Environmental Mastitis: An Everyday Affair

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Introduction

Bovine mastitis continues as a major disease problem of dairy cattle (1). Improved control is needed to maximize dairymen's profits and to improve the quality of milk to consumers. Development of a single uniform method of control has been difficult as mastitis is not caused by a single pathogen but is a disease involving multiple microorganisms (primarily bacteria) which can and do infect the mammary gland. In reality, mastitis is multiple different diseases occurring in the bovine mammary gland.

The primary bacteria associated with mastitis (Table 1) can be grouped according to their primary origin and means of spread within a dairy herd. These pathogen groups are contagious, environmental, and skin flora opportunists. The two major contagious pathogens, *Staphylococcus aureus* and *Streptococcus agalactiae,* exist primarily in infected quarters and are spread to uninfected quarters during the milking process. Progress for control of the contagious pathogens has been achieved (2,3). Implementation of post-milking teat end disinfection and total dry cow therapy can eliminate *Str. agalactiae* from a dairy herd and reduce the prevalance of *S. aureus* infection to less than 1% of quarters.

Table 1. Major causes of bovine mastitis.

Herd Epidemiology	Pathogen	Control by TD[1] + TDCT[2]
Contagious	*Staphylococcus aureus*	Very Good
	Streptococcus agalactiae	Excellant
	Corynebacterium bovis	Excellent
Environmental	Escherichia coli	Poor
	Klebsiella pneumoniae	Poor
	Klebsiella oxytoca	Poor
	Enterobacter aerogenes	Poor
	Serratia spp.	Poor
	Pseudomonas spp.	Poor
	Proteus spp.	Poor
	Streptococcus uberis	Slight
	Streptococcus bovis	Slight
Skin Flora Opportunists	Staphylococcus epidermidis	Moderate
	Staphylococcus hyicus	Moderate
	Staphylococcus xylosus	Moderate

[1]Teat Dipping
[2]Total Dry Cow Therapy

Dry cow therapy functions to reduce the number of quarters infected with contagious pathogens. Effective teat dipping reduces the rate of new infection by the contagious pathogens and the combined impact of teat dipping and dry cow therapy is a significant and progressive reduction of the reservoir of these pathogens in the dairy herd.

Unfortunately, control of contagious mastitis does not mean the elimination of all mastitis from a dairy herd (4). Teat dipping and dry cow therapy simply do not effectively control mastitis caused by the environmental pathogens (5). A major reason for the lack of control is that the primary reservoir of these pathogens is not another infected quarter in the dairy herd, but the environment in which the dairy cow is living. Effective teat dips do kill the environmental pathogens left on teats at the end of milking but the teat ends are continuously exposed to these pathogens between milkings and throughout the dry period. Teat dipping has little impact on exposure to environmental pathogens in the dairy herd.

Skin flora opportunists are a large group of staphylococcal species other than *S. aureus* (6). They are normal inhabitants of bovine skin and hair. These staphylococcal species are frequently referred to as minor pathogens as they cause only modest increases in somatic cell count and infrequently cause clinical mastitis by comparison to the other major contagious and environmental pathogens. However, in herds practicing teat dipping and dry cow therapy they are generally the most prevalent cause of intramammary infection. Ten to 20% quarters infected is common. Their relative importance in dairy herds is increasing as they do influence bulk tank somatic cell counts and in many well managed dairy herds these staphylococcal species infections may represent a barrier to receipt of bonus payments for low somatic cell count milk. Control of the staphylococcal species by teat dipping and dry cow therapy is modest at best.

An effective mastitis control measure will either decrease the exposure of teat ends to potential pathogens or enhance cow resistance to intramammary infection (2,3,4). While contagious mastitis can be controlled with little or no attention to cow resistance, such may not be the case with regard to environmental mastitis. Research data suggests that both teat end exposure and cow resistance are

significant factors determining the total impact of environmental mastitis in a dairy herd (7). The importance of cow resistance may be magnified by our inability to remove the environmental pathogens from the environment of dairy cows.

Environmental Pathogens

The environmental pathogens are comprised of two large heterogeneous groups of bacteria and these are streptococcal species other than *Str. agalactiae* and the gram-negative bacteria, primarily the coliform bacteria (1). These pathogens are associated with fecal material, bedding materials, feed stuffs, dirt, mud, dust, and water. They are present throughout the environment of all dairy herds. They can not be eliminated from dairy herds and a uniform program for control, such as teat dipping and dry cow therapy for the contagious pathogens, does not exist.

