Environmental Streptococcal Mastitis: Facts, Fables, and Fallacies[1]

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Take Home Messages

Rate of environmental streptococcal IMI is greatest during the first two weeks of the dry period and two weeks prior to calving compared with other stages of lactation.

Treating all quarters of all cows with dry cow therapy reduces new IMI two weeks after drying off, but has no effect on new IMI prior to calving. Greatest benefit of using internal teat sealant is the reduction in new environmental strep IMI the two weeks prior to calving.

Using straw bedding and manure packs expose teats to extremely large populations of streptococci. Use of sand bedding reduces exposure to streptococci

Introduction

The prevalence of intramammary infections (IMI) and incidence of clinical mastitis have been measured in a host of surveys during the last 25 years. Environmental streptococci have consistently been reported as a leading cause of both subclinical and clinical mastitis throughout the world (11, 24, 38, 51, 55). A large proportion of variability in incidence and prevalence of environmental streptococcal mastitis, both between geographical locations and within a single herd, can be ascribed to a number of independent variables identified in these surveys. Among these factors were season of the year, stage of lactation, parity, and various management practices (11, 24, 38, 51).

Dynamics of Intramammary Infections

The dynamics of environmental streptococcal mastitis in an experimental herd over a 7 year period were reported (51). The dry period was identified as the time of greatest susceptibility to new environmental streptococcal IMI. The rate of environmental streptococcal IMI was 5.5-fold greater during the dry period than during lactation. Rate of streptococcal IMI was .00312/cow-day during the dry period and .00054/cow-day during lactation. A new streptococcal IMI occurred in 5.6% of quarters during the dry period. A total of 6.3% of quarters became infected

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during lactation.

Rate of environmental streptococcal IMI was influenced by season of the year, stage of lactation and parity. Rate of IMI was greater in the Summer and Fall than during Spring and Winter for both lactating and dry cows. Rate of IMI during the dry period was greater in multiparous cows compared with primiparous cows. An interaction effect on rate of IMI was found between stage of lactation and parity. Primiparous and cows in lactation 4 or greater had higher rates of IMI than did cows in the second and third lactations during the first month of lactation. Rates of IMI did not differ among parity groups at 31 to 150 d of lactation. Rates of IMI in cows in their fourth or greater lactation were greater than in other parity groups the last half of lactation.

Environmental streptococcal IMI tended to be short duration infections with only a relatively few becoming chronic. Geometric mean duration of environmental streptococcal IMI was 12 d with a range from 1 to 370 d. Approximately 41% of IMI had duration of <8 d and only 15% lasted greater than 90 d. The two primary means that IMI were eliminated were spontaneous cure (45.7% of IMI) and antibiotic treatment of clinical cases (33.2% of IMI).

Dynamics of Clinical Mastitis

Approximately one-half of environmental streptococcal IMI cause clinical mastitis during lactation (51). The percentage of environmental streptococcal IMI that caused clinical signs ranged from 42% to 68% in the same herd during different years. Severity of clinical signs is generally limited to local inflammation of the gland. A total of 43% of clinical cases had signs limited to abnormal milk, 49% involved abnormal milk and swollen gland, and only 8% involved systemic signs such as fever and anorexia.

During lactation, the incidence of clinical mastitis was greatest the first week after calving and decreased throughout the first 305 days in milk. Interestingly, rate of environmental streptococcal clinical cases increased in cows with extended lactations (>305 d) to that comparable of cows in peak lactation (24). Therefore, the use of management practices that encourage the use of extended calving intervals, thus a larger percentage of cows with >305 days in milk, may impact the prevalence of environmental streptococcal IMI in a herd.

Exposure

Exposure of uninfected teats to the environmental streptococci can occur during the milking process, between milkings, during the dry period and prior to parturition in first lactation heifers. This is in contrast to the contagious pathogens where exposure occurs primarily during the milking process (3). The environmental streptococci, Streptococcus uberis in particular, have been isolated from bedding materials, soil, rumen, feces, vulva, lips, nares, mammary gland and teats (2, 5, 6, 10, 23, 25, 28, 42). Feedstuffs such as silages may also be a source of these pathogens and infections of the reproductive tract may contribute to environmental and teat end contamination (10). Despite reports that suggested that dynamics of Streptococcus uberis IMI within a herd fit mathematical models representative of contagious pathogens (56, 57), epidemiological typing of isolates virtually exclude any cow to cow transmission of

Streptococcus uberis (53).

