The Effect of Pathogen-Specific Clinical Mastitis on the Lactation Curve for Somatic Cell Count

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ABSTRACT

Data from 274 Dutch herds recording clinical mastitis (CM) over an 18-mo period were used to investigate the effect of pathogen-specific CM on the lactation curve for somatic cell count (SCC). Analyzed pathogens were *Staphylococcus aureus*, coagulase-negative staphylococci, *Escherichia coli*, *Streptococcus dysgalactiae*, *Streptococcus uberis*, other streptococci, and the culture-negative samples. The dataset contained 178,754 test-day records on SCC, recorded in 26,411 lactations of 21,525 cows of different parities. In lactations without both clinical and subclinical mastitis, SCC was high shortly after parturition, decreased to a minimum at 50 days in milk (DIM), and increased slowly toward the end of the lactation. Effects of CM on lactation curves for SCC differed among the pathogens isolated. Before a case of clinical *E. coli* mastitis occurred, SCC was close to the SCC of lactations without both clinical and subclinical mastitis, and after the case of CM had occurred, SCC returned rather quickly to a low level again. Similar curves were found for lactations with cases of CM associated with culture-negative samples. Before a case of clinical *Staph. aureus* mastitis occurred, average SCC was already high, and it remained high after the occurrence. Effects of CM associated with *Strep. dysgalactiae*, *Strep. uberis*, and other streptococci on the lactation curve for SCC were comparable. They showed a continuous increase in SCC until the case of pathogen-specific CM occurred, and afterwards SCC stayed at a higher level. Using SCC test-day records, these typical characteristics of each pathogen may be used to find more effective indicators of CM.

(Key words: somatic cell count, lactation curve, clinical mastitis, pathogen)

Abbreviation key: BMSCC = bulk milk SCC, CM = clinical mastitis, CNS = coagulase negative staphylococci, CSCC = corrected SCC, DIM_CM = days in milk relative to case of CM, HTD = herd-test-date, HF = Holstein-Friesian, SCM = subclinical mastitis.

INTRODUCTION

In mastitis-control programs and for genetic improvement, SCC is often used to monitor udder health. For example, breeding values for lactation-average SCC are used in selection to decrease the prevalence of subclinical mastitis (SCM) and the incidence of clinical mastitis (CM) (Mrode and Swanson, 1996). Average lactation values for SCC are used in selection programs, and variation during lactation among test-day records for SCC is most often ignored (Reents et al., 1995). Variation during lactation can be taken into account by using a test-day model, and it is expected that the longitudinal SCC data provide additional information about the pathogens involved in cases of CM, for example, *Staphylococcus aureus*, *Escherichia coli*, *Streptococcus agalactiae*, *Streptococcus dysgalactiae*, and *Streptococcus uberis*.

Suggestions that longitudinal test-day records for SCC might provide information about pathogens involved is based on comparison at herd level and on cows with experimentally induced pathogen-specific CM. When grouping dairy herds on their bulk milk SCC (BMSCC), the incidence of clinical *Staph. aureus* and *Strep. dysgalactiae* mastitis was higher in herds with high BMSCC than in herds with low BMSCC. The opposite was true for the incidence of clinical *E. coli* mastitis or of CM associated with culture-negative samples (Eberhart et al., 1982; Erskine et al., 1988; Hogan et al., 1989; Sischo et al., 1993; Barkema et al., 1998). These results may indicate that pathogens are associated with either different baseline levels for SCC or different duration of cases of CM, as both of them may affect the BMSCC. Experiments that induced CM showed that 2 d after inoculation with *E. coli* SCC peaks, and the preinfection value is approached within 3 to 4 wk again (Erskine et al., 1992; Pyörälä et al., 1994). However, within 24 h after the inoculation with *Staph. aureus*,...
SCC increases and remains high for at least 48 d (Shohani et al., 2000). These studies support that pathogen-specific effects on lactation curves for SCC might exist for both level and duration of SCC increases.

