

Environmental Streptococcal and Coliform Mastitis

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Summary

Well managed dairy herds with low somatic cell counts (SCC below 200-300,000) often may experience problems with onsets of clinical mastitis. Approximately 40-45% of the mastitis cases in low SCC herds are caused by environmental pathogens which can be difficult to detect because of their short duration. Cows in low SCC herds are most susceptible to environmental streptococci and coliform infections after drying off and just prior to calving but which appear in early lactation. These infections are usually associated with wet and dirty conditions that expose teat ends to bacterial contamination. Dirty housing and calving environments, certain types of bedding materials, improper or inadequate cow preparation for milking and milk letdown, and conditions within milking systems that create liner slips during milking are all factors that can potentially lead to mastitis. Infections by environmental pathogens can be reduced by dry cow therapy, pre- and post-milking teat dipping, clean teats and udders where hair has been removed, proper preparation for milking, milking system maintenance, fly control, and controlling mastitis in heifers at calving. Pathogen identification and treatment records are important.

Introduction

For years, streptococci were the most prevalent bacteriological cause of mastitis. Prior to the widespread use of antibiotics, especially dry cow therapy, and dipping teats in sanitizing solution, *Streptococcus agalactiae* was the major cause of mastitis. This organism is contagious because it lives only in the udder and is transmitted directly from cow to cow, often through use of a common cloth or sponge for washing teats and udders. However, this organism has been eradicated from many dairy herds. Probably **the most important change** in mastitis epidemiology over the past decade has been the rise in importance of environmental pathogens, primarily coliforms and streptococci other than agalactiae. Today, many well-managed farms that have successfully controlled contagious mastitis, including *Staphylococcus aureus*, and consistently produce milk with somatic cell counts (SCC) below 300,000 have problems with increased clinical mastitis. To illustrate the magnitude of these infections, in 20,478 cows from 274 herds in the Netherlands with SCC below 400,000 (the regulatory limit in Europe), 28.5% of the cows

had clinical mastitis during a year and a half period (41 cases per 100 cows per year). Of these, 42% were caused by environmental pathogens which include the “other” streptococci, and now are referred to as environmental streptococci (primarily *Streptococcus uberis* and *Streptococcus dysgalactiae* but also enterobacter) and coliforms (especially *Escherichia coli* and *Klebsiella* species) (Lam et al., 1997)¹. In a subsequent study involving seven herds with bulk tank SCC below 150,000, 610 cows were cultured every 5-6 weeks, again at dry off and calving, and when clinical mastitis developed. Environmental pathogens comprised 46% of total infections and most of these showed clinical signs (94% of *E. coli* and 64% of environmental streptococci). Environmental pathogens are often responsible for most clinical cases of mastitis but only a few become chronic. *Staphylococcus aureus* was responsible for most of 290 recurrent cases.

In the USA, a survey by the National Mastitis Council reported that in 4,957 cows from 67 herds in 14 states environmental streptococci infections may equal those caused by *S. aureus*: environmental streptococci,

¹ Proceedings of annual meetings of National Mastitis Council, Madison, WI.

² Proceedings of Symposium on Udder Health Management for Environmental Streptococci, University of Guelph, Ontario, Canada, June 22, 1997.

11.5% of cows and 3.9% of quarters; coliforms, 5.0% of cows and 1.3% of quarters; *S. aureus*, 11.5% of cows and 4.0% of quarters; and *Streptococcus agalactiae*, 6.5% of cows and 4.3% of quarters (Hogan and Smith, 1997)². These were selected herds that were involved in research projects, problem herds, or herds who sent samples to diagnostic labs. Usually environmental pathogens will not exceed 10% of quarters with coliforms infecting no more than 2-3%. Heifers in 28 herds from California, Washington, Louisiana, and Vermont were sampled around breeding and within four days of calving (Fox et al., 1995). Infection rates caused by environmental pathogens increased from 1.5 to 7.7% of quarters over this period, while *S. aureus* infections were 2.8%.

