

ENVIRONMENTAL MASTITIS CAUSED BY SPECIES OF STREPTOCOCCUS AND ENTEROCOCCUS: RISK FACTORS AND CONTROL

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Introduction

The environmental streptococci and their cousins the enterococci are a significant cause of intramammary infection (IMI) and clinical mastitis in dairy herds and methods to control these pathogens are currently less than adequate (13, 44, 45). *Streptococcus uberis* is the streptococcal species most often encountered and this is true for several studies conducted in the US, Europe, New Zealand and Australia.. The *Enterococcus* species most often isolated are *E. faecium* and *E. faecalis*. These environmental streptococci and enterococci have been isolated from the intestinal tract, manure, infected udders, and the general dairy environment. The enterococci are generally only a minor component of the environmental streptococcal/enterococcal mastitis complex and a major herd problem caused by the enterococci is relatively rare. Currently, control of mastitis caused by *Enterococcus* species is thought to be achieved by those practices used to control *Streptococcus uberis*.

Regardless of the pathogen the fundamental principle of mastitis control is that the disease is controlled by either decreasing the exposure of teat ends to potential pathogens or by increasing the resistance of dairy cows to infection (2). Conversely, factors that increase teat end exposure or reduce the resistance of cows to infection are very likely to result in greater mastitis in the herd and are considered to be risk factors. This paper will attempt to identify and discuss several of these risk factors known to be associated with mastitis caused by the environmental streptococci.

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 (2). The environmental streptococci and *Streptococcus uberis* in particular, have been isolated from bedding materials, soil, rumen, feces, vulva, lips, nares, mammary gland and teats (1, 4, 5, 9, 17, 24, 26, 40). Feed stuffs such as silages may also be a source of these pathogens and infections of the reproductive tract may contribute to environmental contamination (9).

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 to organic bedding materials (2, 17). 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. Some sand bedded herds report that the environmental streptococci/enterococci are the major contributors to herd mastitis.

The numbers of environmental streptococci in organic bedding materials vary with the type of bedding. Where as 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 (1) was able to show that straw bedding was an excellent source of *Str. uberis*. Rendos et al. (40) 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 (19). There are no studies where the numbers of enterococci have been determined and this is due in large part to the lack of a selective media that would block the growth of streptococcal species.

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 (1, 11, 17). 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 (11, 44, 45). 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. Seasonal effects on environmental streptococcal mastitis have been reported in some (47, 48) but not all studies (17, 18)

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 severe 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/enterococcal mastitis but would suggest that overstocking will increase the incidence. There is clear need for additional studies on the relationships/interactions among housing design, management systems, and bacterial populations in the environments of housed dairy cows.

Cows housed in corrals with shades may be at increased risk of environmental streptococcal/enterococcal mastitis during periods of rainy weather. Hot dry conditions should favor low exposure. Areas under shades would be an obvious area of risk. Attempts to reduce this risk by “grooming”, removing the damp material and replacing with dry material from the corral, are probably a positive factor. However, the area under shades are in fact loose housing on a manure pack and could represent a significant degree of exposure. Such exposure would likely be exacerbated by over crowding.

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/enterococci (14). 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 led 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 (36, 51). 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 (12, 33, 37). The key is to milk clean, dry teats and udders with a properly functioning milking machine (34). 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 (33, 34, 37), although this reduction is not observed in all herds (16). 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 (2, 8, 10, 34, 44, 48). However, several studies indicate that environmental streptococcal infections are lower in dipped cows compare to undipped cows (2, 8, 10, 30) and that germicidal dips may reduce teat skin contamination to some extent (13). An exception is the work of Hogan et al. (23). They (23) found that 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 (20, 29, 44).

Resistance

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 (27, 28, 31, 39, 47, 48, 50). 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 (47). Dry cow therapy reduces the rate of new environmental streptococcal IMI during the early dry period (7, 22, 47, 51). The high rates of new infection following drying off may relate to the lack of flushing action due to milking (47), changes occurring in the composition of the mammary secretion that appear to enhance *in vitro* streptococcal growth (25, 49), and/or the lack of a keratin plug in the streak canal (3, 7, 51).

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 immunosuppression associated with the pariparturient period (43, 44, 45).

Conventional dry cow therapy has no effect on rates of infection prior to calving (47) and prepartum teat dipping was reported to be of little or no value (41). The environmental streptococci are a frequent cause of mastitis in heifers at calving and heifers are generally as infected at calving as cows having undergone a dry period (18, 32, 36, 48, 50).