Herd problems with environmental mastitis differ significantly from problems associated with contagious mastitis (7). The prevalence of quarters infected at a point in time with environmental pathogens seldom exceeeds 10% to 15%. The prevalence of quarters infected with a particular pathogen is a function of both the rate of new infection and the duration of those infections (2). A major factor in the low prevalence of quarters infected with the environmental pathogens is the comparatively short duration of these infections (7). Infections of less than 20 days duration are very common regardless of antibiotic therapy and a majority of *Escherichia coli* infections are less than 10 days duration. The low prevalence of infection results in minimal effects on bulk tank milk somatic cell counts and is in contrast to contagious mastitis problems. The short duration of infection also reduces the reliability of individual cow somatic cell counts to detect infected cows and this is particularly so when cell counts are obtained at 30 day intervals.

We recently studied the incidence and cause of mastitis in well managed Ohio dairy herds (8). Herds chosen had a long history of post-milking teat dipping and dry cow therapy of all cows. All herds were total confinement with free stall housing. Herds were studied for a one year period. Prevalence of infection was assessed by culturing milk samples from all four quarters of all cows. Samples were obtained 0-7 days post-calving and during the week prior to drying-off. All herds had eliminated *Str. agalactiae* and prevalence of *S. aureus* infected quarters less than 1% (Table 2). Bulk tank somatic cell counts were determined weekly for one year and the geometric mean for all herds and weeks was 265,000 cells/ml. These data indicate that subclinical mastitis was not a significant problem in these herds.

Table 2. Percent of quarters with intramammary infection at calving and drying off in nine well managed dairy herds.

Bacteriological Status	Calving (n^1 = 4337) $\bar{x}^2 \pm$ Se	Drying Off (n = 4337) $\bar{x} \pm$ Se
Coliform	2.7[a] ± .3	1.0[b] ± .2
Environment Streptococci	2.4 ± .6	2.8 ± .7
NLF[3]	.6 ± .3	.3 ± .2
Staphylococcus species	11.9 ± 1.6	13.5 ± 1.9
Staphylococcus aureus	.3 ± .2	.6 ± .3
Corynebacterium bovis	.6[a] ± .3	8.8[b] ± 4.1
Streptococcus agalactiae	-0- ± -0-	-0- ± -0-
Other microbes	.2 ± .1	.2 ± .1
Bacteriologically-negative	79.7[a] ± 2.2	72.1[b] ± 3.7

[a,b]means within bacterilogical statuses with differing superscripts differ (P<.05).
[1]n = total number of quarters sampled.
[2]$\bar{x} \pm$ Se = mean ± standard error of nine farms.
[3]non-lactose fermenting, gram-negative bacilli.

A major difference between the environmental and contagious pathogens is that a very high percentage of environmental infections will result in clinical mastitis during their short existence. Eighty to 90% of coliform infections will result in clinical mastitis and we find that approximately 10% will be severe and require extensive therapy (7). Approximately 50% of environmental streptococcal infections present in lactation will be clinical in that lactation.

Incidence of clinical mastitis cases is currently the best way to determine the importance of environmental mastitis in low somatic cell count herds. The major mastitis problem in the nine herds described above was clinical mastitis (Table 3) and the overall incidence was approximately one new clinical case per two cow lactations. The major cause of clinical cases was the environmental pathogens.

Table 3. Bacteriological cause and rate of clinical mastitis cases in nine well managed dairy herds.

Bacteriological Status	Clinical cases per 100 cows per 305 days $\bar{x} \pm$ Se
Coliforms	13.6 ± 2.1
Environmental Streptococci	11.7 ± 2.9
Bacteriologically-negative	12.6 ± 2.0
Other pathogens	9.1 ± 3.5
Total	45.7 ± 6.3

Environmental Mastitis and the Dry Period

The dry period contributes significantly to the epidemiology of environmental mastitis in dairy herds

(9,10,11). Intensive studies in a research herd showed that 50% of coliform infections and 40% of environmental streptococcal infections resulting in clinical mastitis during the first one fourth of lactation were the result of new infections occurring in the dry period (9). Studies in nine commercial dairy herds (Table 3) revealed that 20% of all clinical cases were first detected in the first 7 days of lactation and strongly suggested a relationship between these infections and the dry period.

