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Factors affecting mammary gland immunity and mastitis susceptibility

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Abstract

Dairy cattle are more susceptible to mastitis during the periparturient period. It is well established that the incidence of mastitis with respect to lactation stage are directly related to changes in the composition, magnitude, and efficiency of the mammary gland defense system. There exist numerous genetic, physiological, and environmental factors that can compromise host defense mechanisms during the functional transitions of the mammary gland. For example, physiological stresses associated with rapid differentiation of secretory parenchyma, intense mammary gland growth, and the onset of milk synthesis and secretion are accompanied by a high energy demand and an increased oxygen requirement. This increased oxygen demand augments the production of oxygen-derived reactants, collectively termed reactive oxygen species (ROS). The excessive accumulation of ROS can lead to a condition referred to as oxidant stress that plays a central role in mediating uncontrolled inflammatory responses and causing tissue injury. While the last two decades have seen major progress in understanding the bovine mammary gland defense system and its function in preventing disease, diminished host defenses and increased susceptibility to mastitis continue to be a problem for dairy cattle during transition periods. This paper provides an overview of mammary immunobiology and describes those factors known to influence important mammary gland defenses during the periparturient period.

Keywords: Dairy cattle; Bovine; Mammary; Immunity; Periparturient

1. Introduction

Through genetic selection and technological advances in milk removal, the bovine mammary gland yields far more milk than is needed to nourish the newborn calf. While this excess in milk production is the basis of the dairy industry, factors associated with the intense dairy management can profoundly affect mammary gland immunity and the ability of the host to resist mastitis. Technological advances in immunology have led to the availability of new research tools that have facilitated the study of mammary gland immunity and disease pathogenesis. Several recent reviews highlight the progress that has been made in the last two decades at unraveling the bovine mammary gland immune system and its

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function (Burton and Erskine, 2003; Sordillo and Streicher, 2002; Waller, 2000). In the 1980's, considerable attention was focused on the identification and functional characterization of bovine mammary gland leukocyte populations. Different leukocyte subpopulations conveniently were identified with flow cytometry utilizing monoclonal antibodies developed to recognize unique surface molecules. Since 1990, this research has extended to the study of bovine cytokines and their potential role in the pathophysiology and control of mastitis. The availability of cytokine gene and protein sequences led to the development of several identification strategies including monoclonal antibodies, primers for polymerase chain reaction (PCR), and protein quantification with bioassays and ELISA. Moreover, recombinant cytokines were produced to explore their immunomodulatory and therapeutic uses to control disease. More recent advances in animal functional genomics also may prove to be useful when attempting to decipher the complex mammary gland responses that lead to disease resistance or susceptibility (Madsen et al., 2004; Suchyta et al., 2003). It's clear that mammary gland immunobiology is a rapidly evolving field in veterinary medicine. A better understanding of how mammary gland defense mechanisms change during critical periods of the lactation cycle may advance current efforts to reduce the incidence of mastitis in dairy cattle.

2. Susceptibility to mastitis

The incidence of mastitis increases when defense mechanisms of the mammary gland are impaired. Dairy cattle are exposed to numerous genetic, physiological, and environmental factors that can compromise host immunity and increase the incidence of mastitis. For example, emphasis on genetic selection to maximize milk production has increased metabolic stresses associated with milk synthesis and secretion and a negative correlation exists between milk production capacity and resistance to mastitis (Heringstad et al., 2003; Van Dorp et al., 1998). From an environmental standpoint, removal of milk by milking machines can cause trauma of teat end tissues, which facilitates colonization by mastitis-causing organisms. Total confinement housing, increased cow densities per unit area, and use of bedding materials that support bacterial growth also can have a marked impact on the susceptibility of dairy cattle to new intramammary infections by overwhelming important local defense mechanisms (Hogan and Smith, 2003).

The concept that immunocompromised animals are more susceptible to disease has been established. However, it is unlikely that disease susceptibility caused by increased production demands on food-producing animals will wane as animal agriculture strives to compete within a global economy. Intensity of dairy cattle management and genetic selection to increase milk production will continue and most likely result in additional immunological stresses being placed on dairy cows. Such stresses will undoubtedly lead to increased problems associated with mastitis. Completely eliminating any form of stress is impractical, and an alternative approach to reduce the influence of stress on disease susceptibility is to modify the host response to the stressor. If stress-induced changes in host immunity predispose dairy cattle to disease, then methods of up-regulating the immune response during distinct periods of stress should increase disease resistance. The challenge that confronts researchers now is to gain a better understanding of the complex interactions between the pathogenesis of bacteria, host responses needed to eliminate the pathogens from the mammary gland, and ways to enhance the immune potential of these factors before disease is established.

3. Mammary defense mechanisms

The mammary gland is protected by a variety of defense mechanisms, which can be separated into two distinct categories: innate immunity and specific immunity. Innate immunity, also known as nonspecific responsiveness, is the predominant defense during the early stages of infection. Nonspecific responses are present or are activated quickly at the site of infection by numerous stimuli: however, they are not augmented by repeated exposure to the same insult. Nonspecific or innate responses of the mammary gland are mediated by the physical barrier of the teat end, macrophages, neutrophils, natural killer (NK) cells, and by certain soluble factors. Conversely,