In two studies, the effect of infection status on cow SCC was investigated under practical circumstances (Sheldrake et al., 1983; Schepers et al., 1997). Sheldrake et al. (1983) compared lactation curves for SCC of quarters free from CM with lactation curves for SCC of quarters with clinical Staph. aureus, coagulase-negative staphylococci (CNS), and Corynebacterium bovis mastitis. Quarters with clinical Staph. aureus mastitis showed a considerable increase in SCC and quarters with known infection had higher SCC than quarters free from CM. Schepers et al. (1997) showed how different pathogens caused a different increase in quarter SCC. The largest increase was found for Staph. aureus and the smallest for Corynebacterium bovis. Unfortunately, the patterns of SCC before and after a case of pathogen-specific CM were not included in these studies, and specific information on DIM that CM occurred was not available. However, the patterns and the information on DIM of occurrence of CM might be useful in distinguishing between pathogens.

The overall objective in this study was to investigate pathogen-specific effects on SCC during lactation. Most published lactation curves for SCC are determined from a dataset containing lactations both with and without CM (Wiggans and Shook, 1987; Schutz et al., 1990; Weller et al., 1992). Therefore, the first objective was to estimate the effect of CM and SCM on the lactation curve. The second objective was to analyze the pattern of SCC before and after a case of pathogen-specific CM, relative to the lactation curve for lactations without both CM and SCM.

MATERIAL AND METHODS

Herds

Records on CM were available from December 1992 until June 1994 on 274 Dutch farms (Barkema et al., 1998). Lactating cows were housed in free-stall barns, and milking parlors were double-herringbone or two-sided tandem. Herds participated in the milk recording system, and annual milk production quotas were between 300,000 and 900,000 kg. The National Milk Recording System (NRS, Arnhem, The Netherlands) provided information from the three, or four weekly milk recording system. A record included national cow identification, date of occurrence, in-fection, breed, date of milk recording, date of calving, date of drying off, test-day milk yields (kg of milk, fat, and protein) and SCC (cells/ml). The breed of the cow was divided into three subclasses. The main breeds were Holstein-Friesian (HF), Dutch-Friesian, and Meuse-Rhine-Yssel.

Bacteriological Sampling

During the study, farmers were asked to collect milk samples from every quarter that they observed with CM. The aseptic sampling procedures are described by Barkema et al. (1998). Data collection of cases of CM depends heavily on the willingness of the farmers to collect milk samples, and the farmers were continually encouraged to avoid the potential bias in reporting the cases of CM, as described in Barkema et al. (1998). The samples were stored in a freezer at the farm (at approximately −20°C) and were collected for bacteriological examination at intervals of 6 to 8 wk. Bacteriological culturing of milk samples was performed according to the standards of the National Mastitis Council (Harmon et al., 1990). From each milk sample 0.01 ml was cultured, and in each culture the number of colony-forming units of each of the bacterial species was counted. Collected data contained information on the national cow identification, date of occurrence, infected quarter, and the outcome of the bacteriological culturing of the milk samples. For the analyses, only the first case of CM in each lactation was considered, and seven groups of pathogens were defined based on the incidence in the data, i.e., Staph. aureus, CNS, E. coli, Strep. dysgalactiae, Strep. uberis, streptococci other than Strep. dysgalactiae and Strep. uberis, and culture-negative samples. Cases of CM associated with any other pathogen or mixed cultures were grouped together.

Data Selection

Originally, phenotypic records on CM and bacteriological characterization were available on 49,529 lactations that had been recorded for at least 1 d during the study. Selection criteria described by De Haas et al. (2002) reduced the dataset to 47,563 lactations. For the present study, only lactations that had been recorded from calving onward were included in the dataset, to ensure that no previous cases of CM had occurred within the lactation. This criterion reduced the dataset to 26,427 lactations with 178,986 test days recorded before 308 DIM. Herd-test-date (HTD) classes with fewer than two observations for SCC were deleted from the dataset, and, therefore, the final dataset consisted of 26,411 lactations from 21,525 cows (=dataset 1). In these lactations, 178,754 test days with SCC were recorded, and 3781 first cases of CM were observed.
Lactation Curves

