# Heifer Mastitis

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In recent years, awareness of the prevalence of heifer mastitis has increased. More research and practical effort on farms is being devoted to heifer mastitis.

Attempts to measure prevalence of mastitis in heifers have primarily utilized cultures of quarters of animals at various stages of life until their first calving. One study[1] found 48% of heifers and 19% of their quarters to be infected with mastitis pathogens, while in another study[2] 75% of heifers' quarters were found to be infected, mostly with *Staphylococcus* sp. *and Staphylococcus aureus*. In a study[3] of clinical evidence of mastitis, only 3% of all quarters in heifers which were palpated for swollen, hard, or hot quarters had any of these signs of mastitis. However, the absence of clinical signs does not mean that mastitis was not present. When *Streptococcus* species were cultured from heifers at calving, most infections were gone by the second or third month of lactation. Over 90% of these infections did not result in somatic cell counts (SCC) over 1,000,000/ml.[4] It is apparent that not all intramammary infections of heifers become a serious case of production disease.

## Source of Infections

Sources of mastitis pathogens include the nose, vagina, skin, the streak canal, milk fed to calves, and the environment.[5] Streak canal infections were found in 93% of heifers studied, and many of these infections entered the mammary gland and progressed to mastitis; 71% of the same heifers' quarters were mastitic.[6] The entire life of a female calf, up until she has her own first calf, can be divided into three risk periods for contracting mastitis.

**From birth to weaning age**, calves can spread mastitis by suckling themselves or each other. This is more important when discard milk is fed to calves. Calves should not be housed together (or tethered where they can reach each other) until about 14 days after they have last consumed discarded milk.[4] The environment is a potential source of mastitis as always. The calving area where the calf is born, the calf hutch, calf tie stall, box stall, pasture lot, or loose housing area should be as clean and dry as possible.

**During puberty (3 to 9 months)**, heifer management often receives inadequate attention towards housing and bedding. At this age, the ductular system in the gland is developing, and secretion can be found at the teat end. Mastitic pathogens can be harbored and found in teat canals at this age.[7] Contaminated bedding, alleyways, or pasture, soiling of the teat skin, cross suckling, and flies can lead to infections. Housing should be such that teat injuries and chapped teat skin during cold weather are minimized, as these conditions can predispose cows to *Staph aureus* mastitis.[8]

**After breeding and until first calving**, the environment remains an important potential source of heifer mastitis. Increased development of the ducts in the gland, development of udder edema, and housing systems which allow dirt or manure build up on the teats can predispose the gland to mastitis. All methods of housing should be well bedded or drained. Many housing systems designed for bred heifers emphasize convenient and infrequent manure removal. This is even common in well managed herds where mastitis control is quite good, except for heifer mastitis. The environment is often the source of mastitis contracted just prior to and at calving.

## Types of Infections

The types of mastitis in heifers generally reflect the mastitis organisms in the herd. Microorganisms isolated from heifers before and after calving include most of the mastitis pathogens; *Streptococcus agalactiae, Staphylococcus aureus, Streptococcus sp., Staphylococcus sp., Mycoplasma bovis*, and other organisms.

*Streptococcus agalacties* is strongly associated with calf suckling and feeding of mastitic milk. Calves experimentally fed *Strep ag* infected milk and housed in a common pen showed high levels of *Strep ag* mastitis at calving even when the calves were exposed to the mastitic milk only during the first few weeks of life. At calving, these quarters may be subclinical or clinical, or may be observed as blind or agalactic.

Staphylococcal mastitis can also be spread by suckling but infections occur more often during puberty, and

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are often related to teat end injury. During this period, the mammary gland is changing rapidly and cultures of the streak canals have shown that these animals harbor greater numbers of bacteria than at other times.[7] These calves are often housed together under less than clean conditions. Flies and bedding are probably a major source of the infection; herds experiencing difficulty in controlling flies in heifer raising facilities have experienced higher levels of mastitis due to *Staph aureus* and other staphylococcal organisms. Low producing and blind quarters are not uncommon in *Staph aureus* infections.

*Mycoplasma bovis* has been isolated from heifers before and after calving in herd outbreaks of mycoplasmal mastitis.[9] These are frequently associated with purchase of replacement heifers, which is often combined with additional stress of introduction to the herd, including viral and respiratory infections. *Mycoplasma bovis* has been isolated from the blood stream, nasal mucus, and synovial fluids of calves which developed arthritis subsequent to the drinking of infected milk.[10] While the organism can be found in the respiratory tract of heifers and the systemic route of infection is possible, most investigators consider the streak canal the major route of infection of the mammary gland.

Environmental infections include many *Strep* species, other than *Strep agalactiae*, and coliforms found in soil, bedding and feces. It is less clear when environmental infections occur in heifers. These organisms can be detected in the streak canal at all risk times, but calves are probably at greatest risk prior to or at calving. The incidence of environmental infections may be related to the environmental conditions in the calving area and milking herd housing. They can produce clinical or subclinical mastitis, but unlike the contagious organisms they often clear spontaneously in the first couple months of lactation. However, these organisms can be the major source of new infections and affect the herd somatic cell count level.

