

Risk Factors for Clinical Mastitis in a Random Sample of Dairy Herds from the Southern Part of The Netherlands

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ABSTRACT

The incidence of clinical mastitis in dairy cows was estimated in 171 randomly selected dairy herds from the southern part of The Netherlands. A total of 1103 quarter cases was reported. The mean annual incidence rate was 12.7 quarter cases/yr per 100 cows. The modeling incidence rate of clinical mastitis at the herd level indicated that a number of risk factors were associated with a higher rate of clinical mastitis: one or more cows that were leaking milk, one or more cows with trampled teats, no disinfection of the maternity area after calving, consistent use of post-milking teat disinfection, Red and White cattle (Meuse-Rhine-Yssel) as the predominant breed, and an annual bulk milk somatic cell count <150,000 cells/ml.

The following risk factors were associated with a higher rate of clinical mastitis caused by *Escherichia coli*: cows with trampled teats, no disinfection of the maternity area after calving, consistent use of post-milking teat disinfection, use of a thick layer of bedding in the stall, and the stripping of foremilk before cluster attachment.

The following risk factors were associated with a higher rate of clinical mastitis caused by *Staphylococcus aureus*: Red and White cattle (Meuse-Rhine-Yssel) as the predominant breed, cows with trampled teats, the stripping of foremilk before cluster attachment, no regular disinfection of the stall, no regular replacement of stall bedding, and an annual bulk milk somatic cell count <150,000 cells/ml.

(**Key words:** clinical mastitis, risk factors, *Escherichia coli*, *Staphylococcus aureus*)

INTRODUCTION

Among others, the Animal Health Service in the southern part of The Netherlands in cooperation with the dairy industry developed a mastitis control program for subclinical mastitis in 1984. The purpose of the program was to prevent new IMI through a program for milking time hygiene (7) and through the elimination of existing IMI via dry cow antibiotic therapy and culling (30). As a result, the mean annual bulk milk SCC dropped from 357,000 cells/ml in 1985 to 220,000 cells/ml in 1995 in the southern part of The Netherlands.

Reports from a number of countries (9, 11, 12, 14, 17) indicated that, despite the decrease in the bulk milk SCC, the classic control program either had no effect or adversely affected the incidence rate of clinical mastitis. A Dutch study (27) involving 125 herds with a mean bulk milk SCC <150,000 cells/ml reported an incidence rate of clinical mastitis of 17.9 cases/yr per 100 cows. In a previous report on our data (22), an incidence rate of 12.7 cases/yr per 100 cows was estimated; a tendency also existed for more cases to be detected in herds with a lower bulk milk SCC.

Control programs for subclinical mastitis have proven to be effective. However, control programs for clinical mastitis are currently unavailable. An important reason for this lack of availability is the inadequate understanding of risk factors for clinical mastitis. Only a few studies have been conducted that have specifically examined the risk factors for clinical mastitis (28, 32). The major difference in this study compared with the studies of Schukken et al. (28, 29) is the random sampling of the dairy population from the southern part of The Netherlands; therefore, no specific bulk milk SCC cohorts were selected. Furthermore, because the last estimate of the incidence of clinical mastitis was from 1990, an up-to-date estimate of the incidence of clinical mastitis was neces-

Received April 22, 1997.

Accepted September 26, 1997.

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sary. Risk factors represent areas of interest for the prevention and control of disease. The objective of this study was to identify risk factors for clinical mastitis (e.g., housing, hygiene, milking management, and milking machine).

MATERIALS AND METHODS

Study Design and Population

The study design and population have been described previously (22). Briefly, 201 herds were randomly chosen from Northern Brabant and Limburg in the southern part of The Netherlands. Only herds with ≥ 30 dairy cows were selected. The final study base consisted of 171 herds; mean herd size was 52.6 lactating and dry cows, culminating in a total of 8995 cows. Ninety-one percent of the farms had a loose housing system, and 9% used tie stalls. Eighty-five percent of the farmers employed a milk recording system.

