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Effects of Systematic Influences and Intramammary Infection on Differential and Total Somatic Cell Counts in Quarter Milk Samples from Dairy Cows

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Wever, P. and U. Emanuelson: Effects of systematic influences and intramammary infection on differential and total somatic cell counts in quarter milk samples from dairy cows. Acta vet. scand. 1989, 30, 465-474. – Effects of bacteriological status, stage of lactation, parity and season of sampling on differential and total somatic cell counts were estimated in quarter milk samples taken from 39 dairy cows. Log somatic cell count was affected by the bacteriological status of the quarter, as well as by the bacteriological status of adjacent quarters. Differential cell counts were affected by presence or absence of pathogens in the quarters themselves, but not by the bacteriological status of the adjacent quarters. Log somatic cell count was clearly affected by stage of lactation, due mainly to physiological variation, but possibly also accentuated by variation in infection rates throughout lactation. With the exception of early lactation, little physiological variation throughout lactation was detected for differential cell counts. Presence of infections seemed to have some indirect effect on trends throughout lactation as regards percentages of granulocytes and monocytes. Variation in somatic cell counts due to parity could be explained by variation in infection rates, rather than being physiologically determined.

physiological effects; bacteriological status; leukocytes; bovine mastitis

Introduction

Composition of the somatic cell population, in terms of the proportions of different cell types (differential cell count, DCC), in cow's milk and dry cow secretions has been the subject of several studies (e.g. *Blackburn* 1966, *Jensen & Eberhart* 1975, *Guidry et al.* 1976, *Lee et al.* 1980, *McDonald & Anderson* 1981*a, b, Fox et al.* 1985, *Kurzhals et al.* 1985). These reports indicate that the major cell type in milk from pathogen free quarters is the monocyte. Results from other studies indicate that large numbers of polymorph nuclear neutrophils (PMN) enter the mammary gland upon experimentally induced mastitis (*Jain et al.* 1971, *Jain* 1976, *Paape et al.* 1979) as well as in natural infections (*Fox et al.* 1985).

Although DCC has been studied in some detail, very little is known about systematic influences. The total somatic cell count, however, is well known to be affected by a number of factors such as stage of lactation, parity, and season of sampling (e.g. *Honkanen-Buzalski et al.* 1981, *Kennedy et al.* 1982, *Emanuelson & Persson* 1984, *Emanuelson et al.* 1988). Some results also suggest an effect on DCC of lactation stage (*Blackburn* 1966, *Jensen & Eberhart* 1975, *Guidry et al.* 1976, *Lee et al.* 1980) and parity

(*Blackburn* 1966). With the exception of *Blackburn* (1966), none of these reports studied systematically influencing factors in detail.

The purpose of the present study was to examine whether and how differential and total somatic cell counts in quarter milk samples were influenced by bacteriological status, stage of lactation, parity, and season of sampling.

Materials and methods

Individual quarter milk samples were taken during two periods (November 1981-June 1982 and October 1982-May 1983) from 39 cows in the experimental herd of the Dept. of Animal Breeding and Genetics in Uppsala. Samples were taken at weekly intervals between 1 and 3 h after morning milking and processed for bacteriological analysis and total and differential cell counting. Altogether 3663 samples gave information on both total and differential somatic cell counts, though, only 2088 samples had information on bacteriological status. The cows were of the following breeds: Swedish Jersey (n = 11), Swedish Friesian (n = 13) and Swedish Red and White (n = 15).

