

# Identification of Risk Factors for Clinical Mastitis in Dairy Heifers

S. WAAGE,\* S. SVILAND,\* and S. A. ØDEGAARD†

\*National Veterinary Institute,  
0033 Oslo, Norway

†Department of Reproduction and Forensic Medicine,  
Norwegian College of Veterinary Medicine,  
0033 Oslo, Norway

## ABSTRACT

A nested case-control study was conducted to identify risk factors for clinical mastitis in heifers. Cases and controls originated from dairy herds that were enrolled in the Production Recording Scheme. Heifers that had been treated for clinical mastitis prepartum or on the day of parturition were eligible for inclusion as cases. The controls were heifers that had not been treated for clinical mastitis before parturition, during their first lactation, or during the dry period. In the final analysis, 4256 heifers with mastitis and 67,072 control heifers were included.

An increase in the incidence of clinical mastitis in the herd, a decrease in the bulk milk somatic cell count, and an increase in the mean milk yield of the herd were associated with an increased risk for clinical mastitis. The risk varied among regions, and, depending on region, significant influences of both herd size and composition of the diet were observed. Heifers kept on pasture in summer were at a decreased risk for clinical mastitis. Calving in late spring or summer was associated with greater risk than was calving at other times of the year. An increase in age at first calving was associated with increased risk of mastitis. Mastitis was also more likely to occur in heifers leaking milk or in heifers that had a low milk flow rate in the subsequent lactation. For purchased heifers, risk factors were identified in both their previous and current herds. (**Key words:** mastitis, heifer, risk factors)

**Abbreviation key:** **BMSCC** = bulk milk SCC, **C<sub>HL</sub>** = Hosmer-Lemeshow goodness of fit statistic, **CI** = confidence interval, **OR** = odds ratio.

## INTRODUCTION

Examination of mammary secretions collected from heifers prepartum has shown that the mammary

glands of many heifers harbor organisms that frequently cause mastitis. Although results vary, a prevalence of infected quarters between 30 and 50% prior to or at the time of parturition seems to be quite common (1, 6, 26). Minor pathogens, especially coagulase-negative staphylococci, are the most frequent isolates, but major pathogens, primarily *Staphylococcus aureus*, are not uncommon, and a quarter prevalence around 5% (21) or even higher (6) has been reported. Usually, the infection is sub-clinical. However, clinical cases occasionally occur prior to parturition and cause substantial losses (27). *Staphylococcus aureus*, *Streptococcus dysgalactiae*, *Arcanobacterium pyogenes*, *Escherichia coli*, and coagulase-negative staphylococci seem to be the most important organisms that cause clinical mastitis in heifers (10, 16, 31).

A great number of risk factors for bovine clinical mastitis have been identified, including animal, environmental, and management factors (25, 30). Most cases of clinical mastitis occur postpartum, and improper milking practices or inadequate milking equipment are important predisposing factors. With the exception of these factors, one should expect clinical mastitis in heifers prepartum to be associated largely with the same risk factors as those for clinical mastitis in lactating cows. Only a few studies have focused on risk factors for clinical mastitis in heifers, and those studies included prepartum as well as postpartum cases. Myllys and Rautala (17) examined, within different levels of herd milk yield, the univariate relationships between possible herd risk factors and the incidence of clinical mastitis in heifers around parturition. An increase in the mean milk yield per cow in the herd, an increase in the overall herd incidence of clinical mastitis, and a decrease in the bulk milk SCC (**BMSCC**) were associated with increased risk for clinical mastitis in heifers. Certain animal risk factors were identified in the same study. During summer, mastitis caused by *A. pyogenes*, often together with some other bacteria (7), is prevalent in heifers in some regions and seems to be closely

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related to the activity of the fly *Hydrotaea irritans* (13), although other risk factors are also involved (8).

In the present study, data describing dairy herds and heifers that participated in the Norwegian Production Recording Scheme were analyzed to identify risk factors for clinical mastitis that occurred in heifers perpartum or on the day of parturition.

## MATERIALS AND METHODS

### Clinical Mastitis Data

A national health card system for cattle has been in operation in Norway since 1975 for all herds that participate in the Production Recording Scheme (29). In 1995, 85.0% of all Norwegian dairy herds or 90.0% of all dairy cows in Norway were enrolled in the Production Recording Scheme. The health card system is part of the national Health Service for Cattle, a joint program established by veterinary authorities, organizations of the dairy and meat industry, cattle breeding organizations, and the Norwegian Veterinary Association. All disease treatments are recorded by veterinary surgeons. For heifers and cows, individual health cards are employed. At birth, a unique identification based on a combination of farm and animal identity numbers is assigned to each individual calf. Different clinical diagnoses are coded numerically, and separate code numbers are used for acute and chronic cases of clinical mastitis. Recordings are regularly reported to a central database by field personnel of the dairy companies, and data are stored as individual records, accumulating information on each cow until culling.

