The Infection Process of Mastitis

James V. Chambers

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THE INFECTION PROCESS OF MASTITIS

I. UNDERSTANDING & MANAGING THE HOST-PARASITE RELATIONSHIP

COOPERATIVE EXTENSION SERVICE, PURDUE UNIVERSITY, WEST LAFAYETTE, INDIANA
About the "Mastitis Infection Process" Series

This is the first of a series of publications intended to provide an overview of the infection process of mastitis in the dairy herd. Many factors and complex interactions enter into this disease process to influence its rate of incidence and severity. By understanding these factors and interactions, the dairy manager is better able to make mastitis-related management decisions that will not only deal effectively with present problems in the herd, but also minimize future incidents.

Contents

INTRODUCTION ................................................. 3
THE INFECTION PROCESS ............................................ 3
THE PATHWAY TO INFECTION ........................................... 3
Source .......................................................... 4
Cow
Man
Environment
Means of Transfer ............................................. 4
Human-cow contact
Cow-cow contact
Environment-cow contact
Opportunity ..................................................... 6
Teat and udder injuries
Poor hygiene practices in introducing medication
ESTABLISHING THE INFECTION ..................................... 8
Initial Attachment ............................................. 8
Adaptation, Reproduction and Defeat of Host Defenses .......... 9
Level of Infection and Clinical Symptoms ....................... 9
REFERENCES .................................................. 11

Acknowledgement

Joseph E. Griffith, Food Sciences Institute, Purdue University, developed the illustrations used in this publications. The author wishes to express his gratitude to Mr. Griffith for this contribution.
The Infection Process of Mastitis:
I. understanding and managing the host-parasite relationship

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The problem of mastitis is common to all dairy production operations. Unfortunately, the dairyman may not know mastitis exists until it shows up in the form of clotty, stringy milk, "hot" quarters and a high leucocyte count. Unfortunately, most of the management effort concerning mastitis is associated with treatment rather than prevention.

To establish an understanding of what causes mastitis, much of the information released to the dairy farmer is in an overly simplified cause-and-effect explanation. For instance, among the things that have been identified as causing mastitis are: "too much corn", "change in feed", "drafty, cold barns", "running dogs", "heredity" and "slippery floors". While these may indeed be involved somewhere along the pathway to mastitis infection, they do not define its cause.

This publication describes the three factors involved in causing mastitis (source, means of transfer and opportunity) and relates them to the infection process. By recognizing and understanding these factors, the dairy herd manager can establish management practices and procedures that minimize opportunities for the infection pathway to develop. The obvious benefits are a more profitable dairy operation and the production of higher quality milk.

The second publication in this series addresses the disease state of mastitis as it impacts the cow's defense system and her general health state. Such factors as the protective role of antibodies, the allergic reaction response to microbial toxins, tissue damage and systemic changes are discussed.

THE INFECTION PROCESS

The infection process that leads to disease involves a parasite-host relationship. This relationship begins with the exposure of a susceptible host to an environment that contains a potential parasite (1). When relating this idea to mastitis, the parasite could be any variety of microorganisms, and the dairy cow becomes the susceptible host.

When the parasite has the means and the opportunity of transfer, it gains access to the new host and starts to grow in the new habitat. Consequently, the battle for dominance between the parasite and host begins. If the parasite is successful, then the disease process is initiated. However, if the host is successful, the disease process does not occur and immunity or increased resistance may develop.

THE PATHWAY TO INFECTION

Mastitis infection involves a source, a means of transfer, and an opportunity to gain entry—all of which are common to the dairy herd environment (Figure 1). When these factors are combined with poor management, they open a pathway to infection.

To control new incidents of mastitis in the herd, the dairy farmer must use good management skills to prevent this pathway from developing. Therefore, it is essential that he recognize the environmental and equipment factors that are likely to contribute to the spread of mastitis in the herd.
are found on the cow, the herdman and the milk handler, as well as in the cow's environment (Figure 2).

Cow. An examination of the dairy cow will reveal that this animal is an excellent source, where Staphylococcus aureus, Streptococcus agalactiae, Streptococcus dysgalactiae and Streptococcus uberis can collect (5,7,12). S. aureus is found on the udder, skin and frequently on the teat surface. Str. agalactiae is commonly found in the mammary gland. If the cow is experiencing a case of mastitis or is a carrier (i.e., harbors the bacterium without demonstrating clinical symptoms), it becomes a significant source for spreading Str. agalactiae to other noninfected cows. Str. dysgalactiae and Str. uberis are commonly found on the udder, teat skin and interior lip surfaces.

