Mastitis is the more economically important disease in dairy herds. Costs originated by this disease are not only those related to loss of milk production, treatment of sick animals or culled cows, but also those related with the prevention of this disease. Intramammary infections (IMI) are most of the times caused by infection with bacterial pathogens that could be divided into contagious or environmental pathogens. The first of these are characterized to cause cow to cow infections mainly through contaminated milking equipment, milker hands, or cloths used to clean and wipe udders. In this group, *Staphylococcus aureus*, *Streptococcus agalactiae* and *Mycoplasmas spp.* are the main organisms. Environmental pathogens are those that live in the cow's environment and include environmental streptococci (*Streptococcus uberis* and *S. dysgalactiae*) and coliforms (*Escherichia coli*). These two bacterial groups account for more than the 80% of environmental mastitis.

Studies have shown that as the prevalence of contagious mastitis pathogens was reduced, the proportion of intramammary infections (IMI) by environmental pathogens increased markedly (Oliver and Mitchell, 1984; Smith et al., 1985 a, b; Oliver, 1988; Todhunter et al., 1995). Environmental mastitis has become a major problem in many well-managed dairy farms that have successfully controlled contagious pathogens. In these herds, environmental streptococci account for a significant number of both subclinical and clinical IMI in lactating and nonlactating cows (Oliver and Mitchell, 1984; Hogan et al., 1989; Todhunter et al., 1995). Environmental *Streptococcus* species involved in bovine mastitis include several Streptococcus species, but *S. uberis* appear to be the most prevalent, infecting mammary glands as favorable conditions arise (Smith et al., 1985; Oliver, 1988; Todhunter et al., 1995).

In spite of the severe economic impact caused by the high prevalence of environmental streptococci in many well-managed dairy herds, the pathogenesis of *S. uberis* mastitis in dairy cows are not well understood and constitutes a major obstacle for development of strategies to control this important mastitis pathogen.

During the last decade, important advances have been made in the understanding of the epidemiology and pathogenesis of *S. uberis* mastitis. For example, research showed that there are factors that facilitate the onset of *S. uberis* mastitis. Among these, contaminated environment particularly damp organic bedding, inadequate teat disinfection during milking routine, and cow factors such as parity, lactation stage, and nutrition are considered important predisposing factors.

As far as the dynamic of *S. uberis* mastitis concerns, it was reported that *S. uberis* IMI have a mean duration of 12 days (range from 1 to 370 d). Forty five percent of the *S. uberis* IMI showed duration less than 8 days and in 15% the infection remains more than 90 days. Fifty percent of *S. uberis* clinical IMI originate from dry-period infections and success of antibiotic treatment was estimated to be around 35% of clinical IMI. Spontaneous cure was detected in 46% of the cases and duration was typically shorter in cases with clinical than subclinical onset (Todhunter et al., 1995; Bradley & Green, 2004).
Epidemiological studies showed that *S. uberis* IMI in a particular dairy farm are caused by several strains. From these, one or two strains predominate and IMI caused by these are chronic, while IMI caused by non-predominant strains are of short duration and usually cleared by the cow. With regards to the pathogenesis of the infection, it was shown that *S. uberis* from an environment source reach teat ends and penetrates into the mammary gland via the streak canal. Upon entry into the mammary gland *S. uberis* faces mammary gland defense mechanisms, such as phagocytic cells, antimicrobial substances, and flushing effect of milk secretions. Some strains probably the non-predominant ones, induce a short inflammatory reaction band are readily cleared from the mammary gland given as clinical manifestation a short-duration IMI. Still, other strains overcome mammary defense mechanisms thus persisting into the mammary gland and causing chronic IMI. Studies conducted in our and other lab showed that persistent *S. uberis* is capable of evading defense mechanisms of the mammary gland such as phagocytosis, binding host factors and internalizing into host cells. We showed that binding host factors surface caused the masking of surface epitopes thus avoiding recognition by phagocytic cells. In addition, bound host factors form a molecular bridge with host cells receptors, which enhance adherence and internalization of *S. uberis* into bovine mammary gland cells (Almeida et al). Once *S. uberis* gain access into the cell, host defenses such as phagocytosis, antimicrobial substances present in milk or even some antibiotics are ineffective.

A recent study showed that upon infection, *S. uberis* was able to modulate apoptotic events of polymorphnuclear neutrophils (PMN's) considered as the host's first defense line. Once PMN's are initiated into the apoptotic cascade, they become inactive and unable of phagocytize invading pathogens. Thus may explain the why *S. uberis* is capable of growing and cause infection in presence of a massive infiltration of PMN's (Grant and Finch, 1996; Rambeaud et al., 2003). It also was shown that *S. uberis* initiated the apoptotic program in PMN's but delayed their removal by macrophages. The process of removal of apoptotic PMN's by macrophages is characteristic of the resolution stage of the infection. Therefore, delay in this step causes that the transition of the acute inflammatory reaction into a resolution stage shifted into a chronic state instead (Sladek et al., 2006).

In conclusion, results presented indicate that *S. uberis* is very versatile pathogen capable of survive in the mammary gland in spite of the several defense mechanisms of the host. This suggests that probably, the more effective approach to control *S. uberis* IMI is to avoid contact of this pathogen with the mammary gland teat ends. That entails that control of sites in the environment contaminated with *S. uberis* should be minimized by changing the conditions that allow the persistence of this pathogens in such locations. For example, changing damped and dirty to clean and dry bedding will be an approach that will reduce the prevalence of *S. uberis* in the cow's environment. As a general recommendation, reducing the level of environment contamination, conducting early and adequate antibiotic treatments, and maintaining good practice at drying off and use broad-spectrum dry-cow antibiotic are all together approaches that will impact the prevalence of *S. uberis* IMI.

REFERENCES