Bacteriological analyses were performed according to the Nordic recommendations as outlined by *Klastrup & Schmidt-Madsen* (1974). Samples were classified as pathogen-free (not infected, NI), infected with minor pathogens (MIP), or infected with major pathogens (MAP). If bacteriological status could not be confirmed, due to presence of mixed cultures, bacteriological status was designated »unknown«. Two groups were distinguished within NI samples: NI samples from udders in which all quarters were classified as NI at the time of sampling (NINI samples), and NI samples taken from udders in which all quarters had a confirmed bacteriological status at the time of sampling and one or more of the other quarters were classified as MIP or MAP (NII samples). Minor pathogens found in this study were: *Corynebacterium bovis* and coagulase-negative staphylococci. Major pathogens found in this study were: *Staphylococcus aureus, Streptococcus dysgalactiae, Streptococcus uberis, Actinomyces pyogenes* and *Escherichia coli.*

Total and differential somatic cell counts were determined with the two-colour flowcytometry method as described by *Hageltorn & Saad* (1986). Cells were differentiated into 3 groups: lymphocytes (LYM%), granulocytes (GRAN%) and monocytes (MON%). These variables were expressed in percentages of all cells. Somatic cell counts (SCC) were denoted in 1000 cells per ml and transformed to a log scale with base 10 (LSCC). Where appropriate, the geometric mean (GSCC) was calculated from the LSCC.

The total material (referred to as the complete dataset) was used to derive 2 other datasets. The first subset (referred to as the »healthy« subset) consisted of all observations from lactations in which no infection with minor or major pathogens was demonstrated in any quarter at all. For lactations in which infection with minor or major pathogens was demonstrated, the healthy subset included all observations prior to the first positive bacteriological analysis in any quarter. For the latter, data had to be available from the early lactation and onwards. The second subset (referred to as the »bacteriological« subset) consisted of all samples with a confirmed bacteriological status, i.e. that were classified as pathogen-free or infected with major or minor pathogens. Note that the 2 subsets have some observations in common, i.e. NI samples from healthy lactations. The number of quarter milk samples per cow ranged between 4 and 194 in the

complete dataset (altogether 3663 observations), between 4 and 148 in the healthy dataset (altogether 1174 observations) and between 3 and 103 in the bacteriological dataset (altogether 1659 observations).

Effects of parity, stage of lactation and period and month of sampling on LSCC and DCC were determined according to the ordinary least-squares analysis of variance as applied in the SAS General Linear Model procedure (*SAS Institute Inc.* 1985). The following fixed effects model was applied to the complete and the healthy dataset (model I):

$$Y_{ijklmno} = \mu + c_i + q_{ij} + s_k + n_l + sn_{kl} + p_m + m_n + pm_{mn} + e_{ijklmno}$$

where

$Y_{ijklmno}$ = the *ijlkmno*th observation
μ = least squares mean
c_i = effect of *i*th cow ($i = 1...39$)
q_{ij} = effect of *j*th quarter within the *i*th cow ($j = 1...4$)
s_k = effect of *k*th stage of lactation ($k = 1...8$)
n_l = effect of *l*th parity ($l = 1...3$)
sn_{kl} = effect of interaction between *k*th stage of lactation and *l*th parity
p_m = effect of *m*th period of sampling ($m = 1, 2$)
m_n = effect of *n*th (calendar) month of sampling ($n = 1...9$)
pm_{mn} = effect of interaction between *m*th period and *n*th month of sampling
$e_{ijklmno}$ = random error.

Preliminary analyses had shown that breed dit not exert any significant effect on LSCC or DCC and was therefore not included in the final model. Parities were divided into 3 groups: first, second, and third or higher parity (subsequently referred to as first, second and third parity). The number of cows and observations in the 3 parity groups, in the complete dataset, was 12 and 399, 17 and 1231, and 28 and 2052, respectively. The healthy dataset contained only 1 animal in parity 1 and this was included in parity 2. The period from calving to 305 days of lactation was divided into 8 lactation stages: days 1-10, 11-20, 21-30, 31-60, 61-90, 91-150, 151-210 and 211-305. The calendar month of sampling was coded consecutively from 1 to 9, beginning with October.

The same model, complemented with the effect of bacteriological status, was applied to the bacteriological dataset (Model II). However, there were only 8 calendar months of sampling i this model, since no sample in June 1982 had a confirmed bacteriological status. The bacteriological status of the samples was classified as NI, MIP or MAP.