Three different lactation curves for SCC were determined, based on a) the test-day records from all available lactations (=dataset 1), b) only the test-day records from lactations without a case of CM (=dataset 2), and c) only the test-day records from lactations without a case of CM and with one or fewer measure of SCC above 250,000 cells/ml during the first 308 DIM (= dataset 3). Dataset 3 was created to elucidate most of the effects of SCM on the lactation curve for SCC. The threshold of 250,000 cells/ml was chosen based on findings of Dohoo and Leslie (1991). For any threshold between 200,000 and 300,000 cells/ml, they found a reasonably high specificity and quite low proportions of cows that were not at risk of developing a new infection, but which met the test criterion they set. Datasets 2 and 3 contained 151,156 test-day records of SCC in 22,630 lactations, and 117,598 test-day records of SCC in 18,438 lactations, respectively. The lactation curves were determined for heifers and multiparous cows separately by fitting an interaction with parity in the statistical model. This separation was made because multiparous cows might have had CM or SCM in a previous lactation, and there is evidence that these curves differ (Schutz et al., 1990; Weller et al., 1992; Schepers et al., 1997).

Effects of Infection Status

Two steps were involved in investigating the effect of a case of pathogen-specific CM on the lactation curve for SCC. First, SCC was expressed relative to SCC in lactations without both CM and SCM. Therefore, the SCC on each DIM for the lactations without both CM and SCM was estimated using the lactations included in dataset 3. These estimates were subtracted from the SCC recorded on the test days included in dataset 1, matching on DIM. The procedure was carried out separately for heifers and multiparous cows, and this calculated value for SCC is referred to as the corrected SCC (CSCC). Secondly, the DIM of each CSCC record was expressed relative to the DIM of the first recorded case of pathogen-specific CM (DIM_CM). The full range of 308 d before and after CM was used in the analyses, because the spline function estimated CSCC for every DIM_CM recorded in the dataset. Lactations with CM occurring late in the lactation provided information on SCC before a case of CM, and lactations with CM occurring early in the lactation provided information on SCC after a case of CM. Between 69 and 133 CSCC records were available on each DIM_CM around the day of occurrence of CM (−15 to 15 DIM_CM). Curves for CSCC were estimated for heifers and multiparous cows separately by fitting an interaction with parity in the statistical model.

Statistical Analyses

Three lactation curves for SCC as a function of DIM, and the eight CSCC curves as a function of DIM_CM, were estimated for both heifers and multiparous cows, using the spline function in AS-REML (Gilmour et al., 2001). Usually, a spline function is used for smoothing data points, and the function allows maximum flexibility and assumes no prescribed curvature. The spline function was chosen because of the expected sudden changes in SCC around a case of CM and the large amount of data available. A cubic spline is a piecewise cubic function that is constrained so that the function and its first two derivatives are continuous at the breakpoints (knots) between one cubic segment and the next.

The AS-REML program fits conventional smoothing splines, but the number and location of the knots was chosen in advance (White et al., 1999). For the curves of SCC as a function of DIM and of CSCC as a function of DIM_CM, 22 and 45 knots were set, respectively. For SCC, the knots were set closer to each other at the beginning of the lactation, because it was expected that SCC would change rapidly in the first 50 DIM but more smoothly after that toward the end of the lactation. The knots were set at 2-d intervals from 0 to 10 DIM, 5-d intervals from 10 until 30 DIM, 10-d intervals from 30 to 50 DIM, and 25-d intervals from 50 DIM toward the end of the lactation. For CSCC, the knots were symmetrically distributed before and after the case of CM. In the days around the case of CM, it was expected that large changes in CSCC would occur, so the knots were set closer to each other in this period to be able to capture these changes. The knots were fixed on the day of occurrence (d 0), and at 1 and 2 d before and after DIM_CM to be able to estimate the peak of CSCC. Intervals of 5 d were set from 5 to 20 d before and after DIM_CM, followed by 10-d intervals until 100 d before and after DIM_CM and 25-d intervals until 300 d before and after DIM_CM.

