Making Further Progress in Low Somatic Cell Count Herds

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Somatic cell counts (SCC) serve as valuable indicators of prevalence of intramammary infection (IMI). A strong positive correlation between bulk tank SCC and prevalence of infection has been well established.[1 2] However, it is becoming clear that some herds with low SCC and good control of subclinical infection may still have a problem of frequent cases of clinical mastitis.

Streptococcus agalactiae and *Staphylococcus aureus* are the organisms most often associated with elevated SCC. They are believed to be contagious, and the infections they cause are often subclinical in nature. The use of post-milking teat dipping and dry cow therapy to control *Strep. agalactiae* and *Staph. aureus.* is well established.[3 4 5] Most herds with low SCC use accepted mastitis control measures, and consequently have a low prevalence of subclinical mastitis. For example, in a recent study of 16 Pennsylvania dairy herds with a DHIA SCC below 150,000 cells/ml, 14 of the 16 herds were free of *Strep. agalactiae.* The remaining two herds had only one infected quarter. Similarly, 9 of the 16 herds were free of *Staph. aureus,* the remainder having very low prevalences of infection of this type. Other studies have reported that control of *Strep. agalactiae* and *Staph. aureus* is associated with low SCC.[2 3 6]

However, herds with low prevalences of subclinical infection may yet have a mastitis problem, particularly that caused by organisms of environmental origin. These would include primarily streptococci other than agalactiae (non-ag. strep.), and the lactose fermenting organisms of the family Enterobacteriaceae, loosely termed coliforms. This paper reviews the problems facing dairy farmers that have been able to maintain their herd SCC, and thus subclinical mastitis, at a low level. Means to assess the mastitis problem in such herds and mastitis control methods thought to be effective is discussed.

Types of Intramammary Infection Present in the Low Cell Count Dairy Herd

Of the environmental pathogens the coliform group stimulates the most interest due to the possible clinical severity of the infection. However, numerous reports have indicated that only a minority of coliform infections are severe.[6] Nonetheless, Anderson, et al.[7] reported that coliforms were the single most important cause of acute mastitis, accounting for 35% of the total cases in a large dairy herd. Futhermore, the prognosis is more guarded for coliform infections than for other types of infections. Six of 42 acute coliform cases resulted in death of the cow, as opposed to no deaths among 59 acute cases caused by gram-positive bacteria. Bushnell[8] reported that only 10% of coliform cases treated in a large herd were classified as peracute. However, of the cows with the peracute form, 10% died and 70% were culled for agalactia. Thus, while only a small proportion of coliform infections are of the severe form, they are more likely to be so than any other type of infection. In addition, severe coliform mastitis may result in a total cessation of lactation, or in death.

Eberhart and Buckalew[9] reported clinical mastitis remained a serious problem in a dairy herd, despite mastitis control measures that had controlled *Strep. agalactiae* and *Staph. aureus.* The clinical cases were primarily due to streptococci other than agalactiae and coliforms. Other reports have also indicated that subclinical mastitis control measures are ineffective in the prevention of infections caused by environmental bacteria.[6 10 11]

The herd survey, the traditional method of determining the types and prevalence of infection in a herd, is effective in characterizing chronic (and often subclinical) infections. However, it may give an erroneous impression of the occurrence of the more acute infections caused by environmental bacteria. National Mastitis Council (N.M.C.) standard culture technique calls for .01 ml of milk streaked on one-quarter of a blood agar plate. Thus, the minimum number of colony-forming units (CFU)/ml needed to permit detection of an infection would be about 100. Smith[10] presented data giving the average CFU/ml isolated from 139 samples of 21 known coliform infected quarters. The geometric mean was 82.7 CFU/ml, with 53.2% of the samples containing less than 99 CFU/ml. Based upon this and other reports,[12 13] Smith suggested that the N.M.C. guidelines are inadequate for reliable detection of coliform infections. In addition, small numbers of coliform bacteria in a milk sample should be considered as possible significant and not necessarily as contaminants.

It is believed the duration of coliform infections is shorter than that of the typical subclinical organisms. Smith[10] stated that only 24% of 144 coliform infections exceeded 30 days in duration. Thus sampling of a herd on a one-time, or regular interval basis, is very likely to underestimate the occurrence of coliform infection.