Results and discussion

Bacteriological findings

About 75% of all quarter milk samples with confirmed bacteriological status were classified as NI, 20.4% as MIP, and 4.3% as MAP. The proportions of samples with known bacteriological status (NI, MIP or MAP) that were classified as MIP or MAP, were 30%, 21%, 9%, 20%, 20%, 20%, 29% and 34% in the first to the last stage of lactation, respectively. Corresponding figures were 16%, 13%, 26%, 18%, 17%, 18% and 17% from November 1981 to May 1982 (June 1982 had no samples with known bacteriological status), respectively, and 24%, 35%, 31%, 33%, 32%, 42%, 32% and 43% from October 1982 to May 1983, respectively. Thus, infection rates were high in early and late lactation. Infection rates were much higher in the second period than in the first, though no clear seasonal trend was found in either period.

Means and correlations

LSCC was lower in the healthy than in any of the other datasets (Table 1). LYM% and GRAN% were also lower and consequently

Table 1. Numbers of observations (N), means ($\bar{x}$) and standard deviations (SD) for log somatic cell counts (LSCC) and proportions of lymphocytes (LYM%), granulocytes (GRAN%) and monocytes (MON%) of all cells, and the geometric mean of the somatic cell counts (GSCC) in the complete, the healthy and the bacteriological (Bact.) dataset[1].

		LSCC		GSCC	LYM%		GRAN%		MON%	
Data	N	$\bar{x}$	SD	$\bar{x}$	$\bar{x}$	SD	$\bar{x}$	SD	$\bar{x}$	SD
Complete	3663	2.4	.6	246	15.6	10.0	38.5	15.7	46.0	17.4
Healthy	1174	2.1	.3	124	14.8	9.0	36.0	14.4	49.2	15.7
Bact	1659	2.4	.6	234	15.9	10.4	39.2	16.0	44.8	17.7

[1] See main text for description of datasets.

MON% was higher in the healthy dataset as compared with both of the other datasets.
LSCC was higher in infected (I) samples than in non-infected (NI) samples (Table 2) and the difference in geometric means (GSCC) was 6-fold. DCC had shifted towards higher GRAN% and LYM% and consequently lower MON% in I samples as compared with NI samples. This is in agreement with findings of *Fox et al.* (1985). The difference in DCC between the 2 groups indicates that granulocytes and lymphocytes may be the major cell types in clearing foreign organisms from infected quarters. Considering the 6-fold difference in GSCC between I and NI samples, there also seems to have been an influx of monocytes into infected quarters, since the change in DCC would have been more drastic if the increased SCC had been the result of an influx of granulocytes and lymphocytes only (Table 2). Due to a lack of quarter milk production data, this could not be quantified.
GSCC in NII samples was approximately 1.8-fold higher than in NINI quarters. The compositions of the somatic cell population in both groups of pathogen-free samples, however, were fairly similar (Table 2). This suggests an non-specific influx of cells into pathogen-free quarters in udders of which one or more quarters were infected. This does not agree with results from goats presented by *Dulin et al.* (1983). They found a reaction in pathogen-free udderhalves that was similar, though milder, than the reaction in corresponding infected udder halves. Not only SCC, but also GRAN% increased. Clearly this was not the case in the present

Table 2. Numbers of observations (N), means ($\bar{x}$) and standard deviations (SD) for log somatic cell counts (LSCC) and proportions of lymphocytes (LYM%), granulocytes (GRAN%) and monocytes (MON%) of all cells, and the geometric mean of the somatic cell counts (GSCC) in subgroups[1] of the bacteriological dataset.

		LSCC		GSCC	LYM%		GRAN%		MON%	
Subgroup	N	$\bar{x}$	SD	$\bar{x}$	$\bar{x}$	SD	$\bar{x}$	SD	$\bar{x}$	SD
NI	1249	2.2	.4	158	15.2	10.1	36.8	16.8	48.0	15.1
NINI	549	2.1	.4	127	14.9	8.8	37.1	15.0	48.0	15.4
NII	335	2.3	.5	221	16.1	12.6	36.2	16.0	47.7	18.6
I	410	3.0	.6	996	18.1	11.0	46.7	16.3	35.2	16.7
MIP	338	2.9	.5	742	18.2	10.9	46.5	15.5	35.3	15.0
MAP	72	3.6	.6	3966	17.4	11.5	47.8	21.8	34.9	21.2

[1] See main text for description of subgroups.