During lactation the rate of new IMI is not constant but is highest early in lactation and decreases as lactation advances (48). Todhunter et al. (50) reported that the rate of new environmental streptococcal IMI was greater during the first month of lactation than during the remainder of lactation. Parity has been shown to be a risk factor for environmental streptococcal IMI. Smith et al. (48) found that rate of IMI increased as parity increased. Todhunter et al. (50) found that rate of IMI increased as parity increased, however rate of environmental streptococcal clinical mastitis during lactation was elevated in first lactation cows compared to cows in lactations 2 and 3. The rate of IMI during late lactation was higher for older cows than for either heifers or cows in second lactation (50). Rates of clinical mastitis were greatest in lactation 4 or greater cows. Hogan et al (18) reported the highest rates of environmental streptococcal clinical cases in first lactation cows in a study of 9 well managed dairy herds.

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 (6, 35, 38). On the other hand, Pankey et al. (35) 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. (21) 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. These studies strongly suggest that *C. bovis* infected quarters represent a significant risk factor for environmental streptococcal

infections.

Work of the past 10 years has clearly demonstrated that diets of dairy cows can influence the resistance of cows to intramammary infection (43, 44, 45). Specific components of diets that have been shown to be important are the vitamins E, A, and β -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 (43). The risk of low blood and tissue concentrations of vitamin E and selenium appears to be greatest around calving, a period of know high susceptibility to the environmental streptococci.

No vaccines are yet commercially available that can be used to successfully immunize cows and control the environmental streptococci. A *Str. uberis* vaccine is being tested in England (15) and may be of value in environmental streptococcal mastitis control.

Check list for herds experiencing environmental streptococcal/enterococcal mastitis problems.

1. Environment

a. Corrals

- i. Avoid damp areas under shades
 - (1) Daily grooming
 - (2) Dry material in and wet out
- ii. Avoid overcrowding under shades
- iii. Avoid areas that accumulate water following rain
 - (1) Proper grading with slope
- iv. Avoid damp muddy areas around water troughs
- v. Consider sand bedded freestalls under shades

b. Calving areas

- i. Well bedded, clean and dry box stalls
- ii. Separate calving area and sick cow housing
- iii. Use clean sand bedding where possible and avoid organic bedding materials when possible
- iv. Clean stalls frequently and avoid a build-up of damp areas

2. Milking time hygiene

a. If washing occurs in prep-pen

- i. Fore strip to check for clinical mastitis
- ii. Attach units to clean dry teats and udders

b. If no washing in prep-pen

- i. Fore strip to check for clinical mastitis
- ii. Pre-dip/spray
 - (1) Use effective germicidal product
 - (2) Provide a minimum of 20 sec. contact time

- (3) Check for complete teat coverage by the dip/spray
- iii. Thoroughly dry teats with individual paper/cloth towels
- iv. Attach units to clean, dry teats

3. Post milking teat dipping/spraying

- a. Use an effective (tested) germicidal product
- b. Check for adequate teat coverage (teat spraying often results in poor teat coverage)

4. Dry cow therapy program

- a. Dry treat all quarters of all cows
- b. Use FDA approved products labeled for use in dry cows
- c. Administer dry cow therapy using aseptic technique and partial insertion of cannulas
- d. Dip teats in an effective germicidal teat dip

5. Clinical mastitis detection and therapy

- a. Check all quarters of all cows at each milking by fore-stripping
 - i. Lactating strings
 - ii. Newly calved cows and heifers
- b. Treat obviously clinical quarters
 - i. Use FDA approved products according to the label instructions
 - ii. Administer drugs using aseptic technique and partial insertion of cannulas

6. Determine vitamin E and selenium status of lactating cows and both cows and heifers at calving.

- a. Check diets
 - i. Dry cows should receive 1000 IU supplemental vitamin E during the dry period and 400 to 600 IU of E during lactation
 - ii. Typical Holstein cattle need to consume 6-7 mg/day selenium
- b. Check blood concentrations around parturition
 - i. α -tocopherol concentration in blood plasma should be at least 3.5 $\mu\text{g/ml}$
 - ii. Selenium concentration should be at least .075 $\mu\text{g/ml}$ in serum and .2 $\mu\text{g/ml}$ in whole blood

Conclusions

The risk of environmental streptococcal/enterococcal mastitis is a function of exposure of teat ends to the pathogen and the resistance of cows to infection. Resistance of cows is critical to the control of environmental streptococcal/enterococcal 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 (42). 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/enterococci in the short term is most likely achieved by reducing teat end exposure. For cows housed in corrals under Arizona conditions, the area under shades likely represents a major source of exposure to these pathogens. We would contend that exposure is the major risk for environmental streptococcal/enterococcal mastitis in today's dairy herds and we need to continually learn ways to keep cows clean, dry, cool and comfortable. Research that would determine the microbiological environment associated with the area under shades could lead to alternate management strategies and improved environmental mastitis control. Such research will not be easy to fund given today's attitudes that only biotechnology oriented research projects are appropriate for funding.

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