ENVIRONMENTAL MASTITIS: KNOW YOUR OPPONENT

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Introduction

Summertime and the living is easy and this is particularly true for a large group of microorganisms that are present in the environments of all dairy herds. These microorganisms are collectively referred to as the environmental pathogens and they thrive on high temperatures, high humidity, organic bedding materials and they simply love overcrowded, poorly ventilated facilities. The environmental pathogens are major contributors to cases of clinical mastitis and are the primary pathogens responsible for elevated bulk tank somatic cell counts in well managed herds that have successfully controlled the contagious pathogens *Staphylococcus aureus* and *Streptococcus agalactiae.* While producers are confronted with environmental mastitis year round, summer time can be particularly troublesome for increased incidence of clinical mastitis cases. Bulk tank somatic cell counts are almost always higher in summer months compared to winter months and likely represents increased numbers of infections caused by the environmental pathogens. The good old summertime may be the season of choice for environmental pathogens but summertime can often be very unfriendly to dairy cows. Heat stress is common and may reduce the cows resistance to environmental pathogen infections. Surviving the summer months means controlling environmental mastitis and producers need to manage their cows and facilities to minimized exposure to the environmental pathogens.

The Environmental Pathogens

The environmental pathogens represent a very large group of organisms that are present in the environments of cows and can directly infect cows or contaminate products placed on teats (teat dips) or infused into mammary glands such as multiple use containers of antibiotics (20,22). The environmental pathogens most frequently encountered are species of streptococci other than *Str. agalactiae* and the Gram-negative bacteria, primarily the coliform bacteria. Significant streptococcal and enterococcal species include: *Str. uberis*, *Str. dysgalactiae*, *Str. equinus*, *Enterococcus faecalis, Ent. faecium.* Gram-negative bacteria include: *Escherichia coli, Klebsiella spp., Enterobacter* spp., *Serratia* spp., *Pseudomonas* spp., *Proteus* spp. and *Citrobacter* spp. Other environmental pathogens include *Arcanobacterium pyogenes*, *Nocardia* spp. *Bacillus* spp, yeasts, molds and algae. The following discussion centers largely around the environmental streptococci and the coliform bacteria as they are responsible for the majority of environmental pathogen IMI in dairy herds and are of greatest economical significance.

Infection Characteristics

Infections caused by the environmental pathogens are generally of short duration by comparison to the contagious pathogens, are more likely to result in clinical mastitis, and less likely to cause herd problems associated with subclinical mastitis such as high bulk milk SCC (7,18,20). The

majority of environmental streptococcal IMI are present for less than 30 days, and prevalence of IMI at any point in time seldom exceeds 10-15% of quarters in a dairy herd (22,24). However, bulk milk SCC in excess of 750,000 are occasionally linked to a high prevalence of infection caused by the environmental streptococci. Approximately 20% of environmental streptococcal IMI will exceed 100 days duration and chronic infections that are unresponsive to antibiotic therapy do occur. Forty to 50% of environmental streptococcal IMI will be associated with clinical symptoms (22,24). Clinical symptoms are frequently moderate but seldom severe (11,24).

Coliform infections are frequently very short duration with over half of these IMI lasting less than 10 days (16,19,22,23). As a result, prevalence of IMI seldom exceeds 1-2% of quarters at any one time in a dairy herd and coliform IMI are seldom the cause of high bulk milk SCC. Eighty to 90% of coliform IMI present in lactation will result in clinical signs. A high proportion will be associated with moderate symptoms and approximately 10% will result in severe clinical mastitis case (6,22,25). Many studies clearly show that the coliform bacteria are isolated from 30-40% of clinical cases in modern dairy herds and particularly so in total confinement dairy herds. Among the coliform bacteria, *E. coli* are generally the predominant cause of IMI and chronic long duration *E. coli* IMI are rare. *Klebsiella* spp. are responsible for IMI in many herds, particularly those using sawdust for bedding (2,8,12). Some *Klebsiella* spp. IMI do become chronic and can be present for several months and from one lactation to the next. Infections caused by *Serratia* spp., *Pseudomonas* spp., *Proteus* spp., and *Citrobacter* spp. tend to be of longer duration than those IMI caused by *E. coli.* They are generally isolated from less than 10% of clinical cases in dairy herds (11,25). An occasional herd is found to have a major problem with one of these latter genera and the problem is often found to be a point source such as contaminated water or a contaminated teat dip.

Infections caused by *A. pyogenes* are generally present in less than 1% of quarters in a dairy herd. Such infections have been termed "summer mastitis" and are known to be of particular problem in England and northern Europe (9). These infections frequently cause severe clinical cases of mastitis that are refractory to antibiotic therapy.

Primary Reservoir in Dairy Herds

The major reservoir of environmental pathogens is the cows environment and those factors in the environment that result in contamination of teat ends, likely contribute in a major way to the number of environmental pathogen IMI in a dairy herd. Sources of environmental pathogens include manure, dirt, mud, pools of standing water, feed stuffs, and bedding materials (1,9). Bedding materials are a significant source of environmental pathogens in dairy herds and are in frequent and prolonged contact with teat ends (12,18,20). Both environmental streptococci and coliform bacteria survive and grow in the organic bedding materials commonly used in dairy herds. Straw bedding has been shown to support very large populations of *Str. uberis*, while sawdust, shavings, and recycled manure solids as well as solids from methane generators generally elevate exposure to *E. coli* and *Klebsiella* spp.. Populations of both environmental streptococci and coliform bacteria are almost always lower in inorganic bedding such as sand or crushed limestone when compared to organic bedding materials (12).