Clinical Mastitis

Between the summer of 1992 and 1993, each farmer was asked to collect a milk sample aseptically from every quarter with clinical mastitis. Clinical mastitis was identified by the individual dairy farmers on the basis of clinical signs including abnormal milk, a hard or swollen udder, or both. Sampling methods were discussed with the farmer prior to the initiation of the study. The farmer not only recorded cases of clinical mastitis but also noted the identification number of the cow, the last calving date, affected quarters, parity, breed, stage of lactation, and clinical signs of the cow. Samples collected from quarters with clinical mastitis were frozen on the farm (generally at -20°C). Herds were visited every 6 wk for milk sample and data collection. Milk samples were cultured bacteriologically at the lab of the Animal Health Service in the southern part of The Netherlands. Isolated bacteria were identified according to the standards of the National Mastitis Council (13).

Risk Factors

Six clusters of risk factors were deemed to have a potential relationship with the incidence of mastitis: production indexes, housing, hygiene, health disorders, milking management, and milking machine. Details on these variables were obtained by interviews that were conducted using a questionnaire. The interviews were completed in the summer and were repeated at the end of the year to check for con-

TABLE 1. Overview of clusters of variables for the risk analysis of clinical mastitis.

Variable	Description
Basic and production indexes	Breed, standard herd production, herd size, annual milk production, annual bulk milk SCC.
Housing	Width and length of resting area, floor characteristics, volume of the barn, amount of straw in stall bedding, type of housing system.
Hygiene	Hygiene of resting area and stall, frequency of bedding replacement, disinfection of maternity area after calving, disinfection of the stall, milking cows with mastitis separately, separate housing of young stock.
Health	Presence of ≥ 1 cow leaking milk, presence of ≥ 1 cow with trampled teats, presence of ≥ 1 cow with dirty teats, presence of ≥ 1 cow resting on the slatted floor, prevention of mastitis during summer, use of antibiotics at dry-off.
Milking management	Stripping of foremilk before cluster attachment, complete milking out, use of postmilking teat disinfection, use of udder towels, direct attachment of milking claw after pretreatment.
Milking machine	Pulsation frequency, type of pulsation, frequency of replacement of teat cups of the milking claw, height of the milking glass compared with the position of the milking claw, diameter of milking pipeline, vacuum of milking machine, cleaning of milking claw between cows, automatic detachment of milking claw, support of milking outlet pipe, frequency of maintenance on milking machine.

sistency. A high correlation ($r = 0.80$) existed between the answers given in the two interviews, and only consistent answers were included in the analyses. A general overview of the type of variables in the clusters is presented in Table 1. A full list of all variables is available from the authors upon request.

Statistical Analysis

Data were modeled as the incidence rate of clinical quarter cases per herd. To account for quarters that exhibited clinical signs multiple times during the study, a clinical case was defined to have a duration of 2 wk (32). The number of clinical quarter cases was divided by the total number of cow-weeks at risk. Because Schukken et al. (30) previously showed that the rate of clinical mastitis follows a Poisson distribution with overdispersion, a Poisson regression (PROC GENMOD) was performed according to SAS (26): $\ln(\text{clinical cases}/\text{cow-weeks at risk})_j = a + b_i \text{ risk}$

factor_i + error, where $\ln(\text{clinical cases})_j$ is the natural log of the number of clinical cases in herd j ; b_i is the regression coefficient of risk factor i ; $\ln(\text{cow-weeks at risk})_j$ is the natural log of the number of cow-weeks at risk in herd j , and error is a Poisson-distributed residual term. A scale parameter, estimated by the deviance divided by its degrees of freedom, was used to account for overdispersion in the model. The significance of the parameters was measured by the likelihood ratio test. Estimated regression coefficients (RC) of the multivariate Poisson regression model may be expressed exponentially (e^{RC}) and interpreted as relative risk or risk ratio (5). The first step in the statistical analysis involved screening of all single explanatory variables in a bivariate Poisson regression model. Variables with a $P < 0.25$ were considered for further analysis. In the second step, a backward stepwise selection of variables was performed in a multivariate model to analyze variables simultaneously. Two-way interactions with a biologically meaningful interpretation were tested between main effects that remained in the model. Statistical significance in the final model was assumed to have a P value ≤ 0.05 in the likelihood ratio test. The most frequent isolates from clinical cases were *Escherichia coli* (16.9%) and *Staphylococcus aureus* (14.4%) (22). The incidence rate of clinical mastitis was therefore modeled for all cases of clinical mastitis and was modeled separately for clinical mastitis caused by *E. coli* and *Staph. aureus*.