Another less common organism, *Actinomyces* (formerly, *Corynebacterium) pyogenes*, also known in the United Kingdom to cause "Summer Mastitis", has been cultured from heifers in New York. This organism may cause mastitis in non-lactating periods and generally is an infection secondary to other bacteria.[11] *A. pyogenes* is only sporadically isolated from milk samples at the New York Quality Milk Laboratory, Cornell University, but larger outbreaks have been identified in California.[12] These infections can range from mild clinical mastitis to complete destruction of the gland with abscesses draining from tracts in the side of the gland. These infections are associated with poor fly control similar to conditions leading to *Staph aureus* infections.

## Mastitis Control and Treatment

In general, mastitis in heifers should focus on the prevention of intramammary infections. However, with the increasing awareness of heifer mastitis, more emphasis has been placed on the treatment of the infections at or before calving.

**Lactation therapy** is very successful when *Strep agalactiae* is isolated as the causative agent. Penicillin and synthetic penicillin products are highly effective in eliminating this infection from the gland. However, treatment of *Staph aureus* infections is not as successful. Early detection and therapy may be successful, but the cost of therapy and loss of milk must be considered in developing a treatment plan that includes lactation therapy in non-clinical mastitis. *Staphylococcus aureus* was eliminated from 63% of infected glands of heifers in early lactation,[13] but chronic infections do not respond as well to lactation treatment.

**Pre-lactation therapy** with a dry cow product of penicillin and streptomycin resulted in a reduction of mastitis prevalence in randomly selected heifers from 97% in late gestation to 40% at calving, while controls were unchanged (100% to 97%).[6] Another study[14] in a larger herd demonstrated that 91% of untreated glands which were infected before calving remained infected following calving, while only 36% of the treated infected glands remained infected. Although pre-lactation treatment was effective, the overall mastitis level in the herd was unaffected due to a low prevalence of pathogens before treatment. Pre-lactation intramammary therapy requires excellent restraint, skill in partial insertion for intramammary infusion of heifers, the cost of labor and antibiotic as well as screening for antibiotic residue after calving. Due to these limitations, pre-lactation therapy should only be considered and reserved for herds where *Staph aureus* is the predominant pathogen isolated from milk cultures and found to be present at a high level in heifers at calving. The value of treatment must outweigh the risk of accidental introduction of other pathogens, the cost of treatment and risk of residue in milk.

Because heifer mastitis infections can occur far removed from the time the mastitis is exhibited, the source is not always easily determined. Therefore, it is important to evaluate the complete heifer program to eliminate the sources which may be occurring during the first two years of life. Calves should be raised separately to avoid cross suckling, especially if raw milk is fed. Young heifers need a clean environment during the high risk period of puberty. Flies, weather and other

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trauma can be a major source of heifer mastitis. As with all cows, heifers should be placed in a clean area for calving and given special attention following calving. They should be milked first to avoid spreading of infections from older animals during the high risk period following calving. Heifers are the future of the herd and carry the greatest genetic potential, and deserve special attention from birth through their first lactation.

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## Gross, microscopic and ultrastructural findings in calves with congenital cerebellar hypoplasia.


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Gross, microscopic and ultrastructural findings of congenital cerebellar hypoplasia in 5 calves are reported. All of the calves showed clinical signs of ataxia since birth. Macroscopically the cerebellum had large cysts filled with clear watery fluid and a marked reduction of cerebellar parenchyma. Histologically there was cavitation of foliae white matter, loss of Purkinje cells and granuloprival hypoplasia. Electron microscopy clearly showed regressive cellular changes and platelet trombi in the cerebellar capillaries. Intrauterine infection with BVD-virus was suspected.


## Polycystic kidney and brachygnathia in Holstein-Friesian calves.


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The purpose of this report is to describe polycystic kidney associated with brachygnathia observed in 3 Holstein-Friesian calves (2 females and 1 male). Clinical and anatomo-histopathological investigation were performed on the affected calves all of which died within the second week of birth. Laboratory analysis showed anemia, hyperglycemia, hyperazotemia, an increase in the beta and gamma globulin fractions and in the AIP, LDH, gamma GT and CK activity. The Ca and P concentrations were about 9 and 12mg/100ml respectively. At necropsy, bilateral polycystic kidney, pulmonary edema and left ventricular hypertrophy were detected. The kidneys, which were smaller than usual, showed light lobulation and contained a high number of 1 to 5mm cysts filled with clear urinous fluid. Histologically the cysts were lined with flattened or cubic epithelial cells, as found in normal renal tubules. In our cases the clinical and pathologic findings suggest that polycystic kidney and brachygnathia may be related. Skeletal defects may be a manifestation of the so-called osteodystrophia fibrosa, usually most apparent in the jaw, secondary to reduced kidney function.



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