Environmental streptococci account for a significant number of subclinical and clinical mastitis infections in both lactating and non-lactating cows. *Strep. uberis* accounted for 82% of these infections (Bramley, 1997)². *Strep. uberis* often produces a chronic mastitis which is unresponsive to antibiotic therapy. Dry cow therapy will control infections that develop early in the dry period. In the absence of antibiotic dry cow therapy, the number of new *Strep. uberis* infections increased markedly, especially during the early dry period and also near calving (Oliver et al., 1996)¹. The prevalence of environmental pathogens in 52 Ohio herds was environmental streptococci, 7.5% of quarters, and coliforms, 1.4% (Bartlett et al., 1992). Hogan and Smith (1997)¹ reported that the prevalence was 3.7% of quarters at drying-off and 7.5% at calving, or 5.5 times greater than during lactation. The prevalence of coliform infection increased from 1.2% of quarters at drying off to 4.5% at calving. Without dry cow therapy, 8-12% of quarters in average infected herds have been found to have environmental infections. Diagnosing the bacterial cause of mastitis in herds with low SCC can be difficult as *E. coli* and environmental streptococci infections are often of short duration (Godkin, 1997)². The average duration is about 12 days; 50% of coliforms last less than 10 days and 70% less than 30 days, but 50% become clinical. Of the environmental streptococci, 60% last less than 30 days but 80-90% become clinical. Several studies have found that one half of environmental pathogen infections become clinical but few become chronic (18% of environmental streptococci exceeded 100 days). Cure rates for environmental streptococci can be high when detected and treated immediately versus a delay of one week. Unfortunately, rapid detection tests do not exist and since DHI SCC are usually conducted monthly, the inability to detect some of these infections is apparent

as they occur between tests. Antibiotics are ineffective against coliforms. The most effective therapy is frequent and complete milkout.

The environmental streptococci show characteristics of contagious mastitis organisms in that they can be resistant to phagocytosis and killing by leukocytes and produce a chronic mastitis which also is unresponsive to intramammary antibiotic therapy. *Strep. dysgalactiae* are sensitive to penicillin and usually eliminated by intramammary therapy (Bramley, 1997)².

With environmental infections, infected quarters swell and become watery (watery milk can be detected with a strip cup). Toxins produced by the bacteria cause body temperature to increase to 104°. Appetite can be depressed and cows lose weight rapidly. Small amounts of serum like secretions can be milked out of the mammary gland and body temperature can drop to 99°. Immediate veterinary treatment is required along with frequent milking.

The economic loss due to environmental mastitis has been estimated at \$107 per clinical case with 88% of the cost resulting from decreased milk yield and discarded milk (about 1,000 lb reduction milk per case and 570 lb milk discarded) and the rest due to increased labor, treatment and veterinary services, premature culling, and reduced genetic improvement. Losses in older cows were twice as much as in first lactation. Cows that developed clinical mastitis suffered an immediate drop in production and did not regain previous production levels during the 60 days following the clinical onset (Bartlett et al., 1991). Cows with coliform infections may survive; however, 10% may die within one to two days.

Monthly visits over one year were made to 52 herds in Ohio. Quarter milk samples were collected twice and 10 sanitation factors were scored in an effort to evaluate the relative importance of environmental and managerial factors as determinants of infection (Bartlett et al., 1992). Increasing prevalence of environmental streptococci infection was associated with: (1) tie stalls; (2) poor sanitation such as wet and dirty conditions; (3) increasing days dry; and (4) liner slips. Washing and drying cows with a cloth decreased infection. Herds with cattle who had good cow dispositions had a lower prevalence. Poor dispositions could adversely affect milk letdown and emptying of the udder by increasing the number of liner slips when cows kick off milking units or reduce the ability of the milker to adequately

prepare cows for milking. Environmental streptococci have been found in bedding materials (especially organic materials), soil, manure, and at different sites on the animal including lips, nares, and vulva, in the rumen, as well as teats and mammary glands. Long straw used in maternity stalls, which has been recommended for years, has been found to be a source of exposure. Problems with cows calving with environmental streptococci have been associated with straw packs that became heavily soiled with feces and urine, especially in Northern U.S. (Smith and Hogan, 1997)². This effect was especially found in poorly ventilated barns. Poorly designed free stall barns contribute to increased incidence of environmental streptococci.

In the Ohio study, increased prevalence of coliform infection was associated with: (1) comparatively larger amounts of milk left in the udder after milking; (2) longer herd milking times; (3) using free stalls in winter (vs. tie stalls) and levels of sanitation in lactating cow housing areas; (4) running water used to wash cows, implying that cows were probably dirty; (5) use of individual wash cloths, an indication of dirty cows; (6) number of audible liner slips which was strongly associated with impacts on the teat end; (7) sawdust or wood bedding; and (8) clipping udders was mildly associated with decreasing prevalence of coliform infection and incidence of clinical mastitis.

The bottom line was the importance of practices that resulted in clean and dry cows, including housing areas, cow preparation and milk letdown, and properly designed and maintained milking systems.