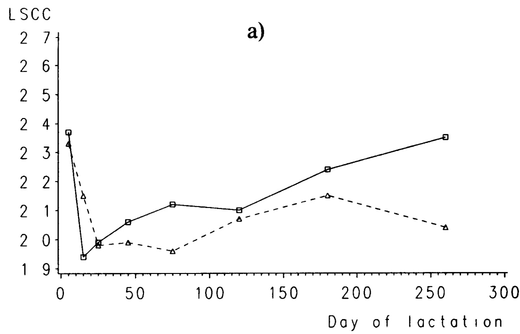
study (Table 2). Furthermore, no indication was found that the reaction in NINI quarters on an infection in any other quarter depended on the relative positions of the infected and the uninfected quarters, as was found in experimentally induced mastitis by *Schultze & Bramley* (1982). Differences between the studies regarding the duration of infections might have caused the discrepancy in results. DCC were rather similar in both infected groups (MIP and MAP), even though GSCC in MAP samples were approximately five times as high as in MIP samples (Table 2). Similar difference in SCC between MIP and MAP quarters were reported by *Hoare et al.* (1980) and *Sheldrake et al.* (1983). The difference between the 2 infected groups in GSCC may have resulted partly from greater milk losses in quarters infected with major pathogens as compared with quarters infected with minor pathogens. However, this could not be quantified in the present study.
The relationship between LSCC and DCC is shown in Table 3. Along with increased LSCC, GRAN% and LYM% also increased, while MON% decreased.
In the unadjusted complete dataset, LSCC was significantly correlated with GRAN% ($r = 0.32$), MON% ($r = 0.35$) and LYM% ($r = 0.12$). Correlations between LYM% and GRAN%, LYM% and MON% and GRAN% and MON% were -0.14, -0.45 and -0.82, respectively. Correlations between LSCC and GRAN%, and LSCC and MON% were similar to those found by *Kurzhals et al.* (1985) ($r = 0.33$ and $r = -0.36$ respectively).

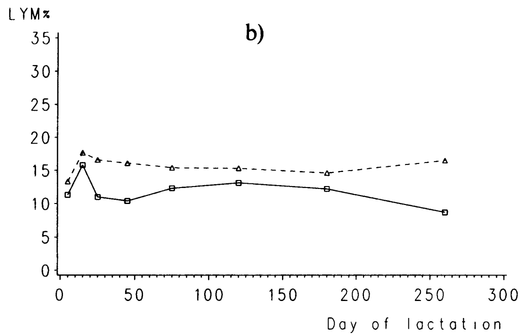
Results from analysis of variance
The proportion of total variation, explained by model I, ranged between 21.8% for LYM% and 55.4% for LSCC in the complete dataset, and between 28.7% and 47.5% for the same variables in the healthy subset. The percentage of the total variation explained by model II ranged between 36.3% for LYM% and 67.0% for LSCC, i.e. somewhat higher than for model I. Effects of cow and quarter were strongly significant in all cases, but will not be discussed further.
When *bacteriological status* was excluded from model II, the proportion of variation explained decreased by 7.2%, 0.2%, 1.9% and 1.3% for LSCC, LYM%, GRAN% and MON%, respectively. Thus, bacteriological status was much more closely related to the number of cells (LSCC) than to the proportion of different cell populations. This was expected, since the difference between infection status groups, expressed in units of