Rates of new infection on a per cow per day basis are higher during the dry period than during lactation (7,9). Rate of new infection is not constant across the dry period but appears to be elevated sigificantly during the two week period following drying-off and the two week period prior to calving. Dry cow therapy with products approved for use in dairy cows appears to be of little value for control of coliform infections but do significantly reduce the rate of new environmental streptococcal infection during the first two weeks of the dry period (9,12). Current dry cow formulations would appear to have two problems with regard to environmental mastitis control during the dry period. First, there is a widespread resistance to the antibiotics used and this is particularly true for the gram-negative organisms. Second, the antibiotics do not persist to the calving period when rate of new infection is again elevated.

The majority of environmental pathogen infections present at drying-off are eliminated during the dry period. Following dry cow therapy, 90% of the environmental streptococcal infections and 71% of the coliform infections present at drying-off were not present at calving (12). However, the percent quarters infected with environmental pathogens at drying-off is generally very low and the true value of dry cow therapy for control of environmental mastitis, particularly the environmental streptococci, is prevention of new infection and not elimination of existing infections. Total dry cow therapy is required to achieve the effect on prevention

Susceptibility of the mammary gland to coliform infection during the dry period is variable among coliform genera (9). *Escherichia coli* infections present at calving would appear to be the result of new infection occurring during the immediate prepartum period. New *E. coli* infections first detected in the early or mid portion of the dry period and persisting to calving are rare. *Klebsiella pneumoniae,* on the other hand, appears to establish infections equally well during the period of active involution and the immediate prepartum period. On a practical basis this suggests that the detection of *E. coli* infections at calving warrants an investigation of calving area sanitation.

Teat End Exposure To Environmental Pathogens

Most research workers agree that exposure to environmental pathogens is greater for housed cows than cows on pasture and that the incidence of environmental mastitis is elevated in housed cows (4,13,14,15). Bedding materials, particularly in free stall housing, can be a significant source of exposure to environmental pathogens.

The numbers of gram-negative and streptococcal bacteria present in bedding materials were monitored for a one year period in nine commercial dairy farms (8). All were total confinement management systems utilizing free stall housing. Bedding materials used were sawdust, chopped straw, sand or crushed limestone. Total gram-negative bacterial, coliform, *Klebsiella* species and streptococcal numbers were significantly higher in the organic materials compared to the inorganic materials (Table 4). Coliform numbers did not differ between sawdust and chopped straw, but *Klebsiella* species were significantly higher in sawdust than chopped straw. Streptococcal numbers were higher in chopped straw than in sawdust.

Table 4. Independent comparisons among mean seasonal bacterial counts[1] in materials used to bed lactating cows.

	Independent Comparisons					
Bacterial counts	Organic[2] (n = 25)	In-organic[3] (n = 11)	Sawdust (n = 13)	Chopped Straw (n = 12)	Sand (n = 7)	Crushed Limestone (n = 4)
Gram-negative	$\bar{x}$ 7.1[a]	6.4[b]	7.0	7.1	6.3	6.5
	SE .0	.1	.1	.1	.1	.1
Coliform	$\bar{x}$ 6.2[a]	5.7[b]	6.2	6.3	5.7	5.8
	SE .1	.1	.2	.1	.2	.1
Klebsiella species	$\bar{x}$ 4.3[a]	3.4[b]	4.8[a]	3.7[b]	3.2	3.8
	SE .1	.2	.2	.2	.2	.2
Streptococcal	$\bar{x}$ 7.5[a]	6.8[b]	7.1[a]	7.8[b]	7.0	6.6
	SE .1	.1	.1	.1	.1	.1

[1]counts are expressed as colony forming units (log_{10})/g dry weight.
[2]organic bedding material were sawdust and chopped straw.
[3]inorganic bedding material were sand and crushed limestone.
[a,b]means within comparisons with differing superscripts differ ($P<.05$)

Coliform numbers in bedding material were influenced by season of the year and were significantly higher in summer and fall compared to winter and spring (Table 5). Interestingly, season of the year had no influence on streptococcal numbers. The total rates of clinical mastitis occurring during lactation were significantly correlated with total gram-negative bacterial and *Klebsiella* species numbers in lactating cow bedding materials.

These studies clearly suggest that the use of inorganic bedding materials such as sand or crushed limestone can reduce teat end exposure to the environmental pathogens by 10 to 100 fold and significantly reduce the risk of environmental mastitis. Where management systems prohibit the use of inorganic materials, efforts should be made to keep bedding as clean and dry as possible. Total daily removal and replacement of sawdust bedding in the back one third of stalls has been shown to reduce the exposure to the environmental pathogens and the

incidence of environmental mastitis (11).