Bedding materials serve as a primary source of environmental streptococcal exposure of teat ends as the teats and udders are in frequent, direct contact with the bedding materials. In general, all bacterial populations are lower in inorganic bedding materials such as sand compared with organic bedding materials (3, 23). The numbers of environmental streptococci in sand bedding will vary depending upon the amount of soil in the sand and the degree of fecal contamination once the sand is placed in the stalls. Washed sand is the bedding of choice and maintenance of stalls is critical in order to achieve low numbers of pathogens in the bedding.

The numbers of environmental streptococci in organic bedding materials vary with the type of bedding. Whereas sawdust and wood products in general tend to emphasize the Gram-negative or coliform bacteria, large numbers of environmental streptococci are often found in straw bedding. Bramley (2) was able to show that straw bedding was an excellent source of Str. uberis. Rendos et al. (42) determined the numbers of environmental pathogens in straw, sawdust and wood shavings used to bed lactating dairy cows and also determined the numbers of these pathogens contaminating teats. Populations of the environmental streptococci were greatest in straw and significantly lower in sawdust and shavings. Numbers of streptococci recovered from teats by swabbing were highest in straw bedded cows. Numbers recovered from teats of cows bedded on sawdust were lower than cows bedded on shavings. Recycled newspaper and pelleted corn cobs have also been associated with substantial populations of the environmental streptococci and teat contamination (20).

Long straw used in maternity stalls or as bedding for loose housed cattle can be the source of considerable exposure of teats to the environmental streptococci (2, 12, 23). Problems with cows calving with environmental streptococci are often associated with cows calving on straw bedding packs that are heavily soiled with feces and urine. Herd problems with high somatic cell counts not associated with the contagious pathogens are frequently the result of high levels of infection caused by the environmental streptococci. Such herds are often found to have cattle in loose housing bedded with deep straw packs, again heavily soiled by feces and urine. In the northern part of the US these problem herds are often associated with poorly ventilated barns and often occur in late spring when outside temperatures are warming, before cows go out to pasture or before barns can be thoroughly cleaned.

Clearly, housing facilities and management practices on the farms contribute to the contamination of bedding materials and the exposure of teats to the environmental streptococci (12, 48, 49). Poorly designed facilities can contribute to increased incidence of environmental streptococcal mastitis. Facilities should be designed to maximize cow comfort and minimize stress and physical injuries during all seasons of the year. Ventilation is critical to maintaining dry conditions and frequently is poor in older facilities. Ventilation problems of older facilities often require great expense to correct or may not be correctable.

Many free-stall barns are poorly designed and contribute to increased incidence of environmental mastitis. In well designed free-stall barns, cows will either be eating or lying down resting. Large numbers of cows standing around or lying in alleyways generally indicate improper design

of the free-stalls or sever overcrowding. Free-stalls built against outside walls or against any solid wall should be avoided as solid walls block the free flow of air, minimize lunge space for cows when attempting to rise and may inhibit stall usage. A slope of 2% to 3% across the building reduces accumulations of urine and water and reduces teat injuries in free-stalls and tie-stalls. A commonly recommended practice in the US is to overstock free-stall barns by 10% with some recommendation up to 20%. We are unaware of any studies designed to determine the impact of such practices on the incidence of environmental streptococcal mastitis but would suggest that overstocking will increase the incidence.

Pastured cows are generally thought to be at reduced risk for environmental streptococcal mastitis when compared to cows in confinement housing. However, conditions do exist in pastures that can lead to high levels of exposure to the environmental streptococci (16). Areas under shade trees can produce conditions of high exposure and pastures that are over grazed or grazed during periods of heavy rain may also lead to conditions of exposure similar to housed cattle. The environmental streptococci are the most significant environmental pathogen in New Zealand dairy herds where cows spend virtually 100% of there time on pasture (38, 54). Additional research on the association between pasture conditions and teat contamination with environmental pathogens would be beneficial.

Improper milking time hygiene and machine function can contribute to environmental streptococcal mastitis (13, 35, 36). The key is to milk clean, dry teats and udders with a properly functioning milking machine (37). An improperly functioning milking machine or improper use by the milker can contribute to increased amounts of environmental streptococcal mastitis in a dairy herd but correcting such faults will not solve environmental streptococcal mastitis in all dairy herds.

