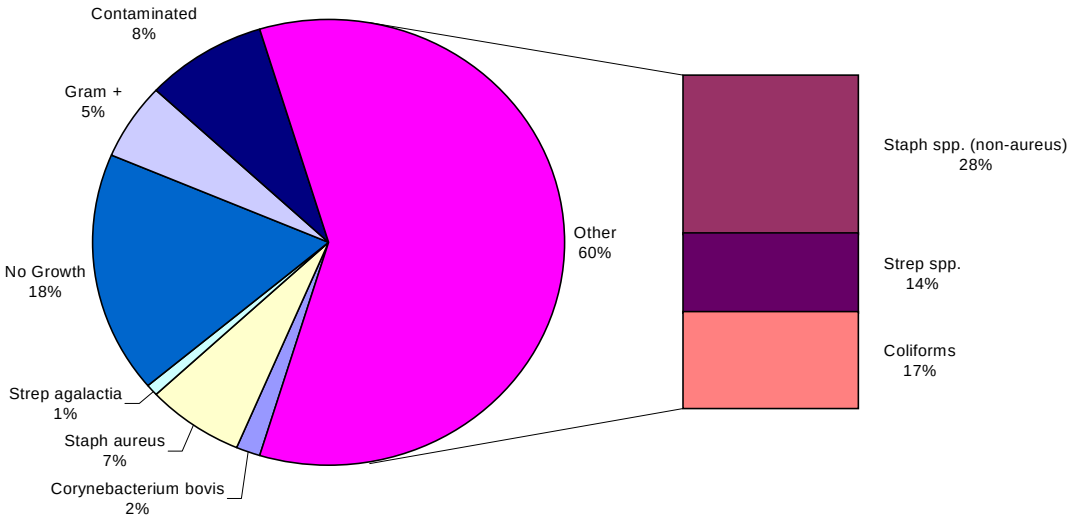

Figure 3. Risk Of Mastitis

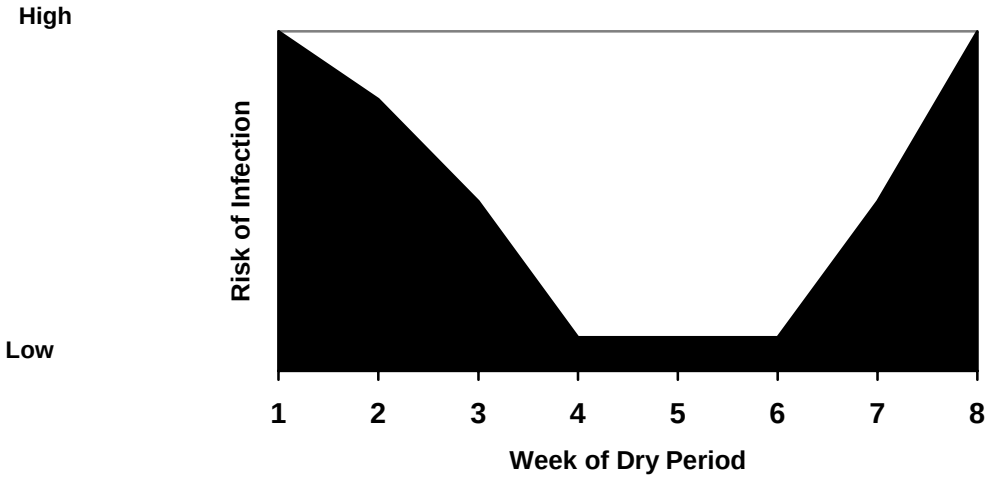

Figure 4. Isolation of Strep spp from High Raw Counts in WA

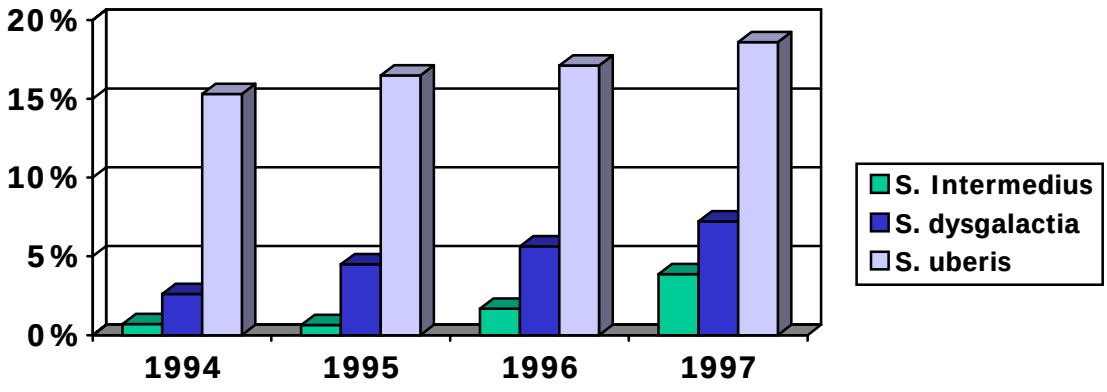

Figure 5. Herds Experiencing New Mycoplama Mastitis