Because of strict regulations regarding the sale of antibiotics and other drugs in Norway, cases of clinical mastitis are treated only by veterinary surgeons. The health card system is part of the official disease recording system for animals, and veterinary surgeons are required by law to register all treatments. Regular reports to the practitioners from the Health Service for Cattle and the possibility of on-line access to the detailed, updated health status of each dairy farming client encourage the loyalty of the practitioners to the system.

In the present study, clinical mastitis was chosen as the disease variable for the heifers; the first recording of either acute or chronic clinical mastitis for each heifer was used.

### Information on Heifers

Data were obtained from the database of the Production Recording Scheme. In addition to disease

recordings obtained from the health card system, the study variables for each individual heifer included date of birth, certain milking characteristics, whether or not the heifer was purchased, dates of first and second calving, number of calves at first calving, and date of culling (if culled). As part of the breeding program, milking speed and the degree of milk leakage is evaluated early in the first lactation period. Milk leakage is graded as none, slight, or pronounced. Milking speed is characterized as rapid, medium, or slow. Ninety-seven percent of the heifers were of the Norwegian Red Cattle breed; the remaining heifers were various other Norwegian breeds (0.3%), Jerseys (0.2%), or various crossbreeds.

### Herd Data

For each herd, information was available on herd size, mean milk yield, composition of the diet, whether or not the heifers were kept on pasture in summer and autumn, BMSCC, and the annual herd incidence rate of clinical mastitis. Herd size was expressed as the number of cow-years in which each cow was included from the date of first calving until the date of culling. The years of first calving and culling contributed only a fraction of 1 yr as determined by the dates of those events. The mean milk yield of the herd was calculated as kilograms of milk corrected for fat and protein yielded per cow-year. The amounts of the various feedstuffs consumed per year by a herd were recorded routinely for herds in the Production Recording Scheme, and the relative percentage of each feedstuff on an energy basis was calculated. The BMSCC is measured in all Norwegian dairy herds twice per month, and a monthly BMSCC was calculated as the arithmetic mean of those values. In the present study, the annual geometric mean of the monthly BMSCC was used. To calculate the annual herd incidence rate for clinical mastitis, recordings for both acute and chronic clinical mastitis were taken into account. All repeated cases of clinical mastitis in the same cow contributed to the herd incidence rate, provided that the time interval between two consecutive disease recordings was at least 10 d. Apart from the data on the herd incidence rate for clinical mastitis, the BMSCC, and whether or not the heifers were on pasture in summer and autumn, which were compiled in 1993, the remaining herd data were from 1994.

Although cases of mastitis in heifers were recorded in 1994, we decided to use the incidence rate of clinical mastitis and the BMSCC of the preceding year as the parameters of udder health for a herd. This procedure was followed to avoid a situation in which the cases of mastitis in heifers were also represented as

TABLE 1. Description of study variables. For continuous (C) variables, the unit used in the logistic regression models is given.

Variable	Description	Level
HEIFMA	Clinical mastitis in heifer	0 = No; 1 = yes
AGE	Age at first calving	C (30 d)
CMO (1-8, 10-12)	Month of calving (11 design variables; September is reference month)	0 = Did not calve in the given month; 1 = calved in the given month
CANUMB	Number of calves at first calving	0 = 1; 1 = >1
LEAK	Milk leakage	0 = None; 1 = slight or obvious leakage
OUTMIL	Speed with which cows milked out	0 = Rapid or medium; 1 = slow
PURCH	Origin of heifer	0 = Own breed; 1 = purchased
REG	Location of herd	0 = Eastern and southern Norway; 1 = western and northern Norway
HESIZE	Herd size (cow-years)	0 = ≤30 Cow-years; 1 = >30 cow-years
BMSCC <sup>1</sup>	Herd bulk milk SCC (annual geometric mean)	C (10 <sup>5</sup> cells/ml)
HEMAST <sup>1</sup>	Herd incidence rate for clinical mastitis	C (20 Cases/100 cow-years)
HEYIEL	Mean herd milk yield	C (500 kg of Fat- and protein-corrected milk)
CONC	Percentage of concentrates in the diet of the herd	C (10%)
GRASIL	Percentage of grass silage in the diet of the herd	C (10%)
HAY	Percentage of hay in the diet of the herd	0 = ≤5%; 1 = >5%
ROOTS	Percentage of roots in the diet of the herd	0 = 0%; 1 = >0%
FRGRAS	Percentage of fresh grass in the diet of the herd (on pasture or fed indoors)	0 = ≤25%; 1 = >25%
OUT <sup>1</sup>	Heifers on pasture in summer and autumn	0 = No; 1 = yes
HEMASTO <sup>1</sup>	For purchased heifers, incidence rate for clinical mastitis in the original herd	C (20 Cases/100 cow-years)
BMSCCO <sup>1</sup>	For purchased heifers, bulk milk SCC (annual geometric mean) in the original herd	C (10 <sup>5</sup> cells/ml)
OUTO <sup>1</sup>	For purchased heifers, heifers on pasture in summer and autumn in the original herd	0 = No; 1 = yes

