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A COMPARISON OF HIGH YIELDING SWEDISH DAIRY HERDS WITH LOW SOMATIC CELL COUNTS AND WITH HIGH OR LOW INCIDENCE OF CLINICAL MASTITIS

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L'objectif de cette étude était de trouver des facteurs discriminants entre les élevages avec une grande incidence et une faible incidence de mammites cliniques. Les élevages ont été sélectionnées sur la base de la moyenne des performances durant 3 années consécutives. Ces élevages devaient : avoir au moins 20 vaches, appartenir au tiers supérieur des producteurs de lait, être dans le quart supérieur des élevages en fonction du taux cellulaire individuel, et faire partie du cinquième des éleveurs les meilleurs ou plus mauvais pour le critère mammites cliniques. Au total, 212 élevages ont satisfait les critères d'inclusion. La médiane de l'incidence des mammites était de 6.9 et 39.1 pour 100 vaches/an respectivement pour les groupes à faible et forte incidence. Le taux d'incidence des mammites cliniques pendant des périodes à risque, comme la période péri-partum a diminué avec la parité dans les troupeaux à forte incidence, alors que le phénomène inverse a été observé chez le groupe à faible incidence. Les élevages à forte incidence ont eu tendance a avoir des valeurs plus faibles que les niveaux de la race pour les caractéristiques de la mamelle et la résistance aux mammites, alors qu'aucune différence au niveau de la qualité génétique générale des vaches n'a été observée.

INTRODUCTION

Considerable progress in reducing the prevalence of intramammary infections has been accomplished by applying standard mastitis control programmes (Neave et al., 1969). The progress can easily be seen in the decrease in average bulk milk somatic cell count, that has been achieved in most countries during the last decade (Booth, 1995). Nevertheless, the rate of clinical mastitis continues to pose a problem, also in some herds with low somatic cell counts. The current trend of trying to decrease the use of antibiotics implies that preventive actions should be taken, rather than increasing the treatments, in order to reduce the incidence of clinical mastitis. Therefore, relevant risk factors have to be identified. Epidemiological studies have also been able to demonstrate some important determinants (eg. Schukken et al., 1990).

In a pilot study of 20 herds with low and high incidence of clinical mastitis (Tivemo-Eftring, 1996) we found tendencies of differences in the distribution of clinical mastitis over parities between the herds. Such a difference could affect the design of future epidemiological studies and the primary purpose of this study was therefore to verify this observation on a larger sample of herds.

MATERIAL AND METHODS

All herds with more than 20 cows and enrolled in the official Swedish milk, AI and disease recording schemes were eligible for inclusion in the study. Herd level data on production, health, fertility and culling were available for three consecutive years, beginning September 1, 1992. Herds were selected based on their average performance during the three years, in order to improve the reproducibility (Ekman & Emanuelson, 1994; Emanuelson, 1995). The selected herds had to be among the best third with respect to kg milk per cow and year, be among the best quarter with respect to frequency of sub-clinical mastitis, and be either among the best fifth ("LOW") or the "worst" fifth ("HIGH") with respect to incidence of clinical mastitis. Incidence of clinical mastitis was recorded as treated cases per 100 cow-years, and sub-clinical mastitis was calculated as the average proportion of cows having a low udder health score (0-2). The score is based on three consecutive individual somatic cell counts, where 0-2 indicates 0-30% risk of having a sub-clinical infectious mastitis (Brolund, 1990). Thresholds for inclusion were >8021 kg milk, >67.2% cows with low udder health score, and <9.9 and >31.4 cases of clinical mastitis per 100 cow-years, respectively.

The data used in this study was from the last of the three years used to select the herds, i.e. from September 1, 1994 to August 31, 1995. Information on monthly milk production and somatic cell counts (SCC), veterinary treatments, dates of calving and exit, and breeding values of sires, was available on all individual cows in the selected herds. Dependent variables were "lactation stage specific" SCC and incidence rate of clinical mastitis, and breeding values of sires. Stage of lactation was numbered from 0 to 22 and defined as "days in milk" (DIM) -10 to -1 (i.e. before calving), 0-6, 7-13, 14-20, 21-27, 28-34, 35-41, 42-48, 49-55, 56-69, 70-83, 84-97, 98-111, 112-139, 140-167, 168-195, 196-223, 224-251, 252-279, 280-307, 308-335, 336-363, and 363-391, respectively. However, the first two and last three stages were omitted in the analysis of SCC, since few milk samples were taken during these periods. Also, some stages had to be combined in the analysis of mastitis incidence, due to the very low rate of clinical mastitis in LOW-herds. The stages were thus defined as DIM -10 to -1, 0-6, 7-27, 28-83, 84-196, and 197-391, and numbered 0, 1, 4, 10, 16, and 22, respectively. The incidence rate was calculated for each cow and stage of lactation as number of cases divided by number of days at risk. Somatic cell counts were transformed to a log-scale with base 10 prior to analysis.