The spline function for DIM was nested within each parity class (n = 2) to be able to estimate lactation curves for SCC for heifers and multiparous cows separately. Using all data simultaneously, rather than splitting the data, allowed more accurate adjustment for HTD. The “predict” statement in AS-REML was used to estimate SCC on each DIM, and the lactation curves for SCC were created by plotting these estimates for SCC per DIM, for both heifers and multiparous cows. For the CSCC curves, the spline function for DIM_CM was nested within each combination of parity (n = 2) and
Results

Lactation Curves for SCC

The lactation curve for SCC based on all available lactations for heifers (=dataset 1) was high shortly after parturition (370,000 cells/ml), decreased to a minimum of 98,000 cells/ml around 50 DIM, and increased slowly toward 139,000 cells/ml at the end of the lactation (Figure 1a). A similar pattern was found for lactations without a case of CM (=dataset 2), but SCC was slightly lower throughout the lactation (10,000 to 30,000 cells/ml). Estimated SCC for lactations without both CM and SCM (=dataset 3) was generally low compared with all lactations and the lactations without a case of CM (=dataset 1 and 2, respectively). Although shortly after parturition, the estimated SCC was approximately 50,000 cells/ml higher in dataset 3 than in datasets 1 and 2. But at 50 DIM, SCC had already decreased to 57,000 cells/ml, and it increased from then on to 82,000 cells/ml at the end of the lactation.

Multiparous cows had generally higher SCC on each DIM than heifers. The difference ranged from 20,000 cells/ml at the start to 145,000 cells/ml at the end of the lactation for dataset 1 and from 10,000 to 125,000 cells/ml for dataset 2 (Figure 1a and b). For the heifers in dataset 3, the estimated SCC until 38 DIM was high compared with those for multiparous cows, but after 38 DIM it was the opposite. For heifers, the three lactation curves based on datasets 1, 2, and 3 were closer to each other than for multiparous cows (Figure 1a vs. b). For instance, the difference between the lactation curves based on datasets 2 and 3 was, on average, 37,000 cells/ml for heifers, whereas for multiparous cows this difference was, on average 105,000 cells/ml.

Somatic cell count rose with increasing age at calving and HF percentage. The linear and quadratic regression coefficients for age at calving indicated a nearly linear increase of SCC between 500 and 3500 d, with differences close to 385,000, 315,000, and 80,000 cells/ml in datasets 1, 2, and 3, respectively (after adjusting for parity differences). The regression coefficients for HF percentage indicated a nearly linear increase of SCC as well between 0 and 100% HF, with differences between the two extremes close to 41,000, 26,000, and 7000 cells/ml in datasets 1, 2, and 3, respectively.

Distribution of Cases of CM

The distribution of pathogens involved in cases of CM is shown in Table 1. This distribution was nearly the same in heifers and multiparous cows. In heifers, CNS and Strep. dysgalactiae were more often isolated than in multiparous cows. In multiparous cows, E. coli was the most isolated pathogen.
Table 1 also shows the distribution of the DIM of occurrence of pathogen-specific CM. Clinical mastitis associated with all pathogens, except *E. coli*, occurred earlier in heifers than in multiparous cows. The mean DIM for clinical *E. coli* mastitis was similar for heifers and for multiparous cows (82 and 80 DIM, respectively). In the first week after calving, 25% of the cases of CM associated with all pathogens except *E. coli* had occurred in heifers. Half of the cases of CM caused by *CNS*, *Strep. dysgalactiae*, *Strep. uberis*, and other streptococci had occurred in the first 14 DIM. In the first 90 DIM, 75% of the cases of CM caused by *Strep. dysgalactiae*, *Strep. uberis*, and other streptococci had occurred in the heifers.

There tended to be a difference between heifers and multiparous cows in the pathogens causing CM in late lactation. In heifers, clinical *E. coli* and *Strep. uberis* mastitis tended to occur until late in the lactation, whereas in multiparous cows, cases of CM associated with *Staph. aureus* and streptococci other than *Strep. dysgalactiae* and *Strep. uberis* tended to occur until late in the lactation.