Preventing Environmental Mastitis Infections

The fundamental principle of mastitis control is that the disease is controlled by either decreasing the exposure of the teat ends to potential pathogens or by increasing resistance of dairy cows to infection (Smith and Hogan, 1997)². Environmental streptococci and *E. coli* are able to survive outside the udder indefinitely, although infected quarters may be a reservoir of infection resulting in mammary gland infections developing as a result of milking. The main factor in controlling infection from the environment is to keep cows clean and dry between milkings, minimizing opportunity for teats to become exposed to environmental pathogens. Dirty teats and udders are difficult to properly clean and dry without upsetting the milking routine. Attention should be given to the following:

1. **Provide an environment that will minimize exposure to dirty conditions.** Filthy, damp, or muddy pens or stalls, lots, or pastures continually expose the teat end to a barrage of bacteria. Pasture has had impact on environmental mastitis by reducing risk, but yet exposure is increased when cows have access to lots with limited shade trees, or pastures that are overgrazed, or grazed during periods of heavy rain. If cows are turned out into pastures or loafing lots, well-drained paddocks are preferred, with no access to ponds, swampy areas, or streams and ditches. Loafing lots or pastures should be managed to prevent muddy areas where heifers or older cows might lie down (see Virginia Cooperative Extension Publication 404-252, Dairy loafing lot rotational management system, 1994). Buildup of mud and manure in shaded areas should be avoided. Cows should be fenced out of streams and ponds, and cattle crossings should be built to keep cows out of water and prevent erosion of stream-banks. Calving areas must be clean and dry. They should be cleaned and sanitized after 1-2 calvings, with one box stall per 15-20 cows and bedded with straw, shavings or sand. When cows are confined to free stall barns, adequate dry bedding should be provided to keep free stalls and pens clean, dry, and comfortable. Daily removal of wet and soiled bedding is recommended. Free stalls should be properly designed and maintained. Organic materials used as bedding, e.g., straw, sawdust, and shavings, have been associated with an increase in mastitis. Sand does not support bacterial growth but could be a problem with liquid manure systems. Overcrowding should be avoided regardless of where cattle are housed.
2. **Prevention is enhanced by dry cow therapy.** The dry period is the time of greatest susceptibility to new environmental streptococcal infections. Risk of such infection is five times greater than during lactation (Hogan and Smith, 1997)². There is a good chance that new infections during the dry period will become clinical during the next lactation and then cure rate is only 50%. During the dry period, new infections develop during the two weeks after drying off and two weeks prior to calving (Smith and Hogan)². The rationale for dry cow antibiotic therapy is to eliminate existing infections and prevent new infections. Administration of dry cow treatment must be followed by teat dipping. Barrier teat dips have not been developed that will persist for 7-14 days during early and the late dry period.

3. **Immunization during dry period and early lactation.** Research conducted in California and Ohio showed that vaccination with *E. coli* J5 bacterin at drying off, 30 days after drying off, and within 24 hours of calving reduced the incidence and severity of clinical coliform mastitis during the first 90 days of lactation by four-fold. Although protection was greatest in older cows, positive effects were found in second and third lactation. Bred heifers did not receive the vaccination. When cows' teats were experimentally challenged with *E. coli*, milk production and dry matter intake were significantly depressed more in control than in vaccinated cows.
4. **Keep accurate treatment records.** These records should include information about cows who have been treated for clinical mastitis, including infective organism, quarter(s) treated, antibiotic used, treatment dose and dates, person administering treatment, results of drug screening tests, and how long milk was discarded.
5. **Identify the major pathogens existing in the herd.** Culture milk samples from representative cows in the herd, including all cows when they show clinical mastitis, first lactation and older cows with high SCC (5 score or actual SCC of 300,000 and higher), and cows with chronic mastitis (high SCC on last two consecutive tests, cows whose milk has been discarded for 28-30 days, or cows treated for mastitis at least three times during the lactation). Samples can be frozen and held at least four weeks. Include drug sensitivity to determine potential drug resistance for both lactating and dry cow treatments.
6. **Establish milking orders** that allow infected and treated cows to be milked separately, either through grouping, use of separate milking units, or automatic backflush units.
7. **Use milking procedures that stimulate milk ejection and result in clean and dry teats.** Start with udders that have been clipped or singed and are reasonably clean and free of mud and manure. Milkers should wear rubber gloves and forestrip 4-5 squirts of milk from each quarter which he/she should examine for abnormal milk. Predip with a tested disinfectant and allow 30 seconds contact time. Predipping has reduced environmental streptococcal infections. Applying predip by spraying onto teats, compared to dipping, has caused increased incidence of infection (new cases). Use

a single service cloth towel to scrub and dry teats. This sequence of forestripping, dipping and drying should not be reversed or altered. Teats should not be touched after they have been sanitized and dried. Properly attach and align milking units to clean, dry teats when milk letdown is near peak milk flow by attaching the milking unit within 1 to 1.5 mins after beginning premilking preparation. Remove teat-cups by shutting off the vacuum at the claw. Examine teat ends for chaps, cracks, or lesions, which harbor mastitis-causing bacteria.