Man. Man serves as a principal source for S. aureus and Str. agalactiae (5,7,12). The skin and nasal cavity provide common habitats for S. aureus. Str. agalactiae usually survives on the skin of those who handle cows frequently.

Environment. The cow's living conditions also provide other sources for mastitis-causing microorganisms (7,12). Such environmental factors as high moisture, contaminated water and soil surfaces, and bedding accommodate these microorganisms. Common mastitis-causing bacteria found on these sources are Escherichia coli, Clostridium perfringens, Klebsiella and the Pseudomonads.

Means of Transfer

The second element of the pathway to infection is the means by which the microorganism (parasite) is transferred to the dairy cow (host). The "means" may be defined as the manner, method or way a microorganism is physically transported to the entry site of the host (Figure 3). In the dairy operation, there are three basic contacts for host infection—human–cow, cow–cow and environment–cow (7,10,11,12).

Human–Cow Contact. This contact occurs principally at milking time. The personal hygiene of the milk handler and the milking practices applied determine

<table>
<thead>
<tr>
<th>Table 1. Microorganisms Associated with Mastitis-Related Incidents.</th>
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<td><strong>Bacteria</strong></td>
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<td>(Gram +)</td>
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<td>Staphylococcus</td>
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<td>Streptococcus</td>
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<td>Mycobacterium</td>
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<td>Corynebacterium</td>
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the degree of parasite transfer to the host. The hygiene of the milk handler is important because the hands, which handle the teat and udder surfaces, can transfer infecting agents to the cow. The milker's hands should be free of sores and cleaned prior to milking.

Milking procedures practiced can also control the transfer of mastitis-causing bacteria. The milk handler should not use dirty, contaminated cloths or sponges to dry off the udder and teats prior to attaching the milking unit, since these wet cloths or sponges act as a means for spreading disease-producing bacteria. A single-service paper towel is best for drying the teats and udder surfaces. A precautionary programmed rinse and sanitizing step for serviceable inflation units between cows in the milking procedure is strongly recommended.

COW-COW CONTACTS. The second basic transfer is the cow-cow relationship. This relationship involves either a direct contact (e.g., teat suckling) or indirect contact (e.g., at a common resting or gathering site in which drainage from an infected vaginal tract or mastitic discharge has been deposited).

Teat suckling can be prevented by using a nose or muzzle-type apparatus. However, it is better that the suckling behavior be dealt with by raising the dairy animal in an individual calf pen beyond its nursing stage. This helps the calf lose the need to suck before making the transition from nursing to eating solid feeds and being grouped with other calves. Ways to minimize the problem from a "common rest site" are to keep bedding clean and to try to prevent this infectious condition from developing in the "rest" areas.

ENVIRONMENT-COW CONTACT. Contact between the environment and the dairy cow is the third basic relationship that contributes in the pathway to infection. The environment is defined as the bedding, manure, soil, barnyard surfaces, polluted water and unclean or defective equipment. As the cow makes physical contact with its living environment, an infection could be starting.
To control and prevent new mastitic infections, the caretaker must utilize consistent, proven management skills in the areas of animal care, procedures and machine operations within the milk production environment. Among these environmental management practices are: (a) providing adequate housing with good ventilation; (b) frequent cleaning of cow yards and free-stall alleys; (c) providing adequate water drainage where needed; (d) maintaining clean, dry bedding in "rest" areas; (e) properly cleaning milking stalls and equipment; and (f) replacing defective, worn parts in the milking apparatus. For instance, a worn or cracked teat cup inflation unit provides a means of parasite transfer, because it can harbor mastitis-causing bacteria and thus transfer infections from one cow to another (see Figure 3). Cleaning and sanitizing efforts have little decontaminating effect on badly worn, cracked inflation units.

**Opportunity**

Opportunity is the third factor in the mastitis infection pathway. It is defined as those management practices, environmental conditions and any other influences that contribute to the defeat of the dairy cow's natural defense network against infection (5,7,11,12,13).

The dairy cow has a very effective defense network, which consists of the sphincter muscle at each teat end, the individual teat streak canal and its mesh-like keratin lining, the circulating white blood cells, and the immune system. Poor management practices and environmental conditions that assist the infection process usually cancel the physical protection provided by the teat's sphincter muscle and/or neutralize the antibacterial zone of the teat streak canal (Figure 4).