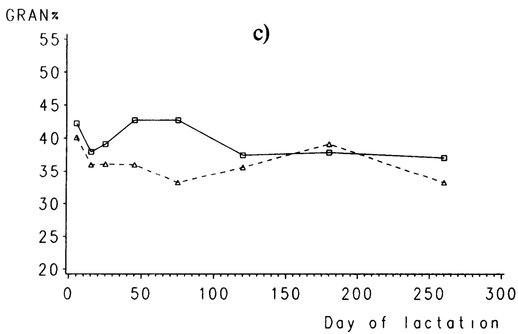
Table 3. Number of observations (N), means ($\bar{x}$) and standard deviations (SD) for log somatic cell counts (LSCC) and proportions of lymphocytes (LYM%), granulocytes (GRAN%) and monocytes (MON%) of all cells, in classes of somatic cell counts for quarters free from infection or infected with minor or major pathogens.

		LSCC		LYM%		GRAN%		MON%	
Class [1]	N	$\bar{x}$	SD	$\bar{x}$	SD	$\bar{x}$	SD	$\bar{x}$	SD
<100	466	1.84	.12	15.7	9.0	31.7	11.2	52.5	13.6
100-200	455	2.14	.08	14.5	9.5	36.9	14.0	48.5	15.8
200-400	263	2.44	.09	14.6	10.5	42.7	16.5	42.7	17.4
400-600	109	2.69	.05	18.4	13.0	42.1	16.3	39.5	17 2
600-800	71	2.84	.04	19.1	12.2	46.6	14.8	34.1	17 0
>800	295	3.42	.42	17.9	11.4	48.7	18.1	33.4	18.7

[1] Somatic cell counts in 1000 cells/ml.







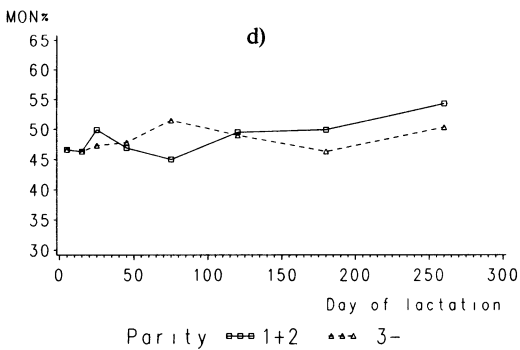
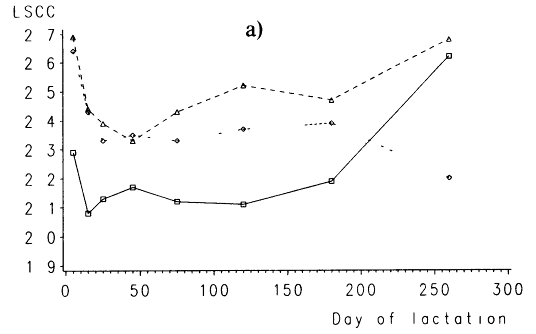
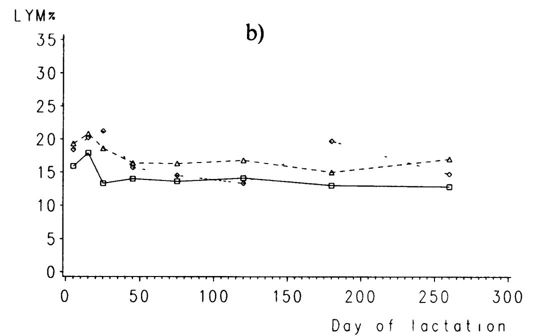
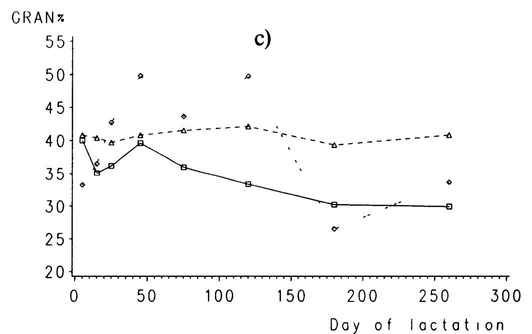


Figure 1. Effects of lactation stage on LSCC, LYM%, GRAN% and MON% in the healthy dataset, distributed by parity (LS-means obtained from Model I).







