New York State Cattle Health Assurance Program
Mastitis Module – Veterinary Resource
Epidemiology of Mastitis

Mastitis is probably the most important health disorder on dairy farms. This is reflected in a relatively high incidence of clinical mastitis and, on many farms a high prevalence of subclinical mastitis. Additionally, mastitis causes the quality of the milk to decrease, resulting in a loss of production and/or a less than optimal product. To this end, the dairy industry faces worldwide penalty programs trying to increase the quality of milk. The most important way to continuously produce milk of good quality is to keep the mastitis situation in the herd under tight control.

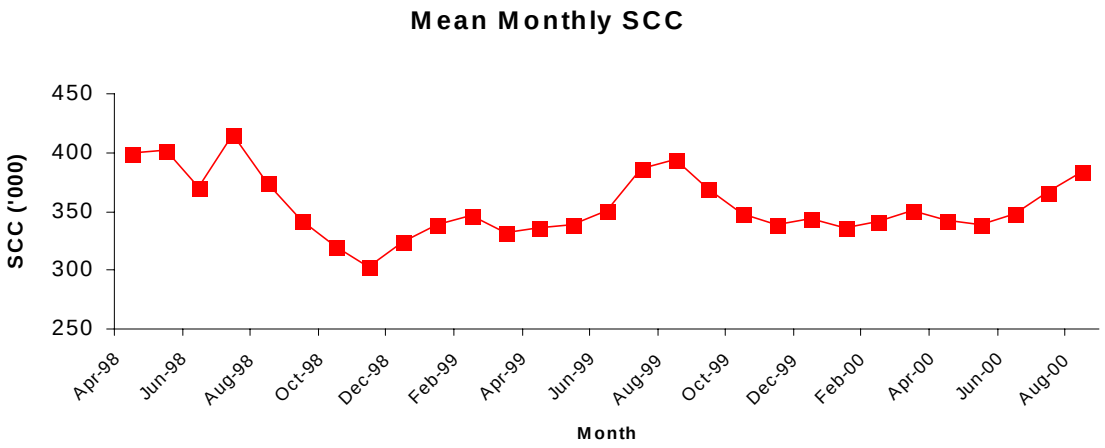
Clinical mastitis can be defined as 'a farmer observed abnormality in the milk and/or the udder'. Clinical mastitis then, is an observable disease. Cows are visibly sick, or the milk is visibly abnormal. The incidence of clinical mastitis ranges from 0 to more than 200 cases per 100 cows per year. In most studies the median incidence is around 20-25 cases per 100 cows per year. Clinical mastitis occurs in all dairy herds. Even well-managed herds, as judged by somatic cell count level and a high level of milk production, may be suffering from a high incidence of clinical mastitis (Schukken et al. 1991, Erskine et al. 1989, Hogan et al. 1990). Clinical mastitis is mostly caused by bacteria. The most important are *S.aureus*, *E. coli*, Klebsiella spp. and Streptococci (*S. uberis*, *S. dysgalactia*).

Clinical mastitis is only the 'tip of the iceberg'. Subclinical mastitis is by far the more costly disease in the majority of herds, and is often defined as the presence of a microorganism in combination with an elevated somatic cell count (SCC) of the milk. Various SCC cut-off points for the definition of subclinical mastitis have been proposed. Most recent research seems to agree to a cut-off point at about 250,000 cells (Reneau, Dohoo and Leslie 1991). Several researchers have shown a log-linear relation between the somatic cell count of the milk and the milk production of the cow. The production decreases linearly with an increase in the log(SCC). A production loss of approximately 2 lbs per natural log step above 5 is reported by several authors. Milk production loss in combination with treatment costs and culling due to mastitis constitute the major costs of mastitis. Subclinical mastitis is mostly caused by *S.aureus* and *S.agalactiae* and to a lesser extent by other Streptococci.

Not only is subclinical mastitis a problem at the individual cow level. The herd is also regularly tested for subclinical mastitis prevalence. When the bulk milk somatic cell count levels exceed certain regulatory criteria the dairy industry imposes a fine on the farmer, or completely rejects the milk. In recent years, these regulatory limits for bulk milk have been considerably tightened. The European Community and Australia/New Zealand have a regulatory limit of 400,000 cells/ml. and since 1995 Ontario, Canada has a limit of 500,000 cells/ml. In 1993, the Pasteurized Milk Ordinance introduced a limit of 750,000 cells per ml.

in the United States. Recent data from New York state show an average bulk milk SCC of approximately 350,000 cells per ml. These data are shown in Figure 1.

Figure 1. Mean Bulk Milk Somatic Cell Count in New York Milk.



