

Controlling Environmental Mastitis: Focus on the Dry Period

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Many Western dairy herds have successfully controlled contagious mastitis pathogens (*Staphylococcus aureus*, *Streptococcus agalactiae* and *Mycoplasma* spp) through bulk tank monitoring, excellent parlor procedures, and dry cow antibiotic treatment. Control of contagious pathogens typically reduces bulk tank somatic cell count (SCC) and presumably also decreases the incidence of clinical mastitis. Why then do we see herds with contagious mastitis under control and bulk tank SCCs greater than 200,000 cells/ml but just as much clinical mastitis?

If the bulk tank is clear of contagious pathogens, then in most cases the clinical mastitis is due to increased number of infections with environmental mastitis pathogens which include environmental streptococci (like *Strep. Uberis*) and the coliform bacteria (like *E. coli* and *Klebsiella* spp.). A somatic cell count of 250-500,000 cells/ml in an individual cow is protective against the environmental pathogens because the bacteria are eliminated by these white blood cells before they have the opportunity to cause clinical mastitis. When contagious pathogens which typically cause long term subclinical infections are eliminated, the SCC drops and the individual cow becomes more susceptible to environmental pathogens. We may see just as much, if not more, clinical mastitis. Reducing exposure of the teat ends of cows to these environmental pathogens is the key to reducing clinical mastitis in the herd.

When cows with clinical mastitis from herds that have controlled contagious mastitis pathogens are routinely milk cultured, 80% of the samples are positive for environmental pathogens or report “no growth”. The “no growth” results typically represent 15-30% of samples and are thought to be mild coliform infections with low bacterial numbers at the time of sampling. To prevent or control environmental pathogen intramammary infection (IMI), exposure of the teat end to these pathogens must be minimized. Most dairy producers and workers are familiar with management recommendations that minimize teat end exposure. These include application of pre-milking teat dip, feeding cows after milking to allow time for teat end closure before they lie down, and maintaining a clean, dry environment. Although these recommendations seem simple enough, they are not always easily followed in modern, intensive dairy housing. Furthermore, producers tend to focus on the lactating cows, even when recent studies show that 4.5–5.5 times more environmental pathogen IMI occurs during the dry period than during lactation. A recent study using DNA fingerprinting determined that 52% of clinical coliform mastitis detected during the first 100 days of lactation occurred in quarters initially infected **with the same strain of bacteria** during the dry period! Another study determined that 50.5% of new environmental streptococci IMI occurred during the dry period. The highest risk of IMI during the dry period is the first three weeks after dry off and the last two weeks before calving. Just like nutritional management of the dry cow, it has become clear that udder health management of the dry cow is critical to lactational performance.

Protocol for Evaluating Dry Period Udder Health Management

1. Evaluate the distribution of clinical mastitis by days in milk (DIM). This can be done easily in DC305 by entering EGRAPH in the command line, choosing the mastitis event and the DIM option. Herds that benefit from improved management in the dry period often find the greatest number of mastitis cases during the 0-10 DIM period.

2. Evaluate the individual cow SCC data. For greatest accuracy you must be testing SCC of the herd monthly.

3. *Heifer new infection rate (HNIR)* is the % first lactation heifers with a first test SCC greater than 200,000. The goal is 10% or less. If the HNIR in your herd is 15% or greater, you should evaluate springing heifer management. Routine culturing of heifers is an excellent way to identify *Staphylococcus aureus* infection which is best treated at this time. If a high percentage of heifers are freshening with environmental streptococci or coliform infection, heifer housing and bedding should be critically evaluated

4. *Dry cow new infection rate (DCNIR)* is the % dry cows with the last test of the previous lactation less than 200,000 and the first test of the next lactation greater than 200,000. The goal is 10% or less. If the DCNIR in your herd is 15% or greater, you should evaluate dry cow environmental management, specifically housing and bedding.

5. *Dry cow cure rate (DCCR)* is the % cows with the last test of the previous lactation greater than 200,000 and the first test of the next lactation less than 200,000. The goal is 80% or greater. If the DCCR in your herd is 70% or less, you should evaluate your dry cow treatment protocol. Often times a lower than desired DCCR suggests bacterial resistance may be developing and a different antibiotic should be used. Culturing a number of cows with high last test and first test SCC and antibiotic sensitivity testing of Gram positive bacteria (ie Staphylococci and Streptococci) will indicate if a change of dry cow treatment is needed. If you find that a large proportion of cultured cows have coliform infection, dry cow treatment will not be curative. Reducing exposure and enhancing resistance is the key to prevent infection in the first place.