The distribution of lactations with CM per class of HF percentage is shown in Table 2. The percentage of lactations with CM ranged from 12.9 to 16.6%, except in the lactations of animals with 1/8 HF (i.e., 33.3%).

### Effect of Pathogen-Specific CM on SCC

In general, the mean CSCC before CM was higher for multiparous cows than for heifers, except before CM associated with CNS or *Strep. dysgalactiae* (Table 3). After a case of CM, the mean CSCC for heifers was always lower than for multiparous cows, but around a case of CM the CSCC for heifers and multiparous cows were similar. The average standard error over all DIM_CM was rather large, so the CSCC curves do not seem to differ between heifers and multiparous cows.

Effects of pathogens differed for CSCC. Before a case of clinical *E. coli* mastitis CSCC was close to 0 (Figure 2b), that is, close to SCC of lactations without both CM and SCM mastitis. However, 100 d before a case of clinical *Staph. aureus* mastitis occurred, CSCC was already on a higher level, that is, 200,000 and 325,000 cells/ml for heifers and multiparous cows, respectively (Figure 2a). After a case of clinical *E. coli* mastitis had occurred, CSCC returned rather quickly to a low level again (100,000 and 200,000 cells/ml for heifers and multiparous cows, respectively) (Table 3). The CSCC curves before and after cases of CM associated with *Strep. dysgalactiae* and *Strep. uberis* were similar to each other (Figures 2c and d). A continual

### Table 1. Number of cases of pathogen-specific clinical mastitis (# cases), relative percentage (Rel %), the mean DIM, and the DIM that 10, 25, 50, 75, and 90% of the cases of pathogen-specific clinical mastitis had occurred, for heifers and multiparous cows separately.

<table>
<thead>
<tr>
<th>Pathogen</th>
<th># cases</th>
<th>Rel %</th>
<th>DIM</th>
<th>Mean</th>
<th>10%</th>
<th>25%</th>
<th>50%</th>
<th>75%</th>
<th>90%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heifers</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Staphylococcus aureus</td>
<td>85</td>
<td>11.7</td>
<td></td>
<td>65</td>
<td>0</td>
<td>2</td>
<td>35</td>
<td>106</td>
<td>153</td>
</tr>
<tr>
<td>Coagulase negative staphylococci</td>
<td>53</td>
<td>7.3</td>
<td></td>
<td>52</td>
<td>0</td>
<td>1</td>
<td>6</td>
<td>101</td>
<td>163</td>
</tr>
<tr>
<td>Escherichia coli</td>
<td>121</td>
<td>16.7</td>
<td></td>
<td>82</td>
<td>1</td>
<td>12</td>
<td>56</td>
<td>118</td>
<td>206</td>
</tr>
<tr>
<td>Streptococcus dysgalactiae</td>
<td>74</td>
<td>10.2</td>
<td></td>
<td>44</td>
<td>0</td>
<td>1</td>
<td>10</td>
<td>56</td>
<td>145</td>
</tr>
<tr>
<td>Streptococcus uberis</td>
<td>34</td>
<td>4.7</td>
<td></td>
<td>66</td>
<td>2</td>
<td>6</td>
<td>14</td>
<td>86</td>
<td>248</td>
</tr>
<tr>
<td>Other streptococci</td>
<td>43</td>
<td>5.9</td>
<td></td>
<td>48</td>
<td>0</td>
<td>3</td>
<td>14</td>
<td>71</td>
<td>165</td>
</tr>
<tr>
<td>Culture-negative samples</td>
<td>124</td>
<td>17.1</td>
<td></td>
<td>72</td>
<td>2</td>
<td>6</td>
<td>38</td>
<td>120</td>
<td>187</td>
</tr>
<tr>
<td>Multiparous cows</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Staphylococcus aureus</td>
<td>406</td>
<td>13.3</td>
<td></td>
<td>80</td>
<td>2</td>
<td>18</td>
<td>64</td>
<td>120</td>
<td>195</td>
</tr>
<tr>
<td>Coagulase-negative staphylococci</td>
<td>108</td>
<td>3.5</td>
<td></td>
<td>85</td>
<td>3</td>
<td>26</td>
<td>74</td>
<td>125</td>
<td>176</td>
</tr>
<tr>
<td>Escherichia coli</td>
<td>658</td>
<td>21.5</td>
<td></td>
<td>80</td>
<td>3</td>
<td>24</td>
<td>61</td>
<td>123</td>
<td>179</td>
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<tr>
<td>Streptococcus dysgalactiae</td>
<td>204</td>
<td>6.7</td>
<td></td>
<td>73</td>
<td>2</td>
<td>23</td>
<td>59</td>
<td>98</td>
<td>176</td>
</tr>
<tr>
<td>Streptococcus uberis</td>
<td>165</td>
<td>5.4</td>
<td></td>
<td>79</td>
<td>2</td>
<td>16</td>
<td>60</td>
<td>119</td>
<td>182</td>
</tr>
<tr>
<td>Other streptococci</td>
<td>173</td>
<td>5.7</td>
<td></td>
<td>85</td>
<td>3</td>
<td>13</td>
<td>68</td>
<td>130</td>
<td>203</td>
</tr>
<tr>
<td>Culture-negative samples</td>
<td>489</td>
<td>16.0</td>
<td></td>
<td>78</td>
<td>2</td>
<td>10</td>
<td>55</td>
<td>120</td>
<td>201</td>
</tr>
</tbody>
</table>