<sup>1</sup>Recorded the year before clinical mastitis was detected in the heifer.

cases that constituted the herd incidence rate, and, moreover, that might have indirectly influenced herd incidence rate by being a source for the infection of cows that contributed to the incidence rate. The correlation between annual herd incidence rates for clinical mastitis for 2 consecutive yr was relatively high within Norwegian dairy herds, justifying the use of rates from the previous year as a parameter of herd infection rate. For example, in the present study, when recordings were compared for clinical mastitis in the herds between 1993 and 1994, respectively, the correlation coefficient was 0.52, and the coefficient for the intraherd correlation between the annual geometric means of the BMSCC for those 2 yr was 0.68 (S. Waage, 1997, unpublished data).

Routines for recording and reporting information on the herds have been described in detail (19). A description of the variables used in the study is given in Table 1, and the median and the first and third quartiles for these variables for all dairy herds represented by at least 1 heifer in the present material are shown in Table 2.

### Materials and Study Design

The study was designed as a nested case-control study. All heifers calving in 1994 (n = 128,027) were

eligible for inclusion. Heifers that had been treated for clinical mastitis prior to or on the day of parturition were included as cases (n = 6410). Of the remaining heifers, those treated for clinical mastitis during the following lactation or during the dry period were excluded. Thus, the controls originated from the same cohort as the cases and comprised all

TABLE 2. Median and first and third quartiles for variables that characterized the 21,719 herds from which the heifers that were eligible for this study were obtained.

Variable	Median	First quartile	Third quartile
Herd size, cow-years	12.1	9.2	15.3
Mean milk yield, kg of FPCM <sup>1</sup> /cow-year	6354	5804	6918
Composition of diet, %			
Grass silage	41	35	46
Concentrates	37	33	41
Hay	0	0	2
Fresh grass	16	11	22
Incidence of clinical mastitis, cases/100 cow-years	44	23	71
Geometric mean bulk milk SCC, 10 <sup>3</sup> cells/ml	136	98	183

<sup>1</sup>Milk corrected for fat and protein.

heifers calving in 1994 that were not treated for clinical mastitis before the second calving or, if culled earlier, before culling ( $n = 94,407$ ). The heifers in the material originated from 21,719 different herds. Table 3 shows the distribution of these herds in relation to the numbers of cases and controls recruited from the herd. For some heifers, recordings on some of the variables were missing.

Of the heifers eligible for the study, 9796 were purchased before first parturition. For each of those heifers, the date of purchase and the identity number of the original herd were recorded, making it possible to collect information regarding herd size, herd health, BMSCC, mean milk yield, and composition of the diet for the source herd.

### Statistical Analysis

Univariate associations between the response variable and each of the potential risk factors were studied using univariate logistic regression models. Associations between the response variable and each of the potential risk factors adjusted for other factors were studied by multivariate logistic regression. Variables at  $P < 0.25$  in the univariate analysis were considered for further analysis. A stepwise selection procedure was applied to aid in the process of selecting variables for the final model.

The relationship between each of the continuous independent variables and the logit produced by the multivariate model was examined; data for each variable were divided into 20 groups of equal size based on percentiles of the variable, and the mean of the logit within each group was plotted against the corresponding group means of the variable.

Biologically plausible interaction terms were added to the model, and possibly significant associations were assessed using the likelihood ratio test.

The  $P$  values of the maximum likelihood estimates of the parameters were based on the Wald chi-square statistic. For variables included in the model as only main effect terms, the 95% confidence interval (CI) for the odds ratio (OR) was obtained by exponentiation of the corresponding confidence limits for the estimated regression coefficients. These estimates were based on the likelihood ratio method. For variables included in the model also as quadratic or interaction terms, OR values were estimated for specified levels of the variables involved in those terms, accounting for the correlation between the variables (9). The fit of estimated models was assessed using the Hosmer-Lemeshow goodness of fit test based on the deciles of risk grouping (9). The Hosmer-

TABLE 3. Distribution of the 21,719 herds represented by at least 1 heifer in the study in relation to the number of eligible cases and controls in the herd.