Use an effective post-milking teat dip and cover most of each teat (at least the bottom 1/2 to 2/3). Postmilking teat dips have limited effect on the incidence of mastitis infections caused by environmental streptococci and coliform exposures between milkings. Consequently, turning cows into clean and dry free stalls or lots is important, but not until 30-60 minutes after milking. The sphincter in the teat end takes about 30 minutes to close. Practices which keep cows on their feet, such as access to feed, are important. Research has shown that barrier teat dips inhibit bacterial multiplication on the teat skin under the film. In addition to being effective against environmental pathogens, barrier teat dips must be effective against contagious pathogens spread as a result of milking.

8. **Design, maintain and use a milking system that minimizes liner slips.** Environmental pathogens need an active force to penetrate the teat canal that is often provided by milk droplet impacts created by liner slips during milking. Try to minimize conditions that are associated with milk droplet impacts against the teat end, such as liner slips, excessive temporary vacuum losses, low vacuum reserve, inefficient vacuum regulation, and abrupt milking unit removal. Liner slipping early in milking often results from low vacuum level, blocked air vents, or restrictions in the short milk tube. Slipping in late milking is commonly caused by poor cluster alignment, uneven weight distribution in the cluster, or poor liner condition.

Incomplete milkout can be caused by type or condition of liner, mismatch between claw inlet and short milk tube, clusters too light, clusters that do not hang evenly under the udder, or high milking vacuum levels.

Regular preventive maintenance is essential. Vacuum controllers (regulators), pulsators, and air

filters need to be cleaned monthly. Rubber that is cracked, flattened, or otherwise deteriorated should be replaced. Teat cup liners, or short pulsator or milk tubes, should be replaced whenever holes are found in them. The milking system should be evaluated every three months or 500 hours of operation.

9. **Fly control.** Flies carry a number of mastitis-causing organisms that can colonize teat lesions. Incidence of environmental mastitis is higher during summer and fall as is fly infestation. Flies may cause mastitis by biting teats and causing damage that provides an excellent site for colonization. This has not yet been proven but many feel this may be a convincing cause. Elimination of fly breeding sites is one aspect of fly control. Flies breed in decaying feed or manure that has accumulated, including exercise yards, calf pens, and box stalls. Other options include backrubbers, feed additives and ear or tail tags.
10. **Pregnant Heifers.** New infections can be found in many first lactation cows, either at calving or in early lactation. Pregnant heifers should not be grouped with dry cows as the common environment or flies may facilitate new infections in heifers. Heifers may be treated with dry cow treatment at 60 days before expected calving date (Nickerson et al., 1995) or with lactating cow mastitis treatment at 7-14 days before expected calving (Oliver et al., 1992). Both options are considered extra-label use of antibiotics and a veterinarian must be consulted in advance. Before treatment, properly clean and disinfect teat ends with teat dip and alcohol and teat dip again after treatment. Check milk for the presence of antibiotic residues at 3 to 5 days after calving and before milk is put into the milk tank. A third option is to start heifers through the milking parlor 2-4 weeks before expected calving and milk them for 1-3 weeks prior to calving. Calves born to these heifers will need fresh or frozen colostrum from older cows.
11. **Nutrition.** Supplementation of the diet with vitamin E and selenium, vitamin A and beta-carotene, and balancing dietary copper and zinc content to meet requirements have reduced mastitis. Selenium injections (4.5 mg/100 lb. body weight) may be given at 21 days before expected calving and the ration of bred heifers supplemented with vitamin E. Together these practices have reduced mastitis in early lactation more than either practice alone (Hogan et al., 1993). The effect of vitamin E on

clinical mastitis was more pronounced for first lactation than older cows (Weiss et al., 1997). Vitamin E levels of at least 1,000 IU/day during the dry period and 500 IU/d during lactation were more beneficial than National Research Council's recommended 100 IU/d. Selenium was added to the ration to provide 0.1 ppm, which resulted in plasma selenium levels lower than accepted as adequate. Selenium also may be added to dry cow and milking herd rations (3 and 6 mg/head daily, respectively). Vitamin E and selenium also reduced retained placenta, metritis, and cystic ovaries. Cows should have access to fresh feed as soon as they leave the milking parlor.

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