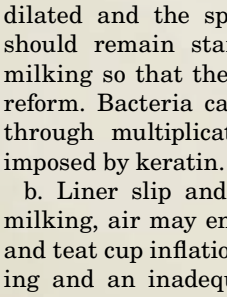
WAGING WAR IN THE UDDER

Despite our best efforts to limit mastitis, bacteria may bridge a cow's natural defenses and cause an infection.

by Stephen C. Nickerson



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1. Exposure to pathogens

The cow's teat has two structures that help prevent the entrance of bacteria: teat canal keratin (a) and the teat sphincter muscle (b).

Keratin is a waxy material that serves as a physical barrier to bacterial penetration and contains certain antimicrobial substances that inhibit microbial growth. The sphincter surrounds the teat canal and maintains tight closure between milkings to compress the keratin and prevent bacterial entrance. Keratin and the sphincter constitute the first line of defense.

Despite these defenses, some bacteria can enter the teat cistern and multiply. It is at this stage of the infection process that the cow's white blood cells, the second line of defense, kick in and attempt to prevent bacterial growth. This defense is composed of special white blood cells called neutrophils or somatic cells that gobble up and kill mastitis-causing bacteria. These somatic cells are present in relatively low concentrations or counts in milk from uninfected mammary quarters. But how do these infections occur in the first place?

2. Pathogens penetrate the teat

Because bacteria cannot be seen by the naked eye, scientists can only guess as to the actual process of bacterial penetration and establishment of infection. With our current knowledge, we believe the following modes of infection exist:

a. Environmental bacteria overload: Organisms are all over the cow's environment, and some bacteria are normal teat skin inhabitants. If a clean environment is not maintained, teat ends become exposed to and overloaded with bacteria. The more bacteria on the teat end, the greater the chances of them penetrating the keratin and invading the teat.

Overloading is particularly a problem immediately after milking when the teat canal is dilated and the sphincter is relaxed. Cows should remain standing for an hour after milking so that the keratin plug has time to reform. Bacteria can grow in the teat canal through multiplication, despite the barrier imposed by keratin.

b. Liner slip and droplet impacts: While milking, air may enter between the teat wall and teat cup inflation. This is due to wet milking and an inadequate vacuum level at the teat end. When this happens, air travels down the short milk tube and into the claw of the milking cluster. Here, the air travels across milk in the cluster, picks up small droplets of milk and carries them up through an opposite short milk tube toward the opposite teat opening. These droplets of milk may contain mastitis-causing bacteria, and they impact against the teat opening. In the open phase of the pulsation cycle, these contaminated droplets of milk pass through the open teat canal and up into the quarter.

c. Capillary action: During milking, the teat skin is bathed in milk residue left in the inflation from the previously milked cow as well as milk from the quarter being milked. If the previously milked cow had mastitis, then these bacteria are bathing the teat surface. When the inflation is removed, bacteria remain on the teat surface in a milk film, which then drifts to the end of the teat by gravity. The teat canal is dilated at this time, and bacteria in residual milk films are drawn upward into the teat canal by capillary action. Teat dipping immediately after milking replaces the bacteria-laden milk film with a germicide to eliminate potential mastitis-causing bacteria.

d. Inserting mastitis tubes: Mastitis-causing bacteria can easily be injected into a quarter when administering dry cow therapy and when treating clinical cases of mastitis. If the treatment syringe cannula is inserted fully into the teat canal, it stretches the sphincter muscle, dilates the teat canal and removes keratin, leaving an open pathway for bacteria. In addition, by fully inserting the cannula, it can carry any bacteria not eliminated by the sanitization process from the teat orifice up into the gland.

e. The milking machine: Under conditions of excess teat end vacuum, overmilking and inadequate pulsation, teat ends may become damaged. Keratin begins to turn inside out, creating a rough or flowered texture at the teat orifice. This greatly increases the surface area, providing nooks and crannies for increased bacterial growth.

3. Infection is established

When bacteria enter the inside of the mammary gland through one of the above mechanisms, they encounter somatic cells. If the somatic cells present in milk are able to eliminate all invading bacteria, then the infection does not become established. But, if the bacteria survive, a chronic or prolonged battle ensues between the somatic cells and bacteria, and the infection takes root.

4. Immune response is generated

Initially, specialized white blood cells (macrophages) in milk send signals to the bloodstream to recruit more white blood cells to the area of infection. These are mainly neutrophils, whose job it is to engulf and destroy bacteria. As bacteria become established, more and more neutrophils move from the bloodstream into the milk of the infected quarter in an attempt to eliminate the infection, resulting in an elevated somatic cell count.

If all goes well, the neutrophils eventually will be successful in eliminating all the infecting bacteria. Fewer of these cells are recruited from the blood, and the SCC will go down. Unfortunately, some of the mammary tissue may be destroyed or temporarily shut down, and milk production may not rebound until the next lactation. Some damage will be permanent. Antibiotic treatment early in the infection process is beneficial and can help minimize such losses.