Controls	Cases					
	0	1	2	3	4	>4
0		154	43	8	3	0
1	2018	427	79	14	3	0
2	2784	538	117	22	7	3
3	3063	592	145	32	6	2
4	2672	554	113	24	4	2
5	2026	445	96	25	8	2
6	1457	310	75	21	7	2
7	960	229	57	15	2	4
8	627	146	39	14	1	2
9	412	80	28	9	4	0
10	284	65	20	8	2	1
11	172	34	10	4	2	0
12	99	29	12	4	0	1
>12	287	106	26	11	5	5

Lemeshow goodness of fit statistic ( $C_{HL}$ ) was obtained from the calculation of the Pearson chi-square statistic from the  $2 \times 10$  table of observed and expected frequencies. A coefficient of determination with the maximum value of 1 (adjusted  $R^2$ ) was used to measure the proportion of variation explained by the model (18). The potential correlation among the responses in heifers from the same herd was measured by the intraclass correlation coefficient.

Data were analyzed using SAS software (22). The logistic procedure was used for logistic regression analyses.

### RESULTS

Fifty-six percent of the included cases were treated for clinical mastitis on the day of parturition or the day before, and 90% were treated between 30 d before parturition and the day of parturition.

The association between potential risk factors and the response variable in univariate logistic models is shown in Table 4. The percentage of grass silage in the diet was not significant. However, when the square of the percentage of grass silage was added to the univariate model, both the original (95% CI for OR, 1.19 to 1.61) and the quadratic terms (95% CI for OR, 0.94 to 0.98) of this variable were highly significant ( $P < 0.001$ ), revealing a nonlinear relationship. Therefore, the percentage of grass silage was studied further in the multivariate models. Age and herd incidence rate for clinical mastitis also had highly significant quadratic terms in addition to the significant linear relationship.

Table 5 shows the significant variables in the final multivariate logistic model. The model included two

TABLE 4. Results of univariate analyses of association between clinical mastitis in heifers and potential risk factors, showing point estimates and 95% confidence intervals (CI) for odds ratios (OR).

Variable <sup>1</sup>	no.	OR	95% CI for OR	P
AGE	99,064	1.01	1.00 to 1.03	0.007
CANUMB	100,817	1.30	0.99 to 1.67	0.048
LEAK	73,735	1.35	1.24 to 1.46	<0.001
OUTMIL	73,735	1.14	1.04 to 1.23	0.003
PURCH	100,817	0.90	0.83 to 0.99	0.024
REG	100,817	1.59	1.50 to 1.70	<0.001
HESIZE	99,718	0.73	0.64 to 0.83	<0.001
GRASIL	99,646	1.00	0.98 to 1.03	<0.857
CONC	99,646	1.24	1.20 to 1.29	<0.001
HAY	99,646	0.69	0.63 to 0.75	<0.001
ROOTS	99,646	0.82	0.69 to 0.97	0.021
FRGRAS	99,646	0.87	0.81 to 0.93	<0.001
HEYIEL	99,718	1.12	1.10 to 1.14	<0.001
OUT	98,086	0.87	0.81 to 0.92	<0.001
BMSCC	98,148	0.79	0.76 to 0.82	<0.001
HEMAST	98,148	1.26	1.24 to 1.28	<0.001

<sup>1</sup>See Table 1 for description of variables.

quadratic terms and three interaction terms. Month of calving was included using 11 design variables; September, which had the smallest estimated coefficient, was the reference. The adjusted R<sup>2</sup> for the

model was 0.05. According to C<sub>HL</sub>, the model fitted well to the observed data (C<sub>HL</sub> = 10.29; df = 8; P = 0.25). When added to the final model, origin of heifer (P = 0.66), the percentage of concentrates in the diet (P = 0.32), and the number of calves (P = 0.056) were not significant. The frequency of twin or triplet births for the heifers was 0.76%.

The heifers included in the final model were from 19,231 herds, and the mean number of heifers per herd was 3.7. The intraherd correlation coefficient for the response of the heifers was 0.086.

In Figure 1, the logit of the final model is plotted against each of the continuous variables included in the model using group means based on grouping according to percentiles. Mean herd milk yield and BMSCC were approximately linear in the logit. Herd incidence rate for clinical mastitis and age were non-linear in the logit, and, for both variables, the quadratic term was significant in the final model.

Whether or not the heifers were on pasture was a significant factor (Table 5). Analyses were performed to examine whether significance could be found both for heifers calving during the potential pasturing season and for those calving during the indoor period. No

TABLE 5. Significant risk factors for clinical mastitis in heifers according to the final multivariate logistic regression model comprising 71,328 heifers; the coefficient (b), standard error of the coefficient (SE<sub>b</sub>), odds ratio (OR), 95% confidence interval (CI) for the OR, and significance level are given for each variable.