High clinical mastitis incidence, prevalence of subclinical mastitis, and associated problems regarding milk quality require a rigid monitoring and control of the udder health situation in the herd.

EPIDEMIOLOGY OF MASTITIS

Mastitis is a complex disease problem. It is a classical example of the interaction of micro organisms, host factors and the environment.

Dynamics of infection

When evaluating data on udder infections, it is important to realize that cows live in an environment with a high exposure to infection. An udder infection itself (including the host response, i.e. rise in cell count, conductivity, temperature etc.) is therefore not a pathologic process, and as such does not require intervention. Only when the infection is not cured by the host-defense mechanisms, does it becomes a patho-physiologic problem. As stated before, single measurements will not be able to distinguish between infection with subsequent self cure, and infection that is not cured by the defence mechanisms. Only measurement over time will be able to distinguish between these two processes. When evaluating the prevalence of infection, a time component should always be included. This

can be two or more SCC measurements, repeated bacteriological cultures, or a combination of these two. Intervention with the host-defence mechanisms should only be undertaken when evidence of failure of this system is present.

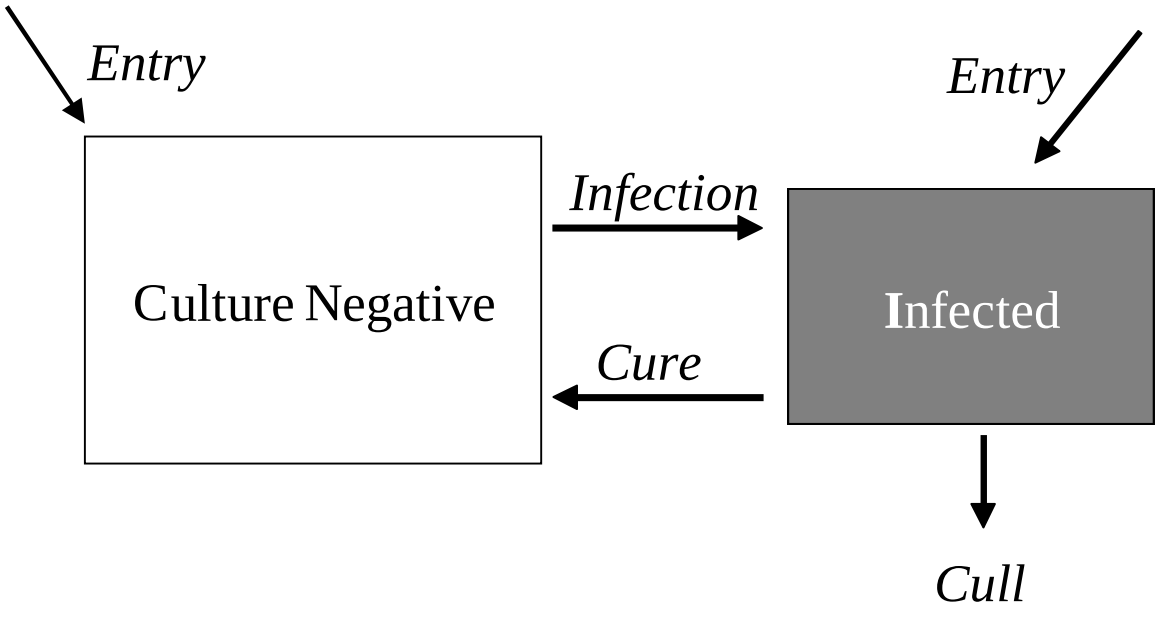
Infection patterns

Two distinct patterns in the epidemiology of mastitis can be recognized. The first pattern is a contagious disease pattern where transfer of microorganisms from animal to animal is essential to propagate the disease. The second pattern is a pattern of opportunistic microorganisms. Host factors and environmental factors put an animal at risk. A wide range of microorganism can then enter the mammary gland and cause disease.

1. Contagious disease pattern:

This pattern involves the transmission of disease from a carrier to a susceptible host. This involves mainly the spread of two major microorganisms in the dairy population. These microorganisms are *S.agalactiae* and *S.aureus* (Natzke 1981). Other epidemic contagious disease outbreaks have been reported, and involve Nocardia spp, Mycoplasma spp. and in some situations environmental streptococci. Contagious diseases only remain endemic when the mean number of susceptible individuals infected by an infected individual is appreciably larger then one (Becker 1989).