Table 5. Bacterial counts (log_{10})/g in lactating cow bedding by seasons of the year in nine dairy herds.

Bacterial counts		Summer (n = 9)	Fall (n = 9)	Winter (n = 9)	Spring (n = 9)
Gram-negative	$\bar{x}$[a]	7.32[c]	7.06[c,d]	6.61[d]	6.66[d]
	SE[b]	.15	.17	.15	.16
Coliform	$\bar{x}$	6.61[c]	6.20[c,d]	5.72[d]	5.90[d]
	SE	.13	.19	.16	.18
Klebsiella species	$\bar{x}$	4.62[c]	4.48[c]	3.58[d]	3.54[d]
	SE	.35	.30	.22	.21
Streptococcal	$\bar{x}$	7.27	7.35	7.44	7.25
	SE	.18	.22	.18	.23

[a]mean of nine farms.
[b]standard error.
[c,d]means with differing superscripts within same row differ ($P<.05$).

A relatively new concept for control of environmental mastitis is the technique of predipping (16,17). Dipping teats in a germicide prior to machine attachment reduces exposure of teat ends to the environmental pathogens during the milking process. Published data show that predipping can reduce the rate of new environmental pathogen infection during lactation by 50%. Predipping does not influence rate of new infection during the dry period. The control achieved by predipping may be greater in herds where machine function is poor or in herds where cow's teats are heavily contaminated with environmental pathogens.

Another technique used to reduce teat end exposure to environmental pathogens is the use of barrier teat dips (18,19). Barrier dips are reported to reduce new coliform intramammary infections. However, their efficacy against the environmental streptococci and the contagious pathogens appears to be lower than that of germicidal dips (1). The addition of germicidal agents to barrier dips may alleviate this problem, however, definitive data have not been published.

Barrier dips increase the cost of teat dipping as well as increase labor and time to prepare udders for milking. There is no indication that barrier dips effectively control new environmental pathogen infection during the dry period (15).

Resistance to Environmental Mastitis

Control measures discussed above concentrate on reduced exposure of teat ends to environmental pathogenes. However, cow resistance and susceptibility to environmental mastitis would appear to be influenced by a number of factors such as stage of lactation, parity or age, level of production, and nutrition (15). These factors appear to be independent of exposure level. Rate of new infection is higher during the dry period than during lactation. Rate is markedly elevated during the two weeks following drying off and the two weeks prior to calving. Rate of new infection is highest in early lactation and declines progressively throughout lactation. Rate of new infection increases with parity and the increase is seen during both the dry period and lactation. The incidence of acute coliform mastitis would appear to be elevated in higher producing cows, cows in early lactation, and stressed cows.

Dietary deficiencies in vitamin E and selenium have been associated with increased incidence of environmental mastitis (20,21,22,23,24). Cows whose diets were supplemented with vitamin E to achieve a daily intake of 1000 units per cow per day during the dry period and injected with 50 mg selenium 21 days prepartum, had significantly fewer cases of clinical mastitis during lactation when compared with unsupplemented controls (20). These data also point out the importance of supplementation during the dry period (22). Vitamin E and selenium are important for efficient phagocytic cell function and phagocytic cells are known to be a major factor in resistance to intramammary infection (22).

Control of Environmental Mastitis

Clearly, environmental mastitis control in dairy herds will require attention to both the level of exposure to the environmental pathogens and maintenance of optimum resistance of the cows. Unfortunately our knowledge of management factors which effectively reduce teat end exposure to pathogens is far greater than our knowledge of effective methods to enhance the resistance of dairy cows to mastitis. Mastitis control beyond teat dipping and dry cow therapy must focus on the environmental pathogens. However, there is currently no single method of control that can be uniformly recommended for all dairy herds.

We are convinced that control of environmental mastitis will be complicated by the fact that the environmental pathogens can not be eliminated from the dairy herd environment. Our recommendations for environmental mastitis control are: 1) know the factors in the herd that can lead to increased exposure and take steps to reduce them; 2) know which cows are most susceptible to new infection and manage these cows to minimize exposure; 3) keep the environment as clean and *dry* as possible; 4) milk cows with clean *dry* teats and udders; and 5) feed cows well balanced diets and make sure the diet is vitamin E and selenium adequate. Finally, do not forget that the environment and nutrition of the dry cow is as important as that of the lactating herd for effective environmental mastitis control.

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