Perhaps the most frequent opportunity for mammary infection to occur is during milking. The dairy cow is at the mercy of the milk handler, and the mammary gland is most vulnerable to potential infection at that time. Add the frequency of milking two or three times a day for 305 days, and it becomes clear that opportunity for infection is constantly with the dairy cow during her lactation period. This is why milking practices continue to be the focus of so many recommended programs for controlling and preventing mastitis in the dairy herd.

Universal milking recommendations stress: (a) proper hygiene preparation of the teats and udder; (b) adequate drying of the teats and udder prior to attaching the milking units; (c) purging the teat canal of bacterial-contaminated milk while checking for evidence of mastitis (performed prior to milking phase); (d) dipping of teats into a germicidal solution after milking; and (e) inducement of the cow to stand for at least 30 minutes (by having her return to fresh feed) after leaving the milking parlor. Common to these recommendations is the natural protection provided to the mammary gland while the teat end is open.

Other opportunities for mammary infection are a result of teat and udder
injury, and poor hygiene practices when introducing medication into the mammary gland.

TEAT AND UDDER INJURIES. These generally lead to localized infections and tend to weaken the primary defensive mechanisms of the teat end and streak canal (see Figure 4). Many of them are associated with the cow's living environment. Thus, management plays an essential role in minimizing the opportunity for such injuries.

Among the management considerations are the following: (a) maintaining good housekeeping around the farm; (b) using appropriate design in the construction of the stalls, steps, ramps, barn lots, and feed bunks; (c) keeping cows comfortable, with the stalls bedded according to need; (d) isolating new or sick cows from the rest of the herd to prevent aggressiveness of the other animals; and (e) segregating cows in standing heat from the rest of the herd to prevent riding.

Weather can also contribute to teat injury. It is not uncommon to observe frozen teat ends when environmental temperatures dip below 10°F. Usually these low temperatures are coupled with a wind-chill factor, which leads to "frost bite" at the teat end and eventual infection. Under these extreme conditions, some protection can be given to the teat end by completely drying the skin immediately after milking machine removal and applying a lanolin-base salve to the area. The salve applied must not interfere with subsequent teat and udder hygiene preparation steps used in the next milking routine.

Milking machine operation can, likewise, lead to teat injury by causing a localized irritation at the teat end (7,11). Overmilking or irregular vacuum fluctuation can lead to such irritation. Irregular fluctuation occurs when the machine is not operating correctly. This may be due to an inadequate vacuum supply, regulator malfunction, excessive lifting of milk, and flooded milk lines caused by undersized pipes or inadequate slope in the pipeline. Irregular vacuum provides the opportunity for new incidents of mastitis through cross-contamination of teats during milking, teat injury or transfer of mastitis-causing bacteria to the teat canal.

POOR HYGIENE PRACTICES IN INTRODUCING MEDICATION. Another opportunity for the start of infection is through the use of bacteria-contaminated needles or tubes when introducing medication into the mammary gland (12,13). Failure to use aseptic practices in the handling of the needles and tubes leads to a potential mastitis infection. This is a common route of entry for the Pseudomonads bacteria.

To prevent this route of entry, take these precautionary measures: (a) the uncontaminated needle or tube attached to the commercial medication package should be protected against environmental contamination until it is introduced into the teat canal; and (b) prior to introducing the needle or tube into the teat canal, the teat end and surrounding skin area should be cleansed with a sterile, alcohol-soaked gauze to remove any contamination.
ESTABLISHING THE INFECTION

If the three factors in the infection pathway are present and the disease-producing microorganisms have gained entry into the mammary gland via the teat opening (see Figure 4), this means that the mastitis-causing bacteria (pathogen) have survived the antibacterial zone of the streak canal. They are now free to begin establishing themselves in the new growth environment. Thus, the battle for survival and the infection process have begun.

The infection process involves the following five steps (1,6,7,8,10,13): (a) initial attachment to the host's cell tissue; (b) adaptation to the new growth environment of the host and multiplication of the pathogen numbers; (c) depression of the host's body defenses against the pathogen; and (d) the release of toxic materials by the pathogen that stimulates allergic reactions of the host.

The ability of the pathogen to complete these steps varies from microorganism to microorganism. Also, these organisms vary in their virulence (i.e. ability to cause infection) and in their susceptibility to the host's body defenses.