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Somatic cell counts were analysed with a general linear model, using PROC GLM of SAS. The model included the effects of herd type (LOW and HIGH), herd, cow, season, parity (1, 2 and 3+), stage of lactation, the two-way interaction between herd type and season, and the three-way interaction between herd type, parity and stage of lactation. Differences in the incidence rate of clinical mastitis were evaluated with a generalised linear model, using PROC GENMOD of SAS, with a Poisson error distribution and a log link function. The model was similar to the model for SCC, but herd and cow could not be included since such a model had analytical problems and did not converge. The breeding values for total merit index, milk production, mastitis resistance and udder conformation of the sires of the cows were modelled with a general linear model, using PROC MIXED of SAS. These models included the effects of herd type, herd, parity, and the interaction between herd type and parity.

RESULTS AND DISCUSSION

Totally 212 herds fulfilled the inclusion criteria. Some descriptive statistics on the herds are presented in Table I. The two herd types were on average very similar for all characteristics, except for the incidence of clinical mastitis.

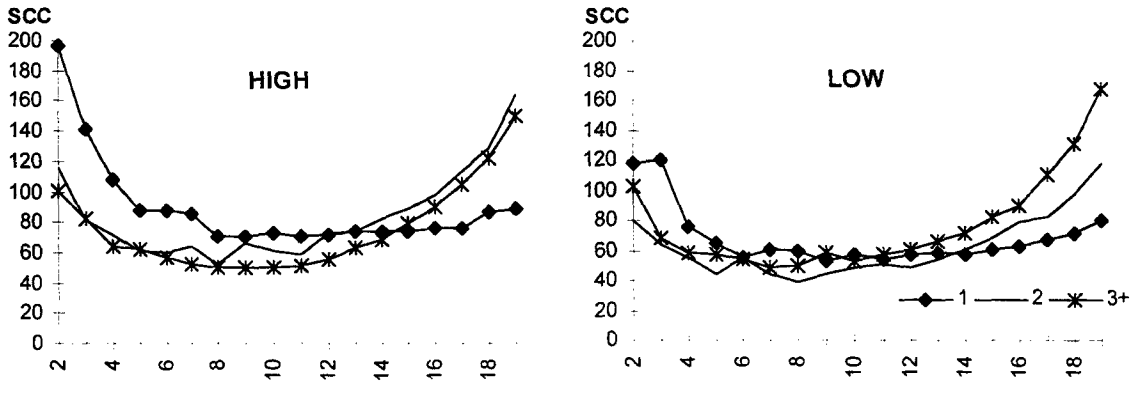
Table I
Lower quartile (25%), median (50%) and upper quartile (75%) for 3-year averages for herds with HIGH (n= 136) and LOW incidence of clinical mastitis (n=76)

	Herd type	25%	50%	75%
Herd size, cows	HIGH	25	30	40
	LOW	27	34	43
Milk yield, kg/cow and year	HIGH	8215	8491	8900
	LOW	8227	8434	8684
Low udder health score, %	HIGH	70	71	75
	LOW	69	72	76
Clinical mastitis, cases/100 cow-years	HIGH	35	39	45
	LOW	5	7	9
Bulk milk somatic cell count[a], 1000/ml	HIGH	124	144	162
	LOW	116	147	169

[a]Geometric average of three yearly geometric averages

The analyses of SCC and mastitis incidence showed a very strong interaction between herd type, parity and stage of lactation for both variables. Parameter estimates for SCC, re-transformed to the original scale, are presented in Fig. 1.

Figure 1
Estimated effects of herd type (HIGH, LOW), parity (1, 2, 3+) and stage of lactation (2-19) on somatic cell counts (SCC)



First parity cows in HIGH herds started off much higher than is usually seen, but decreased rapidly towards normal levels. Differences between the other parities were small, both within and between herd types.
Results from the analysis of the incidence rate of clinical mastitis are presented in Fig. 2 as the rate predicted by the parameter estimates from the Poisson regression. A pattern similar to the SCC curve was seen. Thus, the incidence rate during the high risk period, i.e. around calving, was higher for first parity cows compared with older cows in HIGH herds, while the opposite (and more expected) was found in LOW herds. The incidence rate was, quite naturally, much higher in HIGH herds compared to LOW herds, but was in general rather similar to results found in the literature (Rowlands & Booth, 1989; Østerås & Sandvik, 1996).