Strategies To Reduce Dry Period IMI

Reduce exposure of the teat end to environmental bacteria. Bacterial populations on the teat skin of cows closely reflects those found in the bedding materials on which the cows are housed. Bacteria require moisture, organic nutrients and an appropriate temperature to grow. The ideal bedding is dry, stays dry, and contains very little organic matter. While bedding material choices are most often considered for the lactating cattle, little consideration is typically given to dry cow housing and bedding. Since significantly more IMI occur during the dry period, bedding decisions made for lactating cattle with the goal of reducing teat end bacterial exposure should be applied to the dry cows as well. If your lactating cows are on sand in free-stalls, your dry cows should be too! Many producers consider pasture to be a “clean” environment in which bacterial exposure to teat ends is minimal, but high traffic areas (eg under the shade of a tree) often result in cow density as high or higher than in confinement housing and can have bacterial numbers that exceed those of confinement housing. Total bacterial numbers greater than one million per gram of bedding have been associated with an increased risk of clinical mastitis during lactation and likely relate to increased risk of IMI during the dry period. The Milk Quality Lab at the University of Minnesota has considerable experience with bacterial culture of bedding material and can assist you to determine the bacterial load in the bedding of your lactating and dry cow pens.

Keep cows clean. A cow cleanliness scoring system has been developed assessing cleanliness of the cow’s tail head, flank, belly, udder and lower rear legs. Studies using this system conclude that hygiene scores of udder and lower rear legs had a significant effect on somatic cell counts. Cleaner cows reflect reduced teat end bacterial exposure and reduced IMI resulting in lower SCC. Dry cows with dirty udders, lower rear legs and bellies are likely at greater risk of IMI during the dry period expressed as clinical mastitis in early lactation.

Complete closure of the teat end and keratin formation at the end of lactation is critical to prevent new IMI during the early dry period. A study of teat end closure following abrupt dry off, performed in 300 cows / 1178 quarters, indicated teat ends had not closed in at least 40% of quarters within the first week of the dry period. By 6 weeks 23.4% were still classified as open. The level of milk production the day before drying off significantly influenced time until closure. A greater percentage of cows producing more than 46 lbs experienced delayed teat end closure compared with those producing less than 46 lbs. Cows in which teat end closure was delayed were almost twice as likely to develop a new IMI during the dry period. Gradual dry off of higher producing cows may help reduce delayed teat end closure.

Barrier teat dips or external teat sealants may help reduce exposure by placing a protective film over the teat end. These products typically do not provide protection much beyond 10 days post application as they peel away and fall off. It is important that teats are clean and dry before application to achieve maximal adherence.

Pfizer has recently introduced an internal teat sealant (Orbeseal®) that is infused through the teat orifice at dry off and persists until calving when it is stripped out of the teat end. The product has no antibacterial action, but acts as a physical barrier to prevent bacterial penetration of the teat orifice. In the UK and New Zealand the product is used instead of dry cow antibiotics in cows unlikely to have an IMI at dry off (SCC <200,000 cells/ml and no clinical mastitis during the lactation). In a study comparing dry cow antibiotic treatment versus use of Orbeseal® alone, there were fewer coliform IMI, but no difference in other major pathogens or clinical mastitis in the subsequent lactation. This study suggests that in cows without IMI at dry off, Orbeseal® alone is equally effective as dry cow antibiotics in preventing new infections with environmental streptococci and may provide better protection against coliforms during the dry period. In the US the product is labeled for use in conjunction with dry cow antibiotic therapy. A study comparing the use of Orbeseal® and dry cow antibiotic versus dry cow antibiotic alone found quarters treated with Orbeseal® were 27% less likely to develop a new IMI between dry off and 1-3 DIM and 30% less likely to have an IMI present at 1-3 DIM. Differences in clinical mastitis during early lactation have not been reported yet.

Dry cow antibiotic therapy will eliminate many environmental streptococci infections present at dry off and reduce the number acquired in the dry period. While dry cow treatment does help reduce environmental streptococci infections during the early dry period, it is not as effective during the late dry period in preventing new infections. The dry cow treatments available in the US are not effective against Gram negative (coliform) bacteria. A study in England showed a significant reduction in clinical coliform mastitis in cows treated with a dry cow antibiotic with activity against coliforms versus cows treated with a product having no activity against coliforms. This product is not available in the US currently. Future development of dry cow treatments effective against Gram negative bacteria may help reduce clinical coliform mastitis in the subsequent lactation. The cornerstone of control will remain reducing exposure of teat ends to Gram negative bacterial pathogens.