Because of missing values, 144 herds with complete data on risk factors contributed to the final model. The goodness of fit of the final model was assessed by residual analysis.

RESULTS

During the study period, 15 farmers terminated participation for various reasons (e.g., stopped farming, sold the farm, other personal reasons), and 15 farmers had problems with accurate data collection. A total of 1103 quarter cases was reported. Forty-two cow quarters had one recurrent case of clinical mastitis in the same quarter >2 wk apart: 5 cow quarters had two such recurrent cases. The estimated incidence rate of clinical mastitis in dairy cows from the southern part of The Netherlands was 12.7 quarter cases/yr per 100 cows. Herd distribution of the incidence rate of clinical mastitis is shown in Figure 1. Approximately 10% of the herds had an incidence rate of 0 quarter cases/yr per 100 cows (no reported cases), 42% of the herds had an incidence rate of 1 to 10 quarter cases/yr per 100 cows, and 2.3% of the herds had an incidence rate >40 quarter cases/yr per

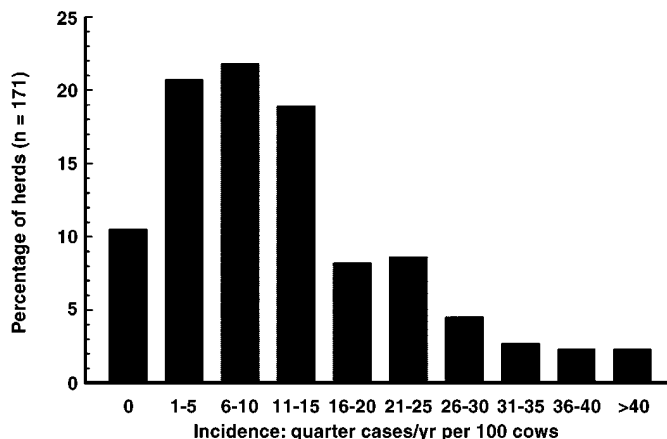


Figure 1. Incidence of clinical mastitis in dairy cows in a random sample of 171 dairy herds in the southern part of The Netherlands.

100 cows. The highest incidence rate observed was 97 quarter cases/yr per 100 cows in one herd.

A total of six risk factors were significantly associated with the incidence rate of clinical mastitis (Table 2). Herds with Holstein-Friesian cows as the predominant breed had a lower rate of clinical mastitis than did herds with Red and White (Meuse-Rhine-Yssel) cows as the main breed. Herds with ≥ 1 cow with trampled teats had a higher rate of clinical mastitis than did herds that did not have any cows with trampled teats. Herds with cows that were leaking milk had a higher rate of clinical mastitis than did herds without cows that were leaking milk. No disinfection of the maternity area after calving was associated with a higher rate of clinical mastitis than was associated with disinfection of the area always or sometimes. For those herds for which postmilking teat disinfection was always practiced, a 1.4 times higher rate of clinical mastitis was reported than that for herds for which postmilking teat disinfection was never or sometimes practiced.

The rate of clinical mastitis was not significantly different between herds with an annual bulk milk SCC between 150,000 and 250,000 cells/ml and herds with an annual bulk milk SCC $>250,000$ cells/ml. However, the rate of clinical mastitis was higher for herds with an annual bulk milk SCC $<150,000$ cells/ml than for herds with an annual bulk milk SCC $\geq 150,000$ cells/ml ($P < 0.05$).