Table 2. The number of lactations (# lact) and the number of lactations with clinical mastitis (# lact CM1) for each class of Holstein-Friesian percentage.

<table>
<thead>
<tr>
<th>Pathogen</th>
<th># lact</th>
<th># lact CM1</th>
</tr>
</thead>
<tbody>
<tr>
<td>Holstein-Friesian</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>1109</td>
<td>170</td>
</tr>
<tr>
<td>1/8</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>2/8</td>
<td>131</td>
<td>20</td>
</tr>
<tr>
<td>3/8</td>
<td>634</td>
<td>83</td>
</tr>
<tr>
<td>4/8</td>
<td>3498</td>
<td>579</td>
</tr>
<tr>
<td>5/8</td>
<td>1286</td>
<td>193</td>
</tr>
<tr>
<td>6/8</td>
<td>9060</td>
<td>1329</td>
</tr>
<tr>
<td>7/8</td>
<td>8243</td>
<td>1061</td>
</tr>
<tr>
<td>8/8</td>
<td>2447</td>
<td>345</td>
</tr>
</tbody>
</table>
increase in CSCC was found during the days before the cases of CM occurred. After the cases of CM had occurred, CSCC tended to stay high for multiparous cows, whereas for heifers CSCC decreased slowly to a lower level. Similar CSCC curves were also found before and after a case of CM associated with streptococci other than *Strep. dysgalactiae* and *Strep. uberis* (results not shown). For heifers, CSCC lowered to a level close to SCC of lactations without both CM and SCM after a case of CM associated with streptococci other than *Strep. dysgalactiae* and *Strep. uberis* (Table 3). The CSCC curves before and after occurrence of cases of CM with culture-negative samples were comparable to the curves before and after clinical *E. coli* mastitis (results not shown).

### DISCUSSION

**Effect of Parity and Stage of Lactation**

The level of SCC has been reported to be influenced by parity (Blackburn, 1966; Lindström et al., 1981), stage of lactation (Blackburn, 1966; Bodoh et al., 1976), season (Bodoh et al., 1976; Kramer et al., 1980), and environmental and management factors (Bodoh et al., 1976). In the current study, we have corrected for environmental and management factors by adjusting for HTD in the statistical model. The effects of parity and stage of lactation have been considered and were found to be similar to what other studies have reported (Emanuelson and Persson, 1984; Wiggins and Shook, 1987; Schutz et al., 1990; Weller et al., 1992; Schepers et al., 1997). All these studies concluded that the increase in SCC toward the end of the lactation was not as great for heifers as for multiparous cows. These results might be affected by infection status, but Schepers et al. (1997) analyzed SCC measures of uninfected quarters. They also found that the increase in the logarithm of SCC toward the end of the lactation was more pronounced for multiparous cows and that the lactation curve for SCC for heifers was relatively flat.