Numerous publications have reported low prevalence of infection with coliform organisms upon whole herd culture,

and a concurrent high incidence of clinical mastitis.[9 10 14] Eberhart and Buckalew,[9] and Smith[10] both reported prevalences of 1% to 2% of quarters on herd culture. Simultaneously, clinical incidence was reported as .88 and 2.26 cases/cow-year respectively. Approximately 34% of the clinical cases in both herds were caused by coliform bacteria. Thus, there were 30 and 77 cases of clinical coliform mastitis per 100 milking head per year in these herds.

In our recently completed study of 16 very low somatic cell count herds, average herd prevalence of IMI with gram-negative rods was 0.7%. Twelve of the 16 trial herds agreed to collect samples from all clinical cases for 6 months. Four herds have completed the trial. The frequency of all clinical mastitis collected from each herd was .06, .27, .31, and .71 cases/cow-year. On average, coliform organisms were isolated from 50% of the cultures. To date two points are apparent from this trial. The first is the existence of a wide range of variation in incidence of clinical mastitis among low cell count herds. It would seem some herds are able to maintain very low rates of clinical mastitis. The second point is the high proportion of clinical cases in these herds caused by coliforms.

A summary of the bacteriological studies in these low SCC herds include the following highlights. These herds have *Strep. agalactiae* and *Staph. aureus* well under control. They are likely to have a low prevalence of IMI with coliforms detected by herd surveys, but a high incidence of clinical coliform mastitis is possible. This disparity between routine culture and clinical mastitis may in part be due to the short duration and low numbers of bacteria in milk from some coliform infected quarters. A wide range of clinical incidence (in terms of cases/cow-year) is apparent, and further study to identify herd factors associated with low clinical rates is needed. An attempt to describe mastitis in a low cell count herd should include incidence of clinical mastitis and identification of causative organisms.

Record Keeping in the low SCC Herd

To monitor mastitis in low SCC herds it is necessary to keep accurate records of the occurrence of clinical mastitis and the types of organisms causing these cases. Such a program requires a willingness on the part of the farmer to collect milk samples from all clinical cases before treatment, and on the part of the veterinarian to culture these samples and identify the organisms isolated. Antimicrobial sensitivity patterns of bacteria isolated are valuable both for definition of the problem and in determing treatment regimens. Samples collected by the farmer may be frozen until they are transported to the laboratory. A sampling period of 6-12 months should provide a representative number of cases, although the summer months should be included.

The frequency of clinical mastitis is easily calculated. Here at the Pennsylvania State Mastitis Diagnostic Laboratory (PMDL) the number of clinical cases observed per milking cow-per year is often used. Thus 20 clinical cases observed over a period of one year in a 50 cow milking herd would equate to 20 cases ÷ (50 cows x 1 yr.) = .4 cases/cow-year. Similarly, 20 cases in a 100 milking cow herd observed over 6 months would be 20 cases ÷ (100 cows x .5 yr.) = .4 cases/cow-year. An acceptable rate of clinical mastitis has not been established for low cell count herds, although some researchers have suggested 2-3% of the milking cows/month.

The use of bacterial culture and antibiotic sensitivity testing in clinical mastitis cases has become more popular among veterinarians. While not infallible, identification and sensitivity results enhance the chances for success of antimicrobial therapy. Culture of clinical cases from low cell count herds will usually reveal that most are caused by coliforms or streptococci other than *Strep. agalactiae.* However, a high incidence of clinical mastitis may also be caused by such organisms as Mycoplasma, Prototheca, Pseudomonas, and yeasts; unless diagnosed promptly these organisms may cause serious herd problems. *Pseudomonas aeruginosa* is a case in point.

Pseudomonas can cause outbreaks of increased incidence of clinical mastitis. Though sometimes acute, most Pseudomonas infections are chronic and often take a subacute clinical form at frequent intervals. Pseudomonas infections are generally refractory to therapy. Contaminated wash lines in milking parlors have been reported,[15] and encountered by us at the PMDL, as a source of infection by this organism. We have recently encountered three herds with low SCC and high frequency of clinical mastitis. On whole herd culture a high prevalence of IMI with Pseudomonas was found. In each herd hoses used to wash udders before milking were heavily contaminated with Pseudomonas. Two of the herds were originally diagnosed as having a coliform mastitis problem by the attending veterinarian. The third was not cultured regularly. Changes in water line sanitation needed to control this organism would not have been made without proper bacterial identification.

