

Environmental Mastitis, Part I

By Ruth Zadoks, DVM

Mastitis is an inflammation of the mammary gland or udder tissue. In cows, mastitis is usually caused by bacteria. Mastitis may also be the result of non-infectious causes, such as mechanical damage. A poorly adjusted milking machine, or narrow stalls and poorly trimmed claws, may cause mechanical injuries to the teats and the udder. Both bacterial infections and mechanical damage can result in mastitis. Furthermore, mechanical damage may open the way for bacterial infections.

Bacterial mastitis is characterized in two groups, based on the way the bacteria are spread. On the one hand, there is contagious mastitis. The main source of bacteria in the case of contagious mastitis is the cow. The main route of spread is the milking process, be it via udder cloths, milking machine, or milkers' hands. Hence, the solution for a contagious mastitis problem is to be found in identification and removal of the source (usually treatment, segregation, or culling), and prevention of spread during milking. On the other hand, there is environmental mastitis. As the name implies, the environment is the main source of bacteria in this case. Different environments harbor different types of bacteria. And different

Most cows are healthy, despite permanent exposure to bacteria in their B environment. That is because they have ways to resist the environmental bacterial challenge. Roughly, there are two categories of protection mechanisms: innate and acquired. The innate protection mechanisms are what the animal is born with. It encompasses physical-mechanical mechanisms, and so-called innate immune responses.

The physical-mechanical mechanisms are relatively simple, but they are extremely important. Genetic factors and management play a role in these mechanisms. Udder and teat conformation is one aspect of the physical-mechanical mechanism: a deep, pendulous udder is more exposed to bacteria and more prone to mastitis than a compact udder with a strong suspensory ligament. Udder conformation is partly genetic, meaning that it can be selected for when breeding, partly the results of milk production (and an animal with high production may have poor udder quality, making breeding decisions difficult), partly the result of nutrition.

Calcium-phosphorus imbalance in the ration and subsequent milk fever may weaken the suspensory ligament, resulting in torn ligaments and low-hanging udders. Milk fever may also affect another mechanism: the sphincter muscle that closes the teat canal and teat opening. Milk fever weakens the sphincter, so that the teat canal stays open, and bacteria have free access, espe-

cially when the cow can't stand because of the milk fever and lies down in a dirty calving stall or sick pen. This sphincter may be the main cause of mastitis in some well-fed, high-producing cows too: if a cow is to produce lots and lots of milk, but people are impatient while milking her, milking speed is increased (by genetic selection) and sphincter quality is compromised. Thus, the high producing cow may have poor teat end closure, resulting in increased susceptibility to mastitis.

The skin of the teat is important too: chapped skin can be a place for bacteria to multiply. Callous formation around teat ends may be the result of overmilking or poorly adjusted milking machines and liners. Teats with very rough and calloused ends are more likely to develop mastitis. Within the teat canal, there is a keratin plug that contains naturally occurring bactericidal compounds. When this plug is disrupted or removed, the teat canal is easily accessible for bacteria. Even in healthy cows, it may take weeks for a plug to form and close the teat canal. Another vulnerability of the dry cow is that she doesn't get milked: milking removes a lot of bacteria, and also debris that may have formed as a result of mastitis. It is because of this that dry cows and heifers are more prone to summer mastitis than lactating animals, and that a quarter with summer mastitis needs to be milked to remove the debris that makes the cow sick.

The immunological component of the innate immune system is comprised of cells and soluble molecules. Several types of white blood cells scavenge bacteria, and several soluble molecules in milk, e.g. lactoferrin and lysozyme, slow down the growth of bacteria.

There is an acquired side to the immune system too: immunologic memory or antibodies that develop over time as the animal comes in contact with bacteria (or viruses, etc.). This is the part of the immune system that we try to harness when using vaccines: we try to generate immunologic memory, so that the cow has a high capacity to generate a quick response when specific bacteria invade her udder. For *E. coli* and related gram-negative bacteria, such as *Klebsiella*, this approach is widely used and seems successful in reducing the severity of disease and the damage resulting from mastitis. For other common mastitis pathogens, such as staphylococci, streptococci, and mycoplasma, vaccines are not available yet. Part of the problem is that within the bacterial species, many different strains exist. A vaccine that may work against one strain of the bacteria may not work against other strains of the same species. And even if they cover multiple strains, many vaccines will only provide protection against a limited number of or just one species.