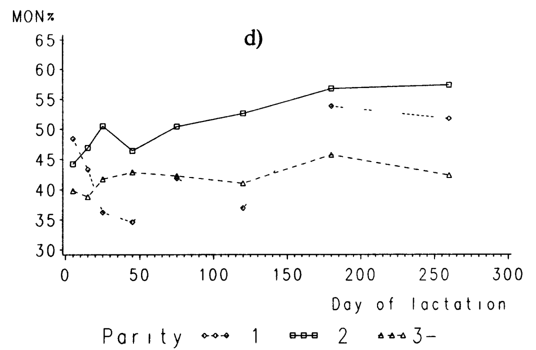


Figure 2. Effects of lactation stage on LSCC, LYM%, GRAN% and MON% in the complete dataset, distributed by parity (LS-means obtained from Model I).

standard deviation, was greater for LSCC than for any of the DCC (Table 2).

Further discussion will concern results from analysis with model I only.

Effect of *stage of lactation* significantly influenced all variables in the complete dataset, but only LSCC in the healthy subset (p <0.001 in all cases). As in earlier reports (e.g. *Honkanen-Buzalski et al.* 1981, *Kennedy et al.* 1982, *Emanuelson & Persson* 1984, *Emanuelson et al.* 1988) a marked change during lactation was found for LSCC. This change was rather similar in both datasets (Figs. 1 and 2).

There was a non-significant tendency for LYM% and GRAN% to decrease and for MON% to increase towards the end of lactation in the healthy subset (Fig. 1). Also there was a marked increase in LYM% and a decrease in GRAN% in early lactation in both parities in the healthy subset. Since the healthy subset was considered to be free from infections, the trends for LSCC and DCC in the healthy subset reflect normal physiological processes. Such processes may include irritation of the udder as a result of initiation of milking in early lactation, transfer of cells that are of importance for immunity in newborn calves, and involutation of udder tissue towards the end of lactation. It is likely that most of the variation in LSCC during the course of lactation in the healthy subset was caused by variation in milk yield, as shown previously (e.g. *Emanuelson & Persson* 1984).

Trends for all variables in the complete dataset were similar to trends in the healthy subset, especially for LSCC and LYM% (Fig. 1 and 2). Overall levels of LSCC and LYM% were higher, while levels for MON% were lower in the complete dataset, as compared with the healthy subset, almost throughout the lactation. Levels of GRAN% were higher in the complete dataset than in the healthy dataset from the second to the fifth stage of lactation and lower in the other stages. Clearly these findings do not apply to individual parities (Figs. 1 and 2). Exclusion of observations for the single animal in parity 1 from the healthy subset did not affect the results to any great extent.

Higher levels of LSCC, LYM% and GRAN% and lower levels of MON% in the complete dataset, as compared with the healthy subset, were expected, since infected quarters were included in the former, but not in the latter. Increased levels of LSCC during early and late lactation coincided with the higher infection rates in these parts of lactation. However, no such direct correlation was found between trends throughout lactation for DCC and a curve during lactation for infection rates (see bacteriological findings). The correlations found were opposite to rather than in agreement with those anticipated based on the trend found for infection rate. This was most obvious for GRAN%, and increased levels of GRAN% were found during stages in which decreased infection rate levels were demonstrated. If any correlation exists between trends throughout lactation for DCC and infection rates, this is clearly not a direct relation. It might be caused by a change in immunological reaction towards the end of lactation or during the course of persistent infections.

Parity significantly influenced all variables in the complete dataset, but only LYM% in the healthy subset (p <0.001 in all cases). Absence of an effect of parity in the healthy dataset could indicate that the effect of parity on LSCC, GRAN% and MON% in the complete dataset was related to mastitis, while for LYM% there seemed to be a physiological effect of parity. For LSCC, this tallies with *Blackburn's* findings (1966).