The stage of lactation and season of the year with highest incidences of clinical cases should also be recorded. The information could be used subsequently in developing a herd mastitis program.

Preventive Measures

Mastitis, particularly that of an environmental origin, is a multifactorial disease. Type and virulence of the organism, degree of exposure, immune status of the cow, and presence of stress factors all play a role. A review of current concepts of the pathogenesis of coliform mastitis is beneficial in evaluating programs for its control.

Clinical mastitis, particularly that of an acute nature, has been long association with the postpartum period. Eberhart and Buckalew[9] reported that the incidence of clinical mastitis due to environmental organisms was highest in the

first month of lactation. New intra-mammary infections occurred with the highest frequency during the dry and immediate postpartum period. The subsequent first month of lactation had the highest rate of new infection of any month of the lactation.

Bramley[16] observed this trend also, suggesting an association between decreasing levels of milk lactoferrin and increased incidence of infection. Decreased concentration of this bacteriostatic protein during the postpartum period would enhance infection of the udder by an invading pathogen. Hill[12] et al., observed a much more severe form of mastitis in newly calved cows as compared to mid-lactation cows experimentally infected with *E. coli.* A delay in the migration of phagocytes into the gland was observed that may have contributed to the rapid multiplication of bacteria in the milk of the postpartum cows.[12] Other factors, such as extreme heat and humidity and heavy lactation may contribute to the stress experience by a cow.

Coliform mastitis is unlike either *Strep. agalactiae* or *Staph. aureus* in that it is not highly contagious and is probably contracted from the environment. This feature may explain the failure of post milking teat dipping to control coliform infections. The major concern of coliform control must focus upon reduction of environmental exposure during the period between milkings.

Bramley incriminated sawdust bedding as a factor in increased incidence of coliform mastitis.[16] Although considerable variation was observed from sample to sample, bacterial analysis consistently found higher levels of coliform organisms in sawdust bedding as compared to sand. Cows maintained on sawdust bedding had an incidence of clinical coliform mastitis 4.5 times higher than herdmates bedded on sand. In addition, both Bramley[16] and Eberhart[9] have cited reports demonstrating multiplication of *Klebsiella* organisms in sawdust. Bacterial counts were found to be higher in used sawdust than in either unused sawdust or fresh cattle feces. Further work by Bramley suggested that coliforms may be kept at low numbers if bedding is regularly changed.[16] While sawdust bedding may play a role in the epidemiology of Klebsiella mastitis, other bedding materials can harbor potential pathogens, particularly if fecal contamination is uncontrolled.

Reduction of potential exposure to coliforms might also include proper washing and drying of the udder at milking time. Bramley[16] reported increased infection rates with *E. coli* and *Strep. uberis* in cows improperly washed and dried as compared to unwashed controls. Work in California has suggested that predipping cows, prior to milking, with a germicidal teat dip may help reduce coliform infections. However, results of controlled studies have not been published to date. Studies recently completed here in the Department of Veterinary Science revealed no significant difference in new infection rate between pre-dipped and non-dipped controls.[17]

Work has been attempted to achieve protection during the inter-milking period with barrier film teat dips. The results have not been highly successful or consistent. A significant reduction in new infection rate with *E. coli* was not observed in dipped versus undipped controls.[18] Similarly, the use of a latex test dip with germicide once daily for approximately 14 days prior to parturition did not reduce new infections at parturition.[19] However, Farnsworth[20] *et al.,* reported a significant reduction in the rate of new coliform infections among lactating cows. While these products are promising in theory, further development is needed.

A sound nutritional program, with special emphasis on the dry cow ration, is essential for any herd plan. Cows that do not have to simultaneously deal with such problems as milk fever, ketosis, retained placentas/metritis, displaced abomasums, etc. are more likely to resist a challenge of intra-mammary infection than their poorly managed counterparts. Recent work by Smith suggested an important role of Vitamin E and selenium in the immune process of the udder.[21] Cows supplemented with vitamin E and selenium during the dry period were found to have a lower incidence of clinical mastitis, and a shorter duration of clinical symptoms, than unsupplemented controls. This has special significance in the northeast portion of this country where soil selenium levels are notoriously low.