Variable <sup>1</sup>	b	SE <sub>b</sub>	OR	95% CI for OR	P
HEMAST	0.3591	0.0236	1.432	1.368 to 1.501	<0.0001
(HEMAST) <sup>2</sup>	-0.0186	0.0028	0.982	0.976 to 0.987	<0.0001
BMSCC	-0.1758	0.0247	0.841	0.801 to 0.882	<0.0001
AGE	0.2419	0.0758	1.286	1.110 to 1.494	0.0009
(AGE) <sup>2</sup>	-0.0041	0.0014	0.996	0.993 to 0.998	0.0023
LEAK	0.3176	0.0438	1.361	1.249 to 1.482	<0.0001
OUTMIL	0.1304	0.0447	1.140	1.044 to 1.244	0.0033
REG	0.8688	0.1755	2.355	1.673 to 3.327	<0.0001
HESIZE	-0.7229	0.2071	0.485	0.315 to 0.712	0.0005
GRASIL	0.0802	0.0360	1.064	0.992 to 1.141	0.0838
HAY	-0.0116	0.0800	0.987	0.843 to 1.152	0.8659
HEYIEL	0.0752	0.0108	1.072	1.049 to 1.094	<0.0001
OUT	-0.1522	0.0420	0.839	0.773 to 0.910	0.0001
REG × HESIZE	0.7576	0.2286	2.101	1.366 to 3.357	0.0012
REG × HAY	-0.5228	0.1549	0.600	0.440 to 0.807	0.0010
REG × GRASIL	-0.1116	0.0414	0.897	0.827 to 0.973	0.0090
CMO1	0.2358	0.0777	1.266	1.086 to 1.472	0.0024
CMO2	0.1646	0.0800	1.179	1.006 to 1.377	0.0397
CMO3	0.2164	0.0798	1.242	1.060 to 1.450	0.0067
CMO4	0.3550	0.0861	1.426	1.202 to 1.685	0.0001
CMO5	0.5634	0.0806	1.757	1.498 to 2.055	<0.0001
CMO6	0.2790	0.0815	1.322	1.125 to 1.548	0.0006
CMO7	0.3967	0.0631	1.487	1.314 to 1.682	<0.0001
CMO8	0.3254	0.0581	1.385	1.236 to 1.552	<0.0001
CMO10	0.1099	0.0647	1.116	0.983 to 1.267	0.0895
CMO11	0.1716	0.0735	1.187	1.027 to 1.370	0.0195
CMO12	0.1683	0.0761	1.183	1.018 to 1.372	0.0269

<sup>1</sup>See Table 1 for description of variables.