The following risk factors were associated with a higher rate of clinical mastitis caused by *E. coli* (Table 3): cows with trampled teats, no disinfection of the maternity area after calving, consistent use of postmilking teat disinfection, use of a thick layer of stall bedding, and the stripping of milk before cluster

attachment. With respect to clinical mastitis caused by *Staph. aureus*, the following risk factors were significantly associated with a higher incidence rate (Table 4): Red and White cows (Meuse-Rhine-Yssel) as the main breed, cows with trampled teats, the stripping of milk before cluster attachment, no regular disinfection of stalls, no regular replacement of stall bedding, and a low annual bulk milk SCC (<150,000 cells/ml). No interactions appeared to be significant ($P > 0.05$) in any of the three regression models.

DISCUSSION

In our survey, clinical mastitis was identified by the individual dairy farmer on the basis of clinical signs including abnormal milk, or a hard or swollen udder, or both. All farmers received clear instructions on the definition of a case of clinical mastitis prior to the start of the study. Despite the instructions, several factors can cause differences in the identification of cases of clinical mastitis among farmers. The motivation of the farmer, the skill of the farmer to recognize a clinical case, and differences in management practices (i.e., stripping the foremilk) can influence the identification of cases of clinical mastitis.

TABLE 2. Summary of relative risks or rate ratios (RR)¹ and accessory 95% confidence intervals of quarter cases of clinical mastitis per cow-weeks at risk in 144 dairy herds from the southern part of The Netherlands. Data were adjusted for herd size.

Characteristic	Herds	RR	95% CI
	(no.)		
Breed			
Holstein Friesian	72	0.7	0.5–0.9
Red and White ²	72	1.0 ³	
Cows leaking milk			
≥1 Cow present	70	1.8	1.3–2.4
No cows present	74	1.0 ³	
Cows with trampled teats			
≥1 Cow present	97	1.5	1.1–2.1
No cows present	47	1.0 ³	
Disinfection of maternity area after calving			
Never	64	1.4	1.1–1.9
Sometimes or always	80	1.0 ³	
Postmilking teat disinfection			
Always	59	1.4	1.1–1.9
Never or sometimes	85	1.0 ³	
Annual bulk milk SCC, cells/ml			
<150,000	16	1.6	1.1–2.2
150,000–250,000	74	1.0 ³	
>250,000	54	0.9	0.7–1.2

¹Rate ratio estimates of the final multivariate Poisson regression model.

²Meuse-Rhine-Yssel.

³Reference category.

TABLE 3. Summary of relative risks or rate ratios (RR)¹ and accessory 95% confidence intervals of quarter cases of clinical mastitis caused by *Escherichia coli* per cow-weeks at risk in 144 dairy herds from the southern part of The Netherlands. Data were adjusted for herd size.

Characteristic	Herds	RR	95% CI
	(no.)		
Cows with trampled teat			
≥1 Cow present	97	2.1	1.3–3.7
No cows present	47	1.0 ²	
Disinfection of maternity area after calving			
Never	64	1.6	1.1–2.5
Sometimes or always	80	1.0 ²	
Use of stall bedding			
Thin layer	107	0.6	0.4–0.9
Thick layer	37	1.0 ²	
Stripping of foremilk before cluster attachment			
Yes	83	1.6	1.1–2.4
No	61	1.0 ²	
Postmilking teat disinfection			
Always	59	2.9	1.9–4.4
Never or sometimes	85	1.0 ²	

¹Rate ratio estimates of the final multivariate Poisson regression model.

²Reference category.

In our study, no significant difference was detected in the mean incidence of all cases of clinical mastitis between herds that did or did not strip foremilk before cluster attachment.

Possible biases in our study were the underreporting of cases because of poor motivation and the overreporting of cases because farmers might have been interested in bacteriological results that were free of charge. An excess in the reporting of subclinical cases as a result of free bacteriological examination is difficult to verify. We estimated the influence of this factor to be fairly small because we checked the reported clinical signs, color, and consistency of milk samples and the date and number of clinical mastitis samples submitted.