Results for parity 1 should be taken with a certain degree of caution, since the number

of observations on first parity cows (399) was lower than for second and third parity cows (1231 and 2052, respectively). Therefore, further discussion on the effect of parity on LSCC and DCC will be restricted to parities 2 and 3.
Third parity cows in the complete dataset had much higher levels of LSCC than second parity cows (Fig. 2). This agrees with results from other studies (e.g. *Kennedy et al.* 1982, *Sheldrake et al.* 1983, *Emanuelson & Persson* 1984). This difference was expected, since the infection rate in second parity was much lower than in the third parity (13% and 35%, respectively). The differences between second and third parity in levels of GRAN% and MON% found in the complete dataset (Fig. 2) were also anticipated, for the same reason. The absence of an effect of parity in the healthy subset (Fig. 1), indicates that effect of parity is related rather to mastitis, than to physiological origin.
The interaction between stage of lactation and parity was significant for LSCC, GRAN% and MON% in the complete dataset and for LSCC and GRAN% in the healthy subset. This indicates that parity and stage of lactation ought to be taken into account simultaneously, when interpreting results on LSCC and DCC. However, apart from level, differences between parities in effects of stage of lactation seemed rather random (Figs. 1 and 2).
The effects of *month and period of sampling,* and their interaction, significantly influenced all variables in both datasets (data not shown). Trends were similar in both datasets, which indicates that seasonal variation was not related to mastitis. Differences between the periods were large in November and December, where LSCC and GRAN% were higher and MON% was lower in the first period than in the second. Differences between the periods were only minor in the remaining months (data not shown).
LSCC was maximal at the start of each period and at its minimum at the end of each period. This corresponds to high levels in autumn and lower levels in spring. This was in accordance with results from *Kennedy et al.* (1982). No clear seasonal trend was found for LYM%, while GRAN% followed the trend for LSCC and the trend for MON% was opposite to that for GRAN% (data not shown). No correlations between infection rates in each month and levels of LSCC and DCC were found.

Concluding remarks

Not only the bacteriological status of the samples, but also stage of lactation, parity, and season of sampling should be taken into account when interpreting total and differential somatic cell counts in quarter milk samples from dairy cows. The results suggest that parity can be disregarded once bacteriological status has been taken into account. Bacteriological status of adjacent quarters is of importance for the total somatic cell counts in pathogen-free quarters.
The present study was performed on a restricted set of data. Cows were all from 1 farm and only a limited number of cows were sampled. Furthermore, there were too few observations on first parity cows to draw valid conclusions regarding differential cell counts in this important category of animals. Therefore further research on differential cell counting is advocated in order to obtain greater knowledge of factors that influence the composition of the somatic cell population in milk from dairy cows. Such information should increase our insight into circumstances under which mastitis develops and factors that play a role in its cure.

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Sammandrag

Effekt av systematiska faktorer samt bakterieinfektion på differentierad och total cellhalt i juverfjardedelsprov.

Effekten av bakterieforekomst, laktationsnummer, laktationsstadium och sasong på differentierad och total cellhalt undersoktes i juverfjardedelsprov från 39 kor. Differentierad cellhalt mattes med flodescytometer och betecknade proportionen lymfocyter, granulocyter och monocyter. Totalcellhalten påverkades av juverdelens bakteriologiska status, men aven av eventuell bakterieforekomst i intilliggande juverdelar. Den differentierade cellhalten påverkades däremot enbart av bakterieforekomsten i samma juverdel. Det fanns en tydlig fysiologisk effekt av laktationsstadium på total cellhalt, men denna effekt var troligen forstarkt av variationen i prevalens av bakterieinfektioner under laktationen. Den fysiologiska forandringen i differentierad cellhalt under laktationen var begransad, forutom under den forsta tiden. Forandringen i proportion granulocyter och monocyter under laktationen sammanfoll i huvudsak med förandringen av bakterieprevalensen. Skillnader i cellhalt mellan olika laktationer forklarades i huvudsak av motsvarande skillnader i bakterieforekomst, medan de rent fysiologiska effekterna var begransade.

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