Initial Attachment

The first step (step a) of the infection process is attachment of the microorganism to the exposed tissue cells within the different regions of the mammary gland (3,5,7,8,10,11,13,14, 16). Evidence from experiments suggests that systems of bacteria prefer a growth environment that involves surface attachment (Figure 5)(10,16). From these attachments, microcolonies of mixed microorganism populations develop (see Figure 5 inset), which are layered according to their respective environmental and nutritional requirements (2,3, 13).

The study of mastitis infections shows that the ability of the mastitis-causing microorganisms to attach to the tissue lining in the mammary gland is directly related to their disease-producing potential (3,4,5,6,8,9, 10,16). This is because the structure of the lining of the teat cistern is basically multi-layered, scale-like cells that have smooth surfaces. Such surfaces make attachment difficult and require the microorganisms to have special adhesion mechanisms (see Figure 5 inset (10). *S. aureus* and *St. agalactiae* possess this capability and are the most commonly encountered mastitis-causing bacteria associated with shallow penetration-type microcolonies.

The deeper the microorganism penetrates into the mammary gland, the more the tissue selectivity decreases, since the availability of surfaces for attachment is increased (3,5,7). Additionally, the structure of the deeper tissues of the duct provides niches for groups of bacteria to survive the harsh environment of the cow. Examples of those bacteria that benefit are *E. coli*, Klebsiella and the Pseudomonads.

Figure 5. Establishment of the infection process.
Adaptation, Reproduction and Defeat of Host Defenses

Following tissue attachment, the next phase (step b) of the infection process requires the microorganisms to adapt to the new growth environment of the mammary gland and to reproduce. This adaptation and growth may occur on the lining of the mammary ducts or in the secreting tissues of the gland.

As adaptation is achieved and cell growth of the microorganism begins, new cells are released via the duct network, with the disease-producing bacteria spreading to other sections of the mammary gland (Figure 6) (6,7,10,13,14). At this stage, the infection is in the subclinical phase, because the visual symptoms of mastitis are not observed. Usually, the cow's immune system is just starting to respond to the newly established infection. Thus, the leucocyte numbers are just beginning to increase. This increase is typically observed approximately 36-48 hours after the infection was established (4).

The initial numbers of the disease-producing microorganism responsible for the infection can significantly influence the degree of leucocyte response (4,6,9). The higher the initial numbers, the quicker the cow's immune system will respond. In any case, there is a race between the response of the cow's immune system and the rate of the microorganism's adaptation and cell reproduction.

If the disease-causing agent is permitted to adapt in low numbers, then adapted microbial cells are permitted to establish a low-grade infection throughout the cow's milking quarter, thereby escaping elimination by the cow's leucocytes. Should the microorganism's numbers be high enough, conceivably the leucocyte response could be rapid enough to eliminate the new infection.

Conversely, if the microorganism infects in significantly high numbers, it is able to defeat the isolation and elimination response of the leucocytes (step c) and begins rapid growth. Then the infection process will progress toward a peracute-, acute- or chronic-type infection. It reaches the clinical phase of mastitis when the symptoms of the infection are evident.

Severity of the infection depends on the rate of the disease-producing cells to reproduce, the amount of released toxic materials (step d) starting the allergic reactions, the amount of tissue destruction, and the cells' ability to produce clotting (or fibrin production) (1,8). Again, these factors vary from microorganism to microorganism and contribute to the disease-producing capabilities of the microorganism.

Level of Infection and Clinical Symptoms

The typical clinical symptoms for peracute mastitis are udder inflammation, fever, chills, loss of appetite and rapid weight loss (7); it is common to experience animal fatalities with this type of infection. The next severe infection is the acute-type mastitis; symptoms include regular disturbances.
such as mild depression and fever, and inflammation of the udder as evidenced by its hardness due to swelling (7). Subacute mastitis shows up as udder inflammation but to a lesser degree than the acute mastitis; in this case, regular signs are absent in the infected cow (7).

Chronic mastitis brings about an inflammatory response that seems to persist for an extended period of time. With chronic mastitis, sporadic flare-ups of subacute- and acute-type mastitis occasionally are evident, followed by moderation to the subclinical stage (7). Chronic mastitis is the most difficult to cure via chemotherapy, since the pathogens are well entrenched within the tissues of the mammary gland. Apparently, this entrenchment provides some protection against the cow's immune system and lowers the effectiveness of the antibiotics to destroy these disease-producing microorganisms.
REFERENCES