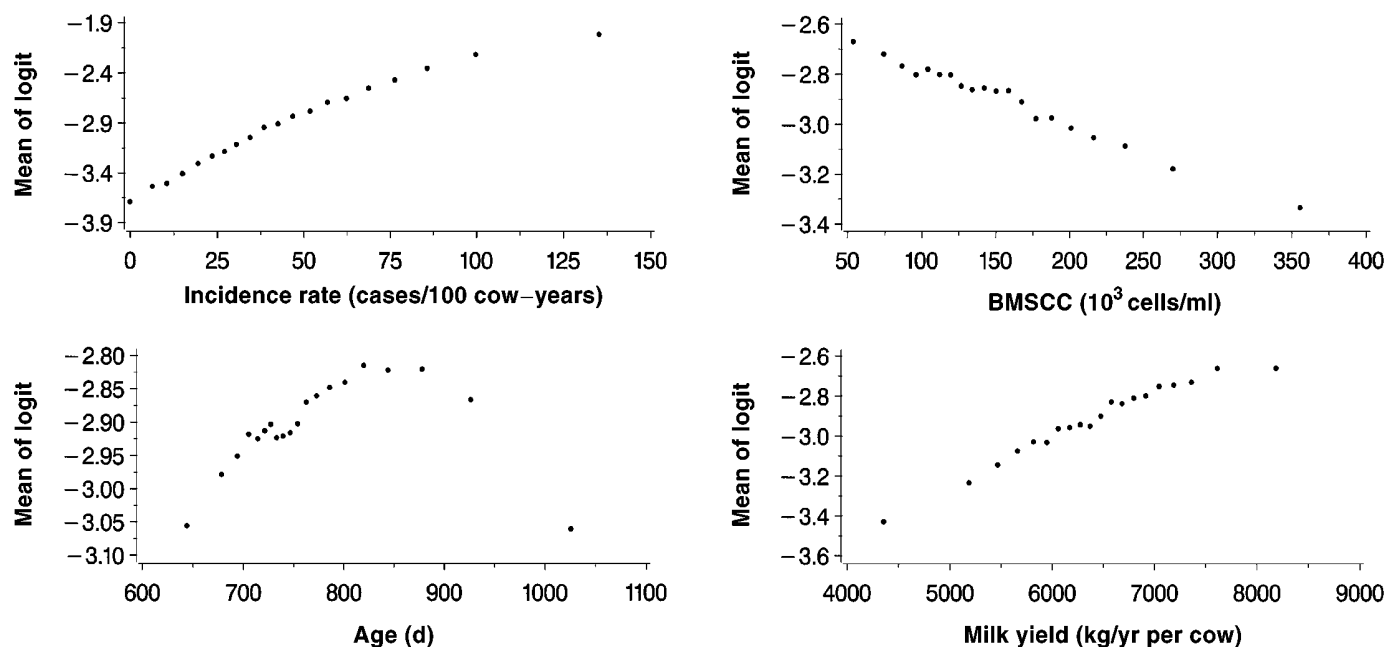


Figure 1. Relationships between the continuous variables included in the final logistic regression model and the logit estimated from the model. For each continuous variable, the observations were divided into 20 groups of approximately equal size based on percentiles, and the group means were plotted against the corresponding means of the logit. Variables were herd incidence rate for clinical mastitis, age of the heifer at calving, mean herd bulk milk SCC (BMSCC) (annual geometric mean), and mean herd milk yield per cow.

association ( $P = 0.23$ ) was found when calving took place in the period from October to May but an association ( $P < 0.0001$ ) was present when heifers calved between June and September (95% CI for OR; 0.691 to 0.877).

Substituting for herd incidence rate for clinical mastitis and BMSCC in the model, the corresponding values of these variables for the subsequent year (i.e., the same year as the clinical cases of heifer mastitis were recorded) did not alter the collection of significant variables contained in the model, and, except for herd incidence rate for clinical mastitis, only minor changes occurred in the estimated coefficients for the

variables. However, the adjusted  $R^2$  for the model increased to 0.10.

Heifers that had been purchased before first calving were studied separately. Variables from the original herd as well as the current herd were included as potential risk factors. The median of the time interval from the date of purchase to the date of calving was 37 d, and the first and third quartiles were 17 and 461 d, respectively. The final multivariate model for purchased heifers included 4099 heifers. Significant variables are shown in Table 6. The  $C_{HL}$  was not significant ( $C_{HL} = 4.41$ ;  $df = 8$ ;  $P = 0.82$ ), indicating acceptable fit of the model. The adjusted  $R^2$  was 0.05.

TABLE 6. Significant risk factors for clinical mastitis in 4099 purchased heifers based on the final multivariate logistic regression model; the coefficient (b), standard error of the coefficient ( $SE_b$ ), odds ratio (OR), 95% confidence interval (CI) for the OR, and significance level are given for each variable.

Variable <sup>1</sup>	b	$SE_b$	OR	95% CI for OR	P
HEMAST	0.1575	0.0312	1.171	1.100 to 1.243	<0.0001
BMSCC	-0.2502	0.0973	0.779	0.641 to 0.938	0.0101
OUTO	-0.4275	0.1722	0.652	0.469 to 0.923	0.0131
HEMASTO	0.0803	0.0356	1.084	1.009 to 1.160	0.0240
CANUMB	1.1367	0.5052	3.117	1.027 to 7.759	0.0244
CMO5 <sup>2</sup>	1.2301	0.4295	3.421	1.529 to 8.418	0.0042

<sup>1</sup>See Table 1 for description of variables.

<sup>2</sup>For all other calving months, the coefficients were not significant.

To examine whether the influence from the original herd was dependent on the interval between the dates of purchase and calving, the purchased heifers were divided into two groups, those purchased between 28 d prior to parturition and the day of parturition and those purchased > 28 d prior to parturition. The two groups were analyzed separately, allowing the significant variables for all purchased heifers (Table 6) to enter the model. In the former group, the final model identified herd incidence rate for clinical mastitis in both the current herd (95% CI for OR, 1.135 to 1.328;  $P < 0.0001$ ) and the original herd (95% CI for OR, 1.033 to 1.232;  $P = 0.006$ ) as significant risk factors. In the latter group, herd incidence rate for clinical mastitis in current herd was significant (95% CI for OR, 1.055 to 1.210;  $P = 0.0004$ ), and herd incidence rate for clinical mastitis in the original herd was not ( $P = 0.13$ ). Both models also contained other significant variables (data not shown).

