

Relationship Between Teat-End Callosity and Occurrence of Clinical Mastitis

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ABSTRACT

A longitudinal study in 15 herds, with a total of 2157 cows, was conducted to examine the relationship between teat-end callosity (TEC) and the incidence of clinical mastitis. During the 1.5-yr study period, clinical mastitis was diagnosed by the farmers based on clinical signs. Teat-end callosity was scored every month according to a teat-end callosity classification system, which discriminates between teat-end callosity thickness (TECT) and roughness (TECR). Differences in TECT between healthy and clinical mastitis quarters within infected cows were small but significant 3 mo before (0.13 higher), in the month during which the clinical mastitis occurred (0.08 higher), and in the following 2 mo (0.06 and 0.05 higher). To compare TECT and TECR between cows with and without clinical mastitis, 199 cows with clinical mastitis were paired with control cows based on herd, days in milk, and parity. Clinical mastitis cows had more TEC than their healthy herd mates, particularly when clinical mastitis occurred between the second and fifth months of lactation. Clinical *Escherichia coli* mastitis in the second or third month of lactation occurred in cows with less TEC than in cows with clinical mastitis caused by other pathogens. Clinical culture-negative, yeast, *Klebsiella pneumoniae*, and *Enterobacter aerogenes* mastitis cows had more TECT and TECR than other cows with clinical mastitis in the same month of lactation. Pointed teat ends had higher TECT and TECR than flat or inverted

teat ends. Teat-end callosity thickness increased with a higher milk yield at peak production.

(Key words: clinical mastitis, teat condition, teat-end callosity)

Abbreviation key: TEC = teat-end callosity, TECR = teat-end callosity roughness, TECT = teat-end callosity thickness.

INTRODUCTION

Mastitis continues to be one of the economically most important diseases in dairy farming. The incidence rate of clinical mastitis ranges from 12.7 to 