The different shapes of the lactation curves with increasing parities indicate that SCC early and late in life may be different traits, which supports the conclusions of Coffey et al. (1985). They suggested that different mechanisms of defense against mammary infections are of primary importance at different ages and that those most important at older ages are genetically more variable.

**Standard Lactation Curve for SCC**

In the present study, the effect of a case of naturally occurring pathogen-specific CM on the lactation curve for SCC was estimated. Records for SCC were preadjusted for the lactation curve based on lactations without CM and with a maximum of one SCC test-day record above 250,000 cells/ml. We assumed that these criteria excluded lactations with both CM and SCM from dataset 1, which is probably not perfect. First, data from cows directly culled after CM, and therefore having only one test-day record above 250,000 cells/ml, were still included in dataset 3. Also, with the threshold of 250,000 cells/ml, the presence of pathogens in the udder might not have completely been excluded. For example, Laevens et al. (1997) calculated geometric mean SCC of bacteriologically negative cows at various stages of...
Figure 2a–d. The pattern of SCC before and after first cases of clinical mastitis associated with (a) *Staphylococcus aureus*, (b) *Escherichia coli*, (c) *Streptococcus dysgalactiae*, and (d) *Streptococcus uberis*, relative to the SCC of lactations without both clinical and subclinical mastitis (CSCC), for heifers (×) and multiparous cows (□).

**Pathogen-Specific Effects on CSCC**

The pathogen-specific effects on the CSCC curves showed clearly differential effects for *Staph. aureus* and *E. coli*. Before a case of clinical *Staph. aureus* mastitis occurred, CSCC was high, higher than SCC in lactations without both CM and SCM, for both heifers and multiparous cows. This suggests that the pathogen is subclinically present for some time already before clinical symptoms are observed. *Staphylococcus aureus* is known to cause chronic mastitis and SCM, with periodic clinical episodes (Harmon, 1994). That might explain why CSCC stays high after the case of clinical *Staph. aureus* mastitis and why it takes long to stabilize on the lowest level, especially for multiparous cows.

Before a case of clinical *E. coli* mastitis, CSCC was low, that is, close to the level in lactations without both CM and SCM. After the case of clinical *E. coli* mastitis, CSCC decreased rather rapidly to a level that was only slightly higher than the preinfection level, which has been reported by Erskine et al. (1992) and Pyörälä et al. (1994) as well. Cows with clinical *E. coli* mastitis have more systemic clinical signs than cows with clinical *Staph. aureus*, *Strep. dysgalactiae*, or *Strep. uberis* mastitis (Miltenburg et al., 1996). According to Harmon (1994), approximately 70 to 80% of the intramammary *E. coli* infections become clinical. Smith et al. (1985) studied the rate of IMI caused by environmental streptococci (e.g., *Strep. dysgalactiae* and *Strep. uberis*) and, on average, 53% of all streptococcal IMI was associated with clinical symptoms.

Pathogen-specific effects on a lactation curve for SCC are a combination of the standard lactation curve and
LACTATION CURVES FOR SOMATIC CELL COUNT

Figure 3a–b. A comparison of lactation curves for SCC for lactations without both clinical and subclinical mastitis (solid line) and lactations with clinical *Staphylococcus aureus* mastitis (△), clinical *Escherichia coli* mastitis (□), clinical *Streptococcus dysgalactiae* mastitis (×), and clinical *Streptococcus uberis* mastitis (○) occurring on the median DIM for heifers and multiparous cows, respectively.