Several risk factors were associated with a higher incidence rate of clinical mastitis. A number of these risk indicators have been reported in other studies. For example, breed [Red and White (Meuse-Rhine-Yssel) vs. Holstein-Friesian] has been shown to be a risk factor (28, 29). In the same study (28), leaking milk was also a risk factor for clinical mastitis. Several studies showed teat lesions to be an important risk factor (1, 33). Interestingly, teat disinfection appeared to increase the risk of clinical mastitis. More than 90% of postmilking teat disinfectants used in our study were iodophors. A recent study (20) also

showed a similar result; specifically, mastitis caused by *E. coli* was increased when teat disinfection was practiced. The highest incidence of clinical mastitis was found around calving and in early lactation (22). Resistance of the cows is low during these periods (18); therefore, factors that increase bacteria load (i.e., poor hygiene of the calving pen) lead to IMI and sometimes result in clinical mastitis, which is especially true for environmental pathogens like *E. coli* (32). Bartlett et al. (2) showed that the use of a separate calving unit as a hygienic measurement was associated with a lower incidence of clinical mastitis.

A lower bulk milk SCC was associated with more clinical mastitis. This result has been suggested previously (10), but no population-based data have been presented until this study. Apparently, cows in herds with a lower bulk milk SCC are at higher risk for clinical mastitis, even after correction for breed, teat lesions, leaking milk, disinfection of the maternity area, and teat disinfection. Milk from cows with clinical mastitis is withheld from the bulk tank by

regulation. Farmers who are better at diagnosing clinical mastitis cases might divert more of the milk with high SCC from the tank, resulting in higher clinical mastitis rates but lower bulk milk SCC. In an earlier report on our data (22), results indicated a lack of evidence for differences in the diagnostic capabilities of farmers (ability to detect visual properties and clinical signs) for farmers of herds with high or low incidences of clinical mastitis and low (< 150,000 cells/ml) or high (>250,000 cells/ml) bulk milk SCC. This result is in accordance with the findings of Lam et al. (19) for farmers of herds with low bulk milk SCC. Further analysis of the data showed the presence ($P = 0.08$) of an interaction between teat disinfection and bulk milk SCC. In herds with a high bulk milk SCC, teat disinfection was not associated with an increase in clinical mastitis. This result was according to expectations because teat disinfection especially appears to be associated with clinical mastitis caused by *E. coli* (Table 3). Herds with a high bulk milk SCC appear to have more cases of mastitis caused by *Staph. aureus* and *Streptococcus agalactiae* (11, 22).

Specific risk factors for the incidence of mastitis caused by *E. coli* and *Staph. aureus* were observed. Generally, mastitis caused by *E. coli* is associated with environmental factors and cow immunity, and *Staph. aureus* is considered to be a contagious pathogen. In our data, cases of mastitis caused by both *Staph. aureus* and *E. coli* were associated with environmental factors (i.e., use of disinfection procedures, replacement of bedding, and hygienic status of stalls).

The stall and its bedding are an important part of the environment of the dairy cow. Bedding may play a key role in the transmission of environmental pathogens to the udder because teats are in close contact with bedding and because the bacteria in bedding survive for prolonged periods (6, 34).

In several studies (3, 4, 8, 16, 24, 28), the relationships among stall cleanliness, log colony-forming units in the bedding, and the incidence of clinical mastitis were observed. The cleanliness of the stalls depends, of course, on the frequency with which stalls are cleaned (8). If a thick layer of bedding is used, the entire bedding will be less often replaced than if a thin layer is used; when the humidity and temperature are high, coliforms multiply rapidly in bedding material (31). Furthermore, *Staph. aureus* can be cultured from bedding, although not as frequently as coagulase-negative staphylococci (15, 21, 23).

Mastitis caused by *E. coli* was specifically associated with teat disinfection, and mastitis caused by *Staph. aureus* was not. This result is comparable with

TABLE 4. Summary of relative risks or rate ratios (RR)¹ and accessory 95% confidence intervals of quarter cases of clinical mastitis caused by *Staphylococcus aureus* per cow-weeks at risk in 144 dairy herds from the southern part of The Netherlands. Data were adjusted for herd size.