Special attention should be given to the environment of calving cows. Maternity pens should be cleaned regularly, sawdust bedding avoided if possible, and ventilation, particularly during the hot summer months, should be inducive to cow comfort. Similarly, measures should be taken to reduce the heat stress of lactating cows. Cows in the recently fresh/high production groups especially should be kept as comfortable as possible. Obvious sources of contamination such as mudholes around feeding bunks or in pasture should be eliminated if possible.

In addition, during periods of increased clinical outbreaks, water sources, especially those used for udder preparation, should be analyzed. This is not often a source of infection with coliform organisms, but, unusual organisms such as *Pseudomonas* and *Prototheca* can occasionally be be found. Also contamination of a teat dip by gram-negative bacteria, though rare, has been encountered.

Some attempts at immunization against coliform mastitis (including autogenous vaccines from "on-farm" strains) have been made. To date no controlled study has been published demonstrating a benefit from such products.

Finally, ongoing trials have been evaluating the use of intramammary devices (IMD) as a means of stimulating the mammary immune response.[22] It is hoped these devices, once implanted, will stimulate a moderate increase in SCC. An increase in the SCC is thought to enhance the immune resistance capability of low SCC cows to overcome an infection challenge. Paape has reported a rise in mean SCC in implanted versus non-implanted quarters.[22] Preliminary data have demonstrated a lower infection rate among quarters challenged with *Strep. uberis* and *E. coli* in implanted versus non-implanted controls. The results are promising, however, long-term effects of IMDs on

production and udder health are as yet unknown. Data from one lactation suggested a small statistically nonsignificant, decrease in milk production.[22] In addition, increased herd SCC due to these devices may cause some herds to lose a bonus currently received from their milk plant for low SCC milk.

Summary

Although low cell count herds have controlled sub-clinical mastitis, a problem may still exist due to environmental organisms. The existence of a problem is best monitored by extensive culturing and good record keeping of clinical cases. Not all low SCC herds necessarily have a problem, as there is a wide variation in the rate of clinical cases among herds.

Although there are numerous potential measures such as IMD's, vaccines and barrier dips available, none of these has been proven effective. Further study in this area is required. For the time being control of coliform mastitis will essentially be one of reducing environmental exposure.

With adequate mastitis control measures available to control *Strep. agalactiae* and *Staph. aureus,* future mastitis research should necessarily be directed to control of environmental organisms.

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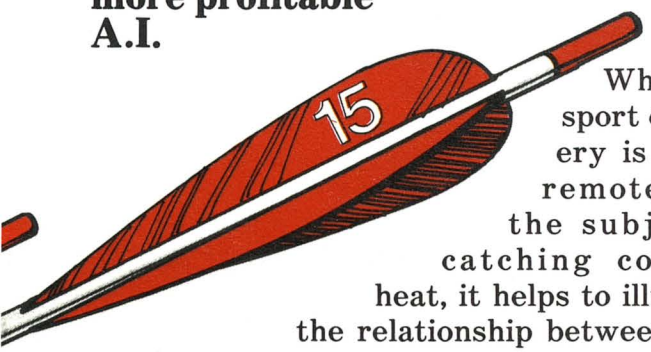

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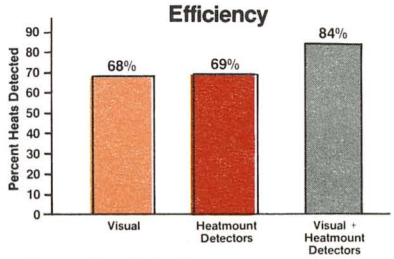

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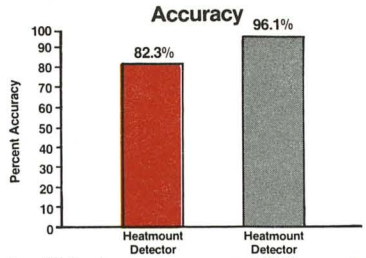

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