# Recent Findings About Mastitis

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NEWARK, Del.—Mastitis is gone.

How we wish this was so!

As a boy milking cows by hand, I learned about mastitis. People often said then that milking machines caused mastitis. Yet even though we milked by hand, our cows had mastitis, too.

When I went to a school for professional milkers, I learned that mastitits is the reaction to many different causes, including bacteria, yeasts, injuries, heat stress and hormonal effects.

I also learned how to operate milking machines properly so that they do not contribute to mastitis.

This was still in the times before antibiotics, when in addition to sulfa drugs, frequent milking-out was usually the primary treatment. Even in today's mastitis control, frequent milking-out is still very effective.

Then antibiotics—the panacea for most anything—arrived on the scene, and vaccinations were used effectively against mastitis.

It soon became apparent that identifying a specific cause from the multiple of mastitis-causing possibilities was required. Culturing of samples was necessary in order to supply specific treatments rather than a "shotgun" approach.

Today, we are much more interested in prevention than in treatments, not only because of the expense, but because of the level of resignation and frustration with the many causes of mastitis.

This situation is compounded by the variety of only partially effective treatments and treatments that are effective for only a limited time.

This is still the case although we have the most sophisticated milking machines ever, with differential pulsation speeds, quatro milk hoses, automatic take-offs, different size teat lines and automated disinfection between cows. We also have ever-new and varied antibiotics.

Still mastitis costs the U.S. dairy farmer approximately $200/cow/year on average, and it remains the number-one disease on the dairy farm.

We have learned the hard way that for every one case of clinical mastitis with visibly abnormal milk, we have at least four other so-called subclinical cases that do not have visibly abnormal milk, but that exhibit beginning inflammations.

These inflammations, which are being fought by the cow's defense system, can break out into clinical cases at any time and have already reduced normal levels of milk production.

The new powerful tool giving us all this information is the measurement of "somatic cell counts" in milk, an indication of the state of the cow's defense activities.

Not all somatic cell counts are the result of inflammation.

Older cows typically have more than young cows. Milk at the beginning and end of lactation is normally high in somatic cell counts, and so is foremilk and after strippings, in particular.

Also, cows in estrus have higher somatic cell counts in their milk, and summer heat stress will also elevate somatic cell count levels.

Nonetheless, monthly testing of every cow in a herd has become a powerful tool for reducing mastitis incidence. It has even led to reducing some typical pathogenic bacteria as causes of mastitis because they are encountered less frequently.

The recent issue of the newsletter of the National Mastitis Council (February-March 1995) gives some disconcerting information in this direction. Typically found mastitis-causing bacteria are Streptococcus agalactiae and Staphylococcus aureus.

Now it is reported that in "well-managed" herds other so-called coagulase-negative staphylococcus species are the most prevalent causes of mastitis.

They used to be considered "minor" pathogens and were ignored in control programs. Now it is fournd that these bacteria cause "mild" mastitis in 5-15 percent of udder quarters and raise somatic cell counts significantly.

They occur more frequently in first-lactation cows than in older cows, and more in the beginning of lactation. They are neither contagious nor "environmental," but are called "skin opportunists."

Fortunately, these pathogens are controllable with teat dip programs as was found in studies of prepartum heifers in very cold weather when teat dipping was interrupted during the dry period.

Different teat-dip preparations are now available to make prevention more effective. Iodine or chlorine have been the logical choices, but other chemical preparations are also useful.

A new compound, sodium dicholoroisocyanurate, provides hypocholorous acid.

Tested in Louisiana studies on 140 cows, Staphylococcus aureus was reduced by 74 percent and Streptococcus agalactiae by 65 percent without irritating teat ends or milker's hands.

But what about the stubbornly remaining 26 or 35 percent?

This is where we get the next generation of resistance to our treatments. Other preventive measures have to be found.

For some time the idea of disinfecting the teat end and actually sealing it against pathogenic invasions has been pursued by researchers, only to find that the ideal sealant material has not yet been discovered.

Iowa research with sealant antibiotic wax significantly prevented new infections, and even cured existing infections, during the dry period if the sealant stayed in place for several weeks. Industry still needs to meet this challenge with new inventions.

Meanwhile, let's get back to what can be done during the dry period.

There is news from New Zealand—that highly successful dairy land—about their new program called SAMM, under which they treat all dry cows if the herd bulk tank test is above 400,000 somatic cell counts. Below that level, only selective dry-cow treatment is recommended.

I wish that National Mastitis Council Newsletter could reach each dairy farmer regularly. For a small membership fee, much can be learned, especially in the area of dairy farm profits.