The origin of environmental pathogens can also be multiple dose containers of antibiotics, or contaminated syringes, cannulas, and needles. Poor intramammary infusion technique together with improper cleaning of the teat end may also introduce environmental pathogens into the mammary gland.

Physiological and Environmental Factors Influencing Herd Infection Patterns

Many IMI caused by the environmental pathogens originate in the dry period (3,11,21,23,24). Rates of IMI are generally higher during the dry period than during lactation. Rate of IMI is not constant across the dry period but elevated during the 2 weeks following drying-off and the two weeks prior to calving. New *E. coli* IMI during the early or mid dry period are very rare and the vast majority of *E. coli* IMI present in lactation, and originating in the dry period, occur during the 7 to 10 days prior to calving (21). New IMI caused by the other Gram-negative genera appear to be evenly distributed between the early and late dry period (21,23).

Rates of IMI and prevalence of IMI during lactation are highest during the first month of lactation and decline progressively as lactation advances (22). Incidence of clinical cases caused by the environmental pathogens is also highest during the first month of lactation (19,22,23,24). These stage of lactation effects are generally more pronounced with the coliform bacteria than the environmental streptococci.

Parity has been reported to influence the rate of environmental pathogen IMI in dairy herds. Rate of environmental pathogen IMI is generally lowest in heifers and highest in the older cows during both the dry period and lactation (23,24).

The rate and prevalence of environmental pathogen IMI is higher in housed than pastured cows. However, muddy conditions in pastures or areas where cows congregate under shade trees can contribute significantly to environmental mastitis in a dairy herd. Patterns of environmental mastitis in herds are influenced by season of the year (8,22). Rates of IMI and incidence of clinical mastitis cases will increase during summer months in confinement housed dairy herds (20,22,23,24). In some areas where cattle are exposed to the elements, environmental mastitis will increase during rainy seasons. The increased rate of environmental mastitis during summer months is thought to be associated with increased numbers of pathogens in the environment and increased exposure of teat ends.

Significant Control Methods

Control of environmental pathogens is achieved by decreasing exposure of teat ends to the pathogens and by increasing the resistance of cows to IMI (9,18,19). Reducing teat-end exposure to environmental pathogens requires attention to all aspects of the environment. Four different environments on dairy farms generally influence teat-end exposure to environmental pathogens: 1) dry cow and close-up heifer housing; 2) the calving area; 3) lactating cow's environment; and 4) the milking parlor or milking time hygiene. Clean, dry conditions should be emphasized in all areas of the dairy cow's environment. Bacteria require food, moisture, and proper temperature to survive and multiply. Eliminating moisture may be one of the more productive ways to reduce the number of environmental pathogens and cases of environmental

mastitis in the dairy herd (12). This particularly applies to bedding materials.

Sand is the ideal bedding material from a bacteriological standpoint (12). Numbers of coliform bacteria, particularly *Klebsiella* spp., and environmental streptococci found in sand or crushed limestone nearly always are lower than numbers found in organic bedding materials. All organic bedding materials support large populations of coliform bacteria and environmental streptococci. In general, wood products such as sawdust and shavings are associated with coliform numbers in excess of 1 million per gram of bedding (2). Straw bedding, long or chopped, generally is associated with large numbers of environmental streptococci (12) and particularly *Streptococcus uberis* (1). When inorganic materials cannot be used, every effort should be made to keep organic bedding as clean and dry as possible. Wet or damp areas in the back one-third of free stalls and tie stalls will lead to increased exposure to environmental pathogens. Deep bedding packs of straw and manure, frequently used in loose housing of dry cows, often are associated with increased environmental streptococcal mastitis at calving. Sawdust used in the calving area likely will lead to increased incidence of coliform clinical cases at calving or early in lactation.

Poorly designed facilities can contribute to increased incidence of environmental mastitis (19). 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 severe overcrowding. Free-stalls built against outside walls or any solid wall should be avoided as solid walls 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 recommendations up to 20%. We are unaware of any studies designed to determine the impact of such practices on the incidence of environmental mastitis but would suggest that overstocking will increase the incidence of environmental mastitis.

Pastured cows are generally thought to be at reduced risk for environmental mastitis when compared to cows in confinement housing (8,9). On the other hand, conditions do exist in pastures that can lead to high levels of exposure to environmental pathogens (8). 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. 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 an environmental mastitis problem. The key is to milk clean, dry teats and udders with a properly functioning milking machine (17). Predipping has been shown to reduce new environmental mastitis infections during lactation by as much as 50% in some herds, although this reduction is not observed in all herds. Failure of predipping to control environmental mastitis in all herds likely reflects the complex epidemiology of environmental pathogens. Milking time hygiene is also important to

prevent the contamination of the milk supply with potential human pathogens present in the cows environment.Post-milking teat dipping with germicidal products will not control environmental pathogens and barrier dips are of limited value (10).

Dry cow therapy is of limited value for control of environmental mastitis but has been shown to significantly reduce the rate of new IMI by the environmental streptococci during the early dry period (3,21,23,24).

Diets can influence the resistance of cows to mastitis and several studies document the importance of dietary vitamin E and the trace mineral selenium (4,5,14,15,26). Diets deficient in vitamin E and/or Se are associated with higher incidence of the environmental pathogen IMI and increased clinical cases. Vaccination with a mutant strain of *E. coli* (*E. coli* J5) has reduced incidence and severity of clinical cases caused by the Gram-negative bacteria (13,16,25). The vaccines have not been demonstrated to reduce rate of new IMI. No vaccine is currently available for control of any of the environmental streptococci.

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