Figure 2. Dynamics of infection in a dairy herd

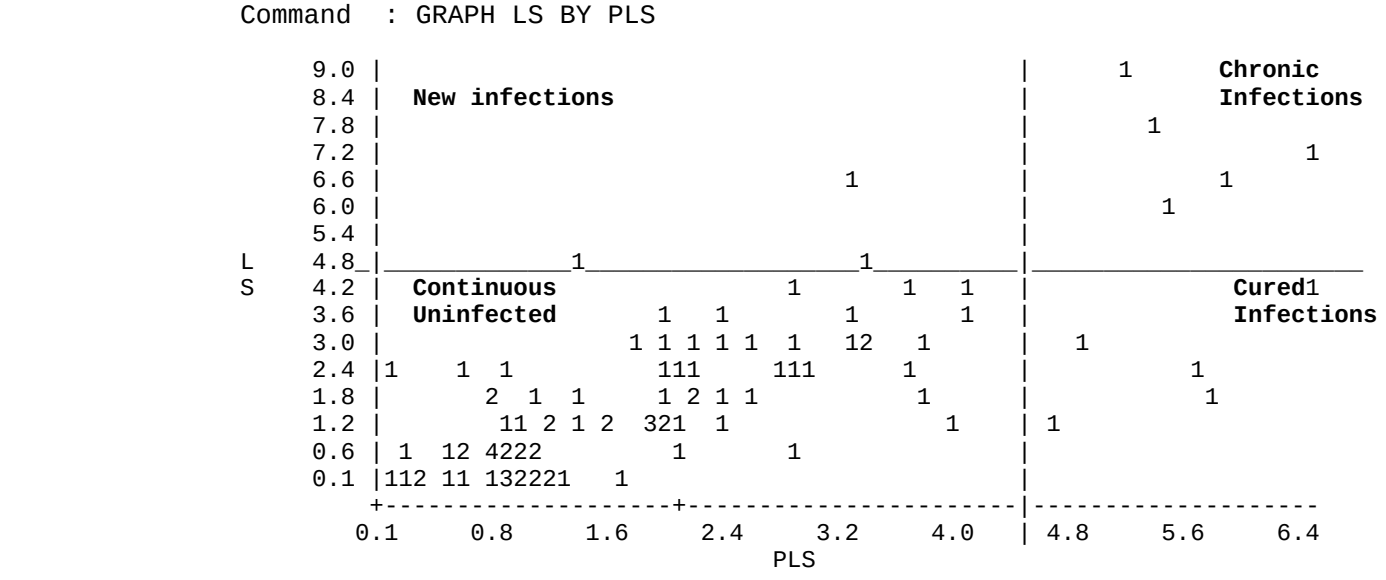


In figure 2, the components of the infection dynamics are illustrated. A herd can be divided into two populations: infected and non-infected (culture negative) cows. Animals can move into the herd in either the infected or non-infected compartment, they can move between these compartments, and they can leave the herd (culled).

It is paramount that biosecurity practices on the farm include screening for the major contagious mastitis microorganisms such as S.agalactia, S.aureus and Mycoplasma bovis. Without such a practice in place, newly arriving animals will continuously populate the group of infected animals in the herd, making containment or elimination of infection virtually impossible.

The reduction of the number of new infections is the major goal of prevention programs. New infections may be reduced by optimizing milking procedures and post milking teat disinfection. These practices will also reduce the number of shedders in the herd, separate the shedders from the uninfected cows, and optimize the immune function of the cow, which are key components of decreasing new infections. Eliminating existing infections reduces the exposure of susceptible quarters and may be obtained by treatment during lactation or at dry off, or by culling of the infected animals. Again, separation of the infected animals from the susceptible group may also be an effective method to limit the exposure of susceptible animals and reduce the risk of new infections.

Figure 3. Plotting the previous linear score versus the current linear score to evaluate new infections (top left), chronic infections (top right), healthy cows (bottom left) and cures (bottom right).



It is difficult to monitor new infections through repeated bacterial culture, but repeated somatic cell counts of individual animals can serve as a proxy measurement for new infection risks (Dohoo and Leslie 1991). Usually, a somatic cell count that increases from a

value below 250,000 cells (Linear Score below 4.5) to a value above this cut-off is considered a new infection. Cows with long term increased cell counts are considered chronically infected. A method for monitoring this infection dynamic is plotting the previous linear score (X-axis) versus the current linear score (Y-axis). The four quadrants in the graph indicate in top left the new infections, top right the chronic infections, bottom left the continuous uninfected cows and bottom right the cured infections. An example of such a scatterplot is shown in Figure 3.

When the herd is performing well, the new infection risk should be at approximately 5% or lower, and the proportion of chronic infections should also be 5% or less. To evaluate the dynamics of infection, the number of new infections should be equal to or less than the number of cures.