## DISCUSSION

The present study identified several risk factors for clinical mastitis in heifers involving herd effects, effects related to individual characteristics, and a regional effect. A significant association was demonstrated between the risk of mastitis for heifers and the total incidence rate for clinical mastitis in the corresponding herd. In the final model, both the incidence rate and the quadratic term of this variable were significant, proving the existence of a nonlinear component in the relationship between the incidence rate and the logit. As incidence increased, the OR for a given difference in the incidence rate decreased gradually.

A high overall herd incidence rate for clinical mastitis may reflect the presence of a prevalent reservoir of contagious udder pathogens in the herd and, thus, increased risk of exposure of the heifers to such pathogens. Because of a national program for the control of mastitis caused by *Streptococcus agalactiae*, which has been in operation for several decades, cases of bovine mastitis caused by *Strep. agalactiae* are extremely rare in Norway. However, other contagious organisms, primarily *Staph. aureus* and *Strep. dysgalactiae*, are important causes of clinical mastitis both in cows and nulliparous heifers (31), which is in agreement with the situation in other countries (10). A relationship between the herd incidence rate for clinical mastitis and the rate for clinical mastitis in heifers or primiparous cows has been reported previously (17). However, in a study on IMI caused by *Staph. aureus*, the proportion of heifers infected at

parturition did not differ significantly between herds with high or low prevalence of IMI caused by *Staph. aureus*, respectively (20).

Demonstration of a significant association between clinical mastitis in purchased heifers and the previous overall incidence of clinical mastitis in the herds from which they originated clearly emphasizes the contagious aspects of the disease. However, for heifers arriving in the new herd >4 wk prior to parturition, the risk for clinical mastitis was not associated with the overall incidence rate for clinical mastitis in the original herd. Observations made by Schalm (23) indicated that the juvenile mammary gland may become infected as a consequence of the calves suckling each other after being fed milk containing *Strep. agalactiae*. This action could subsequently lead to mastitis in heifers. A similar pathogenesis for mastitis caused by *Staph. aureus* in heifers has been suggested (14). The present findings, however, seem to indicate that a clinical attack of mastitis in heifers is primarily a result of a recent infection and not a flare-up of a persistent subclinical infection acquired several weeks or months earlier.

A decrease in BMSCC was associated with an increased risk for clinical mastitis in heifers. The BMSCC was linear in the logit, and the OR that corresponded to an increase of  $10^5$  cells/ml in the annual geometric mean was 0.84. Increased risk for clinical mastitis in heifers and lactating cows in herds with a low BMSCC has been reported by others (5, 17), but, in a study on summer mastitis (8), increased risk was associated with a high BMSCC. Normally, low BMSCC in a herd is closely related to low individual SCC, indicating a low prevalence of subclinical udder infections. Lam et al. (12) have reported that quarters that harbor minor pathogens were less susceptible to new infections by major pathogens than were uninfected quarters, a phenomenon that was possibly related to the protective effect of the cell response triggered by the minor pathogens.

The BMSCC reflects the status of lactating cows. Apart from factors related to milking, heifers and cows in a herd are probably more or less exposed to the same environmental factors. Thus, one might hypothesize that BMSCC is also, to some extent, an indicator of the degree of exposure to various organisms of the udder of the heifers in the herd.

When the herd incidence rate for clinical mastitis and the BMSCC for 1994 were used in the analysis instead of the corresponding recordings for 1993, the proportion of the total variation in the data explained by the final logistic regression model increased. However, the variables that were identified as significant risk factors were the same in both models.

A linear relationship existed between the mean herd milk yield per cow and the logit. An increased rate of clinical mastitis in heifers and cows in herds with a high mean milk yield has been found in other studies (17, 25, 29, 30), and a genetic correlation between milk yield and clinical mastitis has been reported (30). For lactating cows, increased susceptibility to mastitis may be partly related to a high milk yield per se. In the present study, the association between clinical mastitis and the mean herd milk yield must have been due to the common correlation of both variables with certain herd management or nutritional factors not included in the model, or increased genetic disposition to mastitis of heifers in high yielding herds.

Region was included in the analysis as a dichotomous variable created by dividing the country into two parts. Although considerable variations in climate and agricultural conditions are present within each of those regions, there are also some common features. Large parts of western and northern Norway are coastal areas with precipitation higher than that in the eastern and southern districts. In general, summer mastitis in heifers occurs more frequently in the coastal areas of Norway, possibly explaining, to some extent, why a greater risk for clinical mastitis in heifers was observed in the western and northern parts. For the country as a whole, as in Sweden (10), the proportion of all cases of prepartum clinical mastitis caused by *A. pyogenes* in heifers is approximately 10% (31).