Predipping has been shown to reduce new environmental streptococcal infections during lactation by as much as 50% in some herds (35, 36, 37), although this reduction is not observed in all herds (17). Failure of predipping to control environmental streptococcal mastitis in all herds likely reflects the complex epidemiology of environmental streptococci. Post milking teat disinfection with germicidal dips is generally considered not to control environmental streptococcal mastitis (3, 9, 11, 37, 46, 48). However, several studies indicate that environmental streptococcal infections are lower in dipped cows compare to undipped cows (3, 9, 11, 33) and that germicidal dips may reduce teat skin contamination to some extent (14). An exception is the work of Hogan et al. (18) that found environmental streptococcal infections did not significantly differ between dipped and undipped herds. Barrier dips are recommended by some individuals as a means of controlling environmental mastitis but their efficacy against the environmental streptococci is equivocal (21, 34, 48).

Cow Resistance to IMI

Risk of new environmental streptococcal infection is influenced by stage of lactation, parity, nutrition, and immunity in addition to factors that increase teat end exposure. The importance of the dry period in control of environmental streptococcal IMI can not be over emphasized (15, 29, 30, 31, 41, 45, 46, 51). Rate of new IMI during the dry period was reported to be 5.5 fold greater

than the rate during lactation (50) in a total confinement herd practicing total dry cow therapy. The rate of new infection is not constant across the dry period, but is elevated during the 2 weeks following drying off and the 2 weeks prior to calving (45). Dry cow therapy reduces the rate of new environmental streptococcal IMI during the early dry period (1, 8, 22, 45, 54). The high rates of new infection following drying off may relate to the lack of flushing action due to milking (45), changes occurring in the composition of the mammary secretion that appear to enhance in vitro streptococcal growth (27, 52), and/or the lack of a keratin plug in the streak canal (4, 8, 54).

The increase in susceptibility to infection in the two weeks prior to parturition may reflect the absence of milking when the gland is accumulating fluid, loss of keratin plugs from streak canals, or immunosuppresion associated with the pariparturient period (47, 48, 49). Conventional dry cow therapy has no effect on rates of infection prior to calving (45) and prepartum teat dipping was reported to be of little or no value (43). The environmental streptococci are a frequent cause of mastitis in heifers at calving and heifers are generally as frequently infected at calving as cows having undergone a dry period (24, 32, 38, 46, 51).

Several studies have reported that infection by the minor pathogens Corynebacterium bovis and the coagulase negative staphylococci can prevent subsequent infection with the major pathogens (7, 39, 40). On the other hand, Pankey et al. (39) reported that C. bovis infected quarters challenged by immersing in broth cultures of Staphylococcus aureus or Streptococcus agalactiae were 8.5-fold more susceptible to Str. agalactiae infection but 50% more resistant to S. aureus infection. Hogan et al. (19) looked at rates of environmental streptococcal IMI in quarters either infected with C. bovis or uninfected during periods of low and high prevalence of C. bovis IMI. Challenge was by natural exposure. Rate of environmental streptococcal IMI was 3.9 times greater in C. bovis infected quarters than in uninfected quarters. A comparable increase in superinfections of C. bovis infected quarters with Str. uberis was found by Berry and Hillerton (1). These studies strongly suggest that C. bovis infected quarters represent a significant risk factor for environmental streptococcal infections.

Work of the past 20 years has clearly demonstrated that diets of dairy cows can influence the resistance of cows to intramammary infection (47, 48, 49). Specific components of diets that have been shown to be important are the vitamins E, A, and beta-carotene and the trace minerals selenium, copper and zinc. Evidence clearly shows that vitamin E and selenium influence phagocytic cell function and cows fed diets deficient in either component are at greater risk of environmental streptococcal mastitis (47). The risk of low blood and tissue concentrations of vitamin E and selenium appears to be greatest around calving, a period of known high susceptibility to the environmental streptococci.

Commercial vaccines that can be used to successfully immunize cows and control environmental streptococcal mastitis are not yet available (26).

Conclusions

Environmental streptococci have emerged as pathogens that uniquely affect mammary health as

a primary cause of both subclinical and clinical mastitis. The bovine mammary gland appears to be most susceptible to environmental streptococcal IMI during the dry period and early lactation. Resistance of cows is critical to the control of environmental streptococcal mastitis and there is little doubt that healthy teat ends and a properly functioning immune system are critical to the maintenance of low numbers of infected quarters and the elimination of infections that do occur. As cows are bred for greater and greater milk production, their susceptibility to intramammary infection is increasing (44). Clearly, manipulation of the resistance mechanisms of cows requires additional research but such research will be slow to develop given dwindling resources for mastitis research. On the other hand, exposure of teat ends to the environmental streptococci can overwhelm good immunity under most conditions. Effective control of the environmental streptococci in the short term is most likely to be achieved by reducing teat end exposure. Exposure is the major risk for environmental streptococcal mastitis in todays dairy herds and we need to continually learn ways to keep cows clean, dry, cool and comfortable.

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