Characteristic	Herds (no.)	RR	95% CI
Breed			
Holstein-Friesian	72	0.7	0.4–1.0
Red and White ²	72	1.0 ³	
Cows with trampled teats			
≥1 Cow present	97	2.1	1.2–4.0
Number cows present	47	1.0 ³	
Stripping of foremilk before cluster attachment			
Yes	83	1.7	1.1–2.8
No	61	1.0 ³	1.1–2.8
Regular disinfection of stall			
No	102	2.1	1.2–3.8
Yes	42	1.0 ³	
Hygienic status of stalls of dry cows			
Dirty	36	1.9	1.2–3.0
Clean	108	1.0 ³	
Replacement of stall bedding			
<1/wk	18	1.0 ³	
1/wk	59	0.5	0.3–0.9
Almost every day	67	0.7	0.4–1.2
Annual bulk milk SCC, cells/ml			
<150,000	16	3.2	1.8–5.5
150,000–250,000	74	1.0 ³	
>250,000	54	0.9	0.5–1.5

¹Rate ratio estimates of the final multivariate Poisson regression model.

²Meuse-Rhine-Yssel.

³Reference category.

data from Schukken et al. (29) and Lam et al. (20). In those studies, an experimentally designed teat disinfection system specifically increased coliform mastitis. Possible mechanisms explaining this result may be a decrease in the prevalence of minor pathogens, a decrease in SCC, or a decreasing competition for receptors or nutrients in disinfected quarters (20).

Contrary to expectations, mastitis caused by *Staph. aureus* was not associated with milking management or milking machine variables, except the stripping of foremilk before cluster attachment. Across farms, milking machine variables were difficult to compare because of large variation.

The stripping of foremilk may be associated with increased exposure to contagious pathogens (29). Subclinical mastitis caused by *Staph. aureus* is usually transmitted among cows during milking (7). Apparently, expression of clinical signs does not depend on these factors and is more associated with teat lesions, hygiene, the stripping of foremilk, and breed. Whether the stripping of foremilk results in improved detection of clinical mastitis or whether the stripping of foremilk is really a risk factor for IMI is questionable. In herds with a high prevalence of IMI caused by *Staph. aureus*, it has been advised that foremilk not be stripped so that the transmission of this contagious pathogen can be reduced. However, in the etiology of mastitis caused by *E. coli*, stripping is not thought to be important.

Clinical mastitis caused by *Staph. aureus* was observed more in herds with a lower bulk milk SCC than in herds with higher bulk milk SCC. Subclinical infections caused by *Staph. aureus* are likely to be more prevalent in herds with a high bulk milk SCC (11, 14), but apparently these infections do not lead to clinical signs that can be observed by the farmer.

Results of this study and others may be used to formulate prevention programs for farms with high incidences of clinical mastitis. Some caution is required because statistical association does not automatically indicate a causal relationship. Requirements for causality are temporality, consistency, biological gradient, plausibility, and experimental evidence (25). Only causal factors are expected to decrease the incidence of clinical mastitis when eliminated in a control program. A number of factors in our data are likely to be causally associated such as breed, postmilking teat disinfection, cows that are leaking milk, and cows with trampled teats (1, 7, 20, 33), but other factors are not (e.g., stripping of foremilk, disinfection of maternity area, regular disinfection of stalls, use of a thick layer of stall bedding). From these results, we recommend that another experiment be undertaken to show, in an experimental

setting, whether this last group of variables is causally associated with the occurrence of clinical mastitis.

Several variables that were initially associated with the rate of clinical mastitis disappeared in the final models, which was, in part, due to the relatively small number of observations ($n = 144$) and the strong interdependence among management practices. Studies such as this one are necessary components to formulate control programs, but they are not sufficient. Preferably, a control program should show a reduction in the incidence of clinical mastitis when evaluated in a prospective study.

ACKNOWLEDGMENTS

The authors thank the bacteriologists of the laboratory of the Animal Health Service (Boxtel, The Netherlands), the enthusiastic trainees of the Agricultural College (Dronnten, The Netherlands: F. Rijvers, I. Rijvers, and M. V. Kempen, and 's-Hertogenbosch, The Netherlands: H. Groeneveld and A. Huysmans), and the participating farmers.

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