Heifers in herds that were kept on pasture in summer and autumn were at a lower risk for clinical mastitis than were heifers in nongrazing herds. However, this difference was present only for heifers calving during the pasture season, which, depending on the geographic location of the herd, lasts from May or June to September. A protective influence from pasture was also obtained in the analysis of purchased heifers. Heifers originating from farms that allowed the herd to graze were at reduced risk for clinical mastitis compared with heifers from herds that were not allowed to graze. Reduced risk for clinical mastitis that is associated with pasture has also been reported by others (2, 24).

Heifers calving late in spring or during summer were at higher risk for clinical mastitis than were those calving at other times of the year. Because pasture was associated with reduced risk, the increased risk in spring and summer could not be related to the pasture per se. In a study including both primiparous and multiparous cows, the incidence rate for clinical mastitis peaked in spring (30). The

incidence of IMI before parturition has been considerably higher during warm than during cool weather (26). In another study, however, the lowest rate for clinical mastitis was observed in cows calving during spring or summer (29).

The risk for clinical mastitis in heifers was influenced by the proportions of hay and grass silage in the diet. However, the influence of these variables was dependent on region. Although hay had no apparent influence on heifers in eastern and southern Norway, heifers in western and northern Norway were at reduced risk for clinical mastitis when hay supplied >5% of the energy content of the total annual diet. An increase in the percentage of grass silage was associated with a slightly decreased risk for mastitis in heifers in western and northern Norway and a slightly increased risk for heifers in eastern and southern Norway.

In agreement with the results of other studies (25), milk leakage was associated with an increased risk for mastitis. Furthermore, heifers subsequently found to have a low milk flow rate were more susceptible to clinical mastitis prepartum than were heifers with a medium or high flow rate. The milking characteristics of the heifers were assessed during early lactation, and a previous attack of clinical mastitis might have influenced the flow rate of the affected quarter. Thelitis is a common complication of mastitis in heifers (27) and often leads to a narrow and rigid teat canal, thereby altering the flow rate. In theory, this complication might have influenced the overall judgment about milking speed because the personnel assessing the milking characteristics are instructed to pay attention to the milkability of single teats (H. Solbu, 1997, personal communication). Some controversy exists in the literature about the relationship between milking speed and udder health. Some researchers found increased SCC (28) or increased infection rate (3) in fast milking cows, but others (15) found the lowest infection rate in cows with the highest milk flow rate. Based on the milking speed index of the sires, heifers with clinical mastitis were genetically slower milkers than were heifers that did not have mastitis (17). Apparently, an increase in milk flow is not necessarily associated with reduced barrier function of the teat to potential invading organisms, provided there is no milk leakage.

Similar to results of a previous study (4), an increase in age at parturition was associated with an increased risk for clinical mastitis in heifers. The relationship between age and the logit was nonlinear (i.e., the OR associated with a 30-d increase in age declined as age increased), and heifers calving at an

extraordinarily high age were at decreased risk for clinical mastitis. It has been demonstrated that yield from the subsequent lactation increased as age at first calving increased (11), and yield is evidently related to the degree of development of the udder at calving. A relationship between the susceptibility of heifers to clinical mastitis and the degree of udder development is comprehensible.

Approximately 6% of the heifers in the present study were purchased before calving. Although the time of purchase in relation to time of calving varied a great deal, 25% of these heifers were purchased during the last 17 d prior to calving. Pregnant heifers moving to a new environment shortly before parturition might be expected to be more susceptible to udder infection than would those maintained in a single environment. However, whether the heifer was purchased or not was not a significant factor in the final model.

Overall analysis indicated that whether a heifer gave birth to 1 or >1 calf did not influence the risk for clinical mastitis in heifers markedly. However, in a separate analysis of purchased heifers, heifers with >1 calf were at increased risk compared with those with 1 calf. Fewer than 1% of the heifers had >1 calf, probably explaining the wide 95% CI for the OR.

The correlation among responses of heifers from the same herd was low, and the mean number of heifers per herd was small. Thus, herd clustering was considered unimportant and was ignored in the final analysis of the data.

Although several risk factors for clinical mastitis in heifers were identified in the current study, only a small amount of the total variation in the dependent variable was explained by these factors. Obviously, some important risk factors were not included at all, or some of those included were not recorded in sufficient detail. For example, feedstuffs given as percentages of the total diet in the herd were the only variables that described nutrition, and seasonal variations in the diet and the amounts of various feedstuffs supplied in relation to the optimal needs of the heifers were not taken into account. More detailed recordings of environmental, management, and animal factors should be carried out to elucidate the relationships more precisely, a prerequisite for the recommendation of specific preventive measures.

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