The CSCC curves. To illustrate this, the estimates for CSCC (Figure 2a–d) were added to the estimates for SCC in dataset 3 (Figure 1a–b), with the assumption that the case of pathogen-specific CM occurred on the median DIM (Figure 3a–b). For multiparous cows, 50% of all cases of clinical *Staph. aureus*, *Strep. dysgalactiae*, and *Strep. uberis* mastitis had occurred around 60 DIM, whereas for heifers the medians ranged from 10 to 56 DIM (Table 1). The underlying assumption is that the CSCC curves do not depend on the DIM of occurrence of pathogen-specific CM and are, therefore, the same before and after all cases of CM. However, with the median DIM so close to calving, there is little information available on SCC before the cases of CM, and the CSCC curves will be determined predominantly by cases of CM occurring later during the lactation. The lactation curves for heifers with a case of clinical *Staph. aureus*, *Strep. dysgalactiae*, and *Strep. uberis* mastitis starts at a high level immediately after calving, and this suggests that the pathogens are already present at calving. Matthews et al. (1992) reported a rather high prevalence of *Staph. aureus* in heifers before calving (± 7%), and a higher prevalence of *Staph. aureus* in heifers than in multiparous cows at parturition.

Kehrli and Shuster (1994) argued that cows with very low SCC might be more susceptible to CM because their ability to respond to IMI would be reduced. However, the results in this study do not support that cows with low SCC during early lactation are more susceptible for CM. For none of the pathogens was SCC before a case of CM below the level of lactations without both CM and SCM. For heifers, the SCC was virtually the same before a case of clinical *E. coli* mastitis, and for multiparous cows slightly higher. This, together with the increase in SCC both before and after a case of CM associated with most pathogens, suggests that avoiding high SCC is an important tool to reduce CM.

**Use of Test-Day Records**

Heuven (1987) analyzed test-day records of SCC to predict the presence of pathogens and developed a method to identify abnormal observations of SCC, in order to exclude them from the dataset. An observation was considered to be abnormal on the basis of its deviation from the normal lactation curve. While using this exclusion method, he concluded that cows with high deviations from the normal lactation curve were more likely to be treated for CM. Besides the SCC test-day records, other sources could provide additional information for a more accurate prediction of the pathogen that is involved. This additional information can, for example, be related to 1) the cow, that is, lactation stage and parity, 2) the presence of general clinical signs, that is, body temperature and condition of the quarter (Green, 1998), and 3) the occurrence of another case of pathogen-specific CM earlier in the lactation (Lam et al., 1997; Barkema et al., 1999). However, the advantage of using SCC is that routinely recorded data can be used on a large scale.

By using the lactation average of SCC, the dynamics in SCC during the lactation are ignored, whereas a priori we expected that these might be informative for the susceptibility to CM. This is important because different pathogens affect SCC differently and because it was demonstrated that the risk for CM changes during the lactation (Barkema et al., 1998). The results in this study confirm that SCC curves and the DIM of occurrence differ for pathogens (Figure 3a–b), and by applying the SCC test-day records more effectively, the typical characteristics of each pathogen could be used to predict the pathogen involved in a case of CM. Further
analyses will be done on a more effective use of SCC test day records, by replacing the lactation average of SCC with newly defined traits for SCC, depending on the lactation curve for SCC. These new traits for SCC should distinguish pathogen-specific effects on the lactation curve for SCC.

CONCLUSIONS

The lactation curve for SCC for lactations without CM and SCM started off high shortly after parturition, decreased to a minimum around 50 DIM, and increased slowly toward the end of the lactation. The effect of CM on the lactation curve for SCC was large but differed by pathogen. Somatic cell count always remained elevated after the occurrence of pathogen-specific CM, although the effect was smaller when the interval between the occurrence of CM and the day of sampling was larger. After the occurrence of clinical E. coli mastitis, SCC approached the preinfection value after 50 DIM, whereas SCC remained high after clinical Staph. aureus mastitis. Whereas SCC was low before the occurrence of clinical E. coli mastitis, increased SCC was shown before cases of CM associated with Staph. aureus, Strep. dysgalactiae, Strep. uberis, and other streptococci. These typical characteristics of each pathogen might be useful when, instead of the lactation average of SCC, the SCC test-day records will be used in mastitis control programs or for genetic improvement. Test-day SCC may be used as indicators of the pathogen involved in a case of CM.

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