SOMATIC CELL COUNTS: A PRIMER

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Introduction

The mammary gland is made up of a remarkably sensitive tissue which has the capability of producing a large volume of secretion, milk, under normal or healthy conditions. When bacteria enter the gland and establish an infection, inflammation is initiated accompanied by an influx of white cells from the bloodstream, altered secretory function, and changes in the volume and composition of secretion. Since cell numbers in milk are closely associated with inflammation and udder health, these somatic cell counts (SCC) are accepted as the international standard measurement of milk quality. For this reason, somatic cell counts are readily available to every dairy farmer in the United States at least on a monthly basis and to farmers in most of the developed countries. Somatic cell counts are rapidly being made available in developing countries which have not previously utilized them. Extensive data are now available worldwide on large numbers of cows concerning factors affecting SCC in milk. Several comprehensive reviews or individual studies have addressed issues surrounding somatic cell counts, their variation, and the potential use of SCC for monitoring milk quality (Bodoh et al., 1976; Brolund, 1985; Dohoo and Meek, 1982; Eberhart et al., 1979 & 1982; Harmon, 1994; Miller and Paape, 1985; Raubertas and Shook, 1982; Reneau, 1985 & 1986, Reneau and Packard, 1991; Schultz, 1977; Sheldrake et al., 1983). The purpose of this overview is to establish some basic concepts regarding the cells present in milk, their numbers, and their function.

What Are Somatic Cells?

Although cell counts and leukocyte (white blood cell) counts in milk have been used for over a century in mastitis research, Prescott and Breed (1910) suggested the use of the term “body” cells because research at that time had suggested that the cells in milk were detached epithelial cells. By the late 1960's the term “somatic” (meaning body) cell count became commonplace. Today we recognize that milk somatic cells are primarily leukocytes or white blood cells, which include macrophages, lymphocytes, and polymorphonuclear neutrophils (PMN). Studies identifying cell types in milk have shown that epithelial cells or the cells which produce milk are infrequently found in udder secretions, including those from the dry gland, and range from 0 to 7% of the cell population (Table 1; Lee et al., 1980). Thus, increases in SCC at the end of lactation are not due to sloughing epithelial cells. Macrophages are the predominant cell type in normal milk and constitute between 30 and 74% of the total cells in milk from uninfected glands (Burvenich et al., 2000).
Why are Cells Present in Milk?

The cellular presence in milk is one of the important protective mechanisms of the mammary gland and may be considered to have a surveillance function in the uninfected gland. Macrophages and PMN are phagocytic cells which engulf and kill bacteria. The lymphocytes include both B-cells and T-cells that play key roles in specific immune reactions that may follow the initial response to infection.

An inflammatory response (mastitis) is initiated when bacteria enter the mammary gland through the teat canal and multiply in the milk (Bramley et al., 1996). Bacteria or their components may have a direct effect on the function of the mammary epithelium but also interact with the cells in milk, especially macrophages, and stimulate the production of numerous mediators of inflammation that may be directly involved in the pathogenesis of the disease (Gallin et al., 1992; Zeconni and Smith, 2000). These mediators include complement components, prostaglandins, leukotrienes, histamine, serotonin, interleukins, tumor necrosis factor, interferon, and other cytokines (Anderson et al., 1985; Babiuk et al., 1991; Daley et al., 1991; Giri et al., 1984; Kehrli et al., 1991; Rose et al., 1989; Schalm et al., 1971; Shuster et al, 1993; Zia et al., 1987). The classical symptoms of inflammation include increased vascular permeability, vasodilation, edema, increased blood flow, neutrophil margination and migration, decreased mammary synthetic activity, pain, and fever.

One of the initial components of the inflammatory response that is a major line of defense for the udder is the influx of PMN leukocytes into the mammary tissue (Craven and Williams, 1985; Harmon and Heald, 1982; Nickerson and Pankey, 1984; Paape et al., 1979). The PMN normally flow freely or roll through capillaries with only minimal adherence to vessel walls. During infection and inflammation, adhesion molecules are expressed, and PMN marginate or adhere to the endothelium of smaller blood vessels and pass between cells lining the vessel (Kehrli et al., 2000). Chemical messengers or chemotactic agents released from leukocytes normally in the milk or from damaged tissues attract PMN into milk in large numbers (Craven and Williams, 1985). Over 90% of the cells present in milk early in inflammation may be PMN. The PMN appear in large numbers lined up outside some alveoli (Harmon and Heald, 1982; Nickerson and Pankey, 1984). In other areas, damage to milk-synthesizing cells may be apparent, and masses of PMN may pass between epithelial cells into the lumen of the alveolus. Thus, the end result of this process is an increase in the SCC in milk resulting from PMN migration to the site of infection. The speed of the influx of PMN is believed to be a key factor in the resolution of an infection and the severity of the disease (Burvenich et al., 2000). The PMN also infiltrate the linings of teat and duct cisterns and the teat duct (Harmon and Heald, 1982; Nickerson and Pankey, 1984 & 1985). These areas may be sites of migration during the initial response to invasion. Marked mononuclear leukocyte infiltration may be noted in chronic infections (Nonnecke and Harp, 1986). Thus, increased SCC is a result of white cells being attracted into milk and is not a random event.

