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Invited review: Low milk somatic cell count and susceptibility to mastitis

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ABSTRACT

An enduring controversy exists about low milk cell counts and susceptibility to mastitis. The concentration of milk leukocytes, or somatic cell count (SCC), is a well-established direct indicator of mammary gland inflammation that is highly correlated with the presence of a mammary infection. The SCC is also used as a trait for the selection of dairy ruminants less prone to mastitis. As selection programs favor animals with less SCC, and as milk cells contribute to the defense of the mammary gland, the idea that susceptibility to mastitis could possibly be increased in the long term has been put forward and is still widely debated. Epidemiological and experimental studies aimed at relating SCC to susceptibility to mastitis have yielded results that seem contradictory at first sight. Nevertheless, by taking into account the immunobiology of milk and mammary tissue cells and their role in the defense against infection, along with recent studies on SCC-based divergent selection of animals, the issue can be settled. Apparent SCC-linked susceptibility to mastitis is a phenotypic trait that may be linked to immunomodulation but not to selection.

Key words: dairy ruminant, mastitis, genetic selection, somatic cell count

INTRODUCTION: ARE COWS WITH LOW MILK SOMATIC CELL COUNTS AT GREATER RISK OF MASTITIS?

The concentration of milk cells, or the SCC, is a very sensitive biomarker of mammary gland inflammation. Variations in SCC depend mainly on the recruitment of leukocytes from blood to tissue and finally to milk, most often in response to an inflammatory reaction elicited in the mammary tissue by the intrusion of bacteria into the mammary gland (**MG**). The SCC, or parameters derived from this count, is often used to distinguish between infected and uninfected MG and to monitor udder health (Schukken et al., 2003). The SCC is thus used as an indirect indicator of MG infection. The recruited leukocytes play an important role in the defense of the MG, as shown by the very severe mastitis that develops when the leukocyte influx into milk is delayed or blocked (Craven and Williams, 1985; Vangroenweghe et al., 2005). The SCC, or often its log-derived SCS (Ali and Shook, 1980), is also widely used as a selection criterion for the genetic improvement of mastitis resistance (Odegard et al., 2003). In most countries, estimated breeding values for genetic selection on mastitis are usually based on linear models of log-transformed SCC, without a limit for low SCC. Average lactation SCC is generally used because its heritability is moderate (about 0.15) and the data are widely available (Rupp and Boichard, 2003; Rupp and Foucras, 2010). One limitation is that this trait is linked mainly to chronic subclinical infections and more loosely to short-lasting clinical infections that are typically induced by coliform bacteria. Nevertheless, a good genetic correlation (on average 0.6 to 0.7) is present between SCC and clinical mastitis (Rupp and Boichard, 1999; Heringstad et al., 2000; Carlen et al., 2004; Govignon-Gion et al., 2016), supporting the use of SCC as a surrogate for clinical mastitis, but it also shows that these 2 mastitis indicators are not the same trait (Mrode and Swanson, 1996).

The consensus is that high SCC should be selected against, but opinions diverge on the significance of low SCC. The question arises of the risk that could result from the selection of animals with very low concentrations of cells in their milk, which would predispose them to IMI and clinical mastitis. Some authors think that it is sensible to keep a reasonable concentration of cells in milk (Kehrli and Shuster, 1994; Schukken et al., 2001), whereas others have not seen a lower limit to the low SCC associated with reduced incidence of mastitis (Philipsson et al., 1995; Rupp and Boichard, 2000). It has also been put forward that the SCC has 2 distinct components, the baseline SCC under physiological and