The immune system is not only influenced by breeding and vaccination. Nutrition plays a role too. In particular, vitamins and spore elements such as vitamin E and

selenium are necessary for a good immune function. In addition, the energy content of rations is important. High producing cows that can't meet their energy demand with the energy they take in with their feed, will mobilize energy reserves in their body. To a certain extent, this is normal. But when the imbalance between supply and demand is more than the animal can cope with, ketosis may develop. Ketosis may be accompanied by visible signs, such as production drop, apathy, etc., but there are invisible consequences too: among other things, the immune response is slowed down, and the rate at which white blood cells move to the udder to protect it from mastitis will be decreased. This may give bacteria the opportunity to out compete the immune system, resulting in an increased risk of mastitis in high producing cows. Again, there is a balancing mechanism at work here: not all cows with high productions develop ketosis and mastitis, and not all cows with mediocre productions stay free of ketosis and mastitis. It's the cow's ability to balance demand and supply, partly dependent on the feed she is provided and partly genetically determined, as well as the balance between host resistance and bacterial challenge that determines the outcome.

Bacterial Challenge

The higher the number of bacteria, the harder the cow has to work to stave off the bacterial challenge. Thus, maintaining a clean environment is essential for udder health. Some bacteria are omni-present in the environment, e.g. *E. coli*, because they are shed in manure. Cows that have mastitis and leak milk can also be an important source of bacteria. Therefore, it is important to identify infected cows and treat them (if possible), or separate them from the rest of the herd, temporarily (sick pen, mastitis string) or permanently (culling). Remember that not only cows with clinical mastitis shed large numbers of bacteria! In fact, the numbers of bacteria are sometimes higher in cows with subclinical mastitis. Subclinically infected animals can infect herd mates via the milking machine (unless they are milked last or with a separate unit) or via contamination of the bedding material.

Bedding material can be the source of bacteria in two ways. It may be the original source, that is, the material with which the bacteria were brought into the barn to begin with. This is often true for *Klebsiella* in green or red wood shavings or sawdust. Or the bedding material may act as an incubator for bacteria that are deposited in it with feces, milk, saliva, hair, dust etc. This is particularly true for *Streptococcus uberis*, one of the so-called environmental streptococci. But it also applies to *E. coli*, and to a lesser extent to *Staphylococcus aureus*. Both staphylococci and non-agalactiae Streptococci can be found in bedding material, on fodder, on the skin of cows, on the skin and in the nose of dogs, cats and people, in water,

etc.

Keeping the bedding clean and dry is key to control of the bacterial load. Clean stalls at least twice a day, refresh bedding frequently, and make sure that there is adequate ventilation of the barn so that the bedding material doesn't stay damp and warm. The amount of time that cows spend lying down also affects bacterial counts in bedding: a healthy cow spends part of her day lying down, and part of her day moving around and eating. A lame cow is reluctant to stand and move around, and will spend more time lying down, contributing to higher bacterial loads in bedding material. The higher the organic content of bedding material, the more likely bacteria are to grow in it. As a rough guideline, bacterial growth decreases from straw to sawdust to wood shavings to pelleted manure to recycled newspaper to mattresses to sand. Not only use of bedding is important, but also storage of bedding material before use. Again, ventilation, dry and clean are key words.

Three special sources of bacteria in the environment are water, insects and humans/pets. Water can be contaminated with bacteria, such as *Proteus*, *Pseudomonas* or *Pasteurella*, and with algae such as *Prototheca*. If contaminated water is used for cleaning of equipment or barns, or if cows have access to the contaminated water as drinking water, bacteria and algae present in this water may cause mastitis. Filters and UV treatments may reduce this risk but are not flawless. When one cow in a herd is diagnosed with one of these pathogens, there is no cause for alarm. But when multiple cows have infections with one of these organisms, it is important to identify the source of infection and change the cleaning, drenching, or water-treatment practices.

Insects can carry bacteria from cow to cow, or from milk buckets to cows, or from other sources to cows. Flies have been shown to transmit *S. aureus*, wasps have been shown to transmit *Streptococcus dysgalactiae*, another important environmental streptococcus, summer mastitis is spread by flies, and fly damage to teat ends may make teats and udders more open to infections. Insect control may therefore need to be part of a mastitis control program. Finally, in some cases cats, dogs or people may carry bacteria that can also cause infections in cows. People may carry antibiotic resistant *S. aureus* or other bacteria and infect the cows they milk. Multi-resistance of *S. aureus* to antibiotics is rare in dairy cow mastitis, but there are cases where an infected milker caused an outbreak among dairy cows. Similarly, group G streptococcus or *Streptococcus canis* is very rare in cows, but the rare outbreaks that have been reported have often been linked to the presence of infected cats or dogs in the barn. In those cases, the hard part is to find the source of infection. Once the source individual is identified, it is easy to treat them or deny them access to the cows, and the mastitis problem can be solved.

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