2. Opportunistic disease patterns:

This involves an interaction of microorganism, host factors and the environment. When solving herd problems, this epidemiologic triangle should always be kept in mind.

Microorganisms

The most important microorganisms involved are the Coliforms (E.coli and Klebsiella spp.) and the environmental streptococci (Smith et al. 1985). Microorganism related factors (serum resistance, antigen determinants) are related to the severity of clinical mastitis. However, a problem solving approach only based on microorganism reduction is likely to fail. Most cow and environmental risk factors will remain. It is likely that another microorganism will fill the niche that is created by expelling one specific species.

Host factors

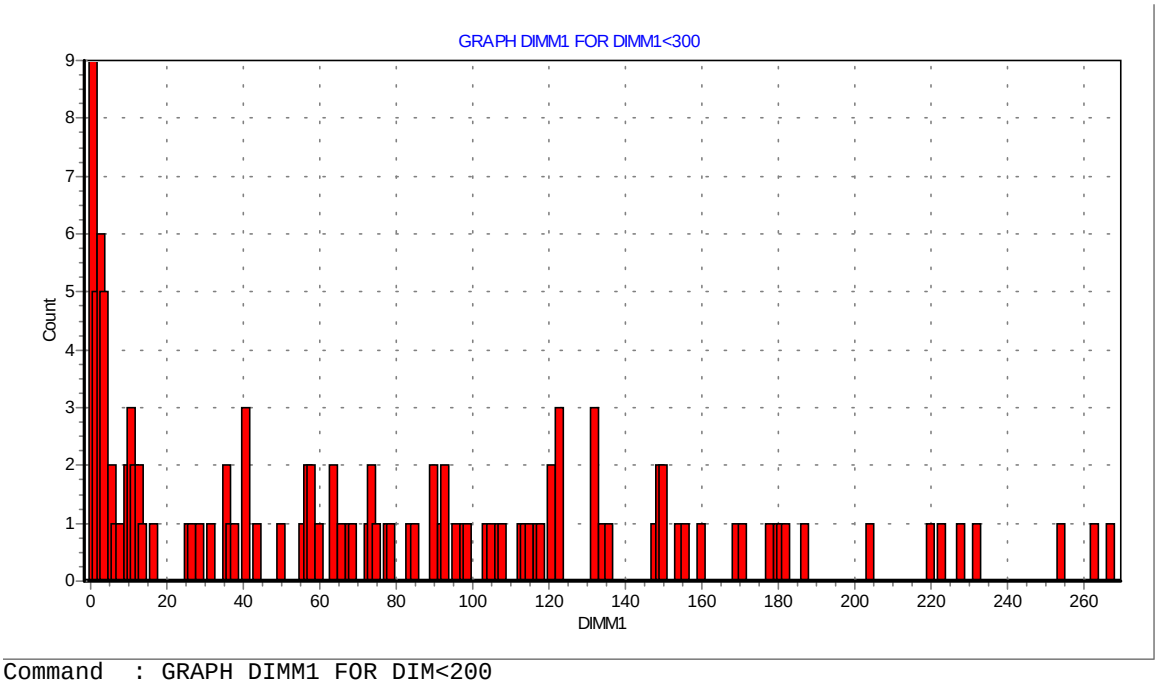
Several host factors are important in determining the outcome of an infection. Actually, most infection result in very little clinical signs, and host parameters like peripheral blood leukocyte activity, blood leukocyte count, and presence of antibodies partially predict the outcome of infection (Lohuis 1989). Other factors such as age of the animal, its metabolic status (noticeably ketosis), mineral nutrition (Selenium and vitamin E), periparturient stress and milk production level also effect the outcome of infection. Cows in early lactation appear to be specifically susceptible to clinical mastitis and have a relative high probability of becoming severely ill (see figure 4).

Environmental factors

Several factors in the environment affect the exposure of a cow to microorganisms. Sources of environmental exposure are manure, bedding, feeds, dirt, mud and water. A good example of this is *E. coli*,.which is present in the environment of the cow. Several studies have indeed linked the cleanliness of the barn, and the colony count in the bedding with the

incidence of clinical mastitis (Bramley and Neave 1975). Of critical importance is hygiene in the dry period. Observational studies have shown that most infections with coliform and environmental streptococci take place in the last two weeks before calving, and often only show signs of clinical mastitis after calving. Reducing exposure of the mammary gland by improving hygiene or providing a physical barrier at the teat end have shown to reduce the incidence of infections in this period. A typical pattern of clinical mastitis incidence is shown in the Dairy Comp 305 output in figure 4.

Figure 4. Clinical mastitis cases during the first 200 days of lactation.

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