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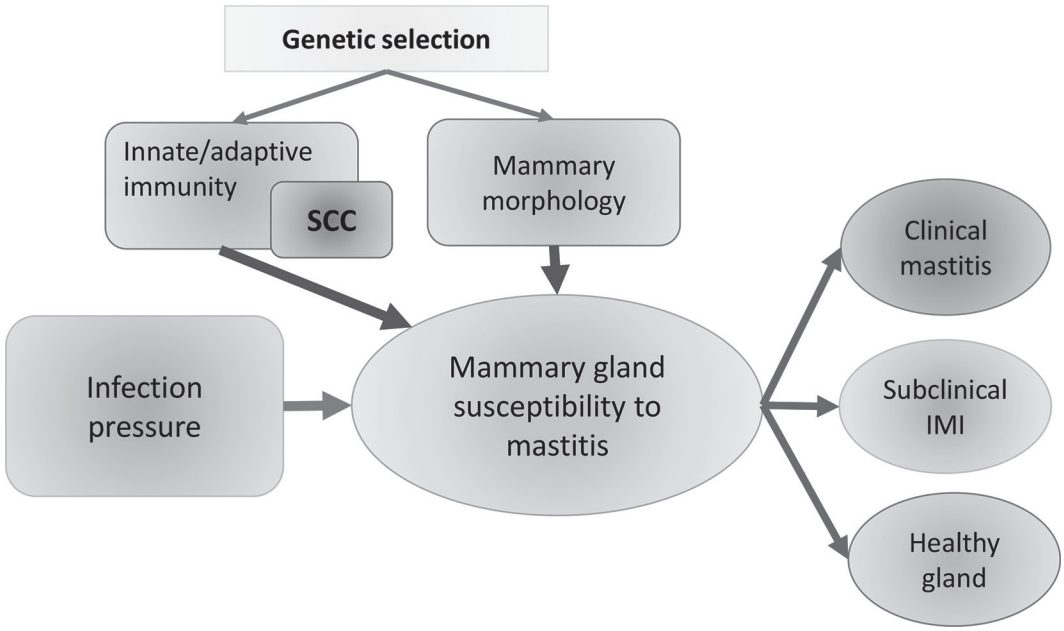

Figure 1. Relationship between some major determinants of the mammary gland (MG) health status. Genetic selection can affect the innate and adaptive immunity, for which the SCC is an important effector and marker, and important morphological traits of the MG. The innate/adaptive immunity is also determined by previous and current interactions with the microbial environment. The integration of these determinants will condition the MG susceptibility/resistance to mastitis. The MG will then respond to the infection pressure in more or less efficient ways, with the end result being clinical mastitis, transient or chronic subclinical mastitis, or no infection. Color version available online.

environmental influence and the component related to the susceptibility to mastitis (Odegard et al., 2005), and that SCS is not the same trait in cows with and without mastitis (Heringstad et al., 2006). Several epidemiological surveys have tackled the question of low SCC and susceptibility to mastitis (Green et al., 1996; Barkema et al., 1998; Beaudeau et al., 1998; Rupp et al., 2000; Suriyasathaporn et al., 2000; Whist and Osteras, 2007; van den Borne et al., 2011). Their analyses provide seemingly contradictory results that do not resolve the issue, so that the question still lingers (Wellnitz et al., 2010). Other factors have to be considered to get over this apparent deadlock. By taking into account the different mechanisms that allow somatic cells and the mammary tissue to deal with IMI, it is possible to show that the apparently contradictory epidemiological studies are not irreconcilable. Moreover, recent experimentations on dairy ruminants selected on the basis of the SCS provide new information on key points of the issue, so that a convincing settlement can be put forward.

SEEMINGLY CONTRADICTORY EPIDEMIOLOGICAL STUDIES

Numerous epidemiological surveys have grappled with the issue of the relation between SCC levels and resistance to mastitis. It is difficult to work out the resulting picture because the studies are based on recordings of clinical mastitis, bulk milk (herd) SCC, cow SCC, or quarter SCC. Moreover, the identification of the causing pathogens by bacteriological analyses have seldom been carried out. In most cases, clinical mastitis is monitored, but subclinical mastitis is often estimated through cow SCC values over the lactation period. Moreover, it is the integration of several major independent or interrelated factors that determines the MG health status (Figure 1). All this is somewhat confusing, but from this heterogeneous collection of data and concepts it is nevertheless possible to extract useful information.

The relationship between the SCC at the herd level (measured through the bulk milk SCC) and the incidence of clinical mastitis seems to depend on the type of infection prevalent in the herds. In a survey comparing herds with low (<150,000 cells/mL) and high (>750,000 cells/mL) bulk milk SCC, the incidence of clinical mastitis was higher in the latter (Erskine et al., 1988). Bacteriological analyses showed that most clinical MG infections were caused by streptococci and staphylococci in the high bulk milk SCC herds, and caused by coliform bacteria in the low bulk milk herds. This trend of a higher incidence of mastitis by coliform bacteria in a low bulk milk SCC herd has been reported by others (Hogan et al., 1989). Another study found that herds with bulk milk SCC less than 150,000 cells/