The function of PMN in milk is to engulf and to digest the invading bacteria (Burvenich et al., 2000; Paape et al., 1979). When PMN enter milk they also engulf other particles such as fat globules and casein, which decreases their efficiency compared with that of blood cells.
However, PMN still remain a key defense mechanism in the udder. The leukocytes in milk may also release specific substances that change the permeability of blood vessels or attract more leukocytes to the area to fight the infection. In persistent bacterial infections, leukocyte numbers may fluctuate up and down, but will generally remain abnormally high (Table 2). Such abnormal numbers of somatic cells will continue after bacteria are eliminated until healing of the gland occurs. Schultz (1977) reported that it may take days, weeks, or longer for SCC to decrease after the pathogens have been eliminated from the gland.

What is a Normal SCC?

The major factor affecting SCC is an infection of the mammary gland (Dohoo and Meek, 1982). This holds true at the quarter, cow, or bulk tank level. At the cow and quarter level the normal SCC (i.e. from uninfected quarters) is generally below 200,000 but may be below 100,000 in first lactation animals. One study estimated that 50% of uninfected cows had SCC under 100,000 per ml and 80% were under 200,000 (Eberhart et al., 1979). A study of 44 uninfected cows in their first to third lactation showed that the geometric mean SCC was 49,400 per ml (Laevens et al, 1997). A 16-month survey recently completed at the University of Kentucky showed that 4,213 bacteriologically negative quarters had a geometric mean SCC of 29,000 per ml. Thus, an elevation above the 200,000 level is generally considered abnormal and an indication of inflammation in the udder. Today we see many well-managed, high-producing herds with bulk tank SCC which remain below 200,000 and others below 100,000. No evidence exists that SCC in normal secretion from uninfected quarters is significantly influenced (i.e. exceeds 200,000 per ml) by parity, stage of lactation, or heat stress (Harmon, 1994).

There is a normal (diurnal) variation in SCC with the fraction of milk collected throughout a milking and during the time between milkings (Dohoo and Meek, 1982; White and Rattray, 1965). In general, cell counts are highest in the strippings and lowest immediately before milking. The elevated SCC may persist for up to 4 hours after milking and then gradually decline. This difference in high and low SCC in strippings vs foremilk at milking time may vary from 4- to 70-fold in individual quarters (White and Rattray, 1965). Either foremilk at milking time or composite (bucket) milk samples should be routinely used to collect SCC data, because a high correlation (r = 0.86) exists between SCC in these two sources of samples.

Summary

Somatic cells in milk are predominantly white blood cells or leukocytes which are present as one of the primary protective mechanisms of the mammary gland. Over 90% of the cells in milk during early inflammation are PMN which migrate into milk to engulf and kill bacteria. Since marked increases in SCC are a result of cells being attracted to the mammary tissue to fight an infection, it would seem unlikely that events that do not affect udder health would have a direct and dramatic effect on SCC. The major factor affecting SCC at the herd and individual cow level is the presence of intramammary infections or inflammation in the mammary gland. There is little evidence that any factor other than normal diurnal variation has a major influence on SCC in the absence of intramammary infection.
References


Table 1. Cell types found in normal bovine milk.1

<table>
<thead>
<tr>
<th>Cell type</th>
<th>% Cells (range)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neutrophil (PMN)</td>
<td>0 - 11</td>
</tr>
<tr>
<td>Macrophage</td>
<td>66 - 88</td>
</tr>
<tr>
<td>Lymphocyte</td>
<td>10 - 27</td>
</tr>
<tr>
<td>Epithelial (ductal)</td>
<td>0 - 7</td>
</tr>
</tbody>
</table>

1 From Lee et al., 1980.

Table 2. Somatic cell counts in uninfected quarters or quarters infected with S. aureus.

<table>
<thead>
<tr>
<th>Date</th>
<th>Cow 1</th>
<th>Cow 2</th>
<th>Cow 3</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Infected Qtr.</td>
<td>Uninfected Qtr.</td>
<td>Infected Qtr.</td>
</tr>
<tr>
<td>09-16</td>
<td>Fresh</td>
<td>Fresh</td>
<td>621</td>
</tr>
<tr>
<td>09-30</td>
<td>419a</td>
<td>169</td>
<td>1484</td>
</tr>
<tr>
<td>10-14</td>
<td>151</td>
<td>90</td>
<td>940</td>
</tr>
<tr>
<td>10-28</td>
<td>203</td>
<td>117</td>
<td>838</td>
</tr>
<tr>
<td>11-18</td>
<td>350</td>
<td>54</td>
<td>193</td>
</tr>
<tr>
<td>12-09</td>
<td>243</td>
<td>117</td>
<td>220</td>
</tr>
<tr>
<td>01-06</td>
<td>278</td>
<td>128</td>
<td>385</td>
</tr>
<tr>
<td>02-04</td>
<td>1551</td>
<td>99</td>
<td>431</td>
</tr>
<tr>
<td>03-04</td>
<td>377</td>
<td>84</td>
<td>471</td>
</tr>
</tbody>
</table>

a All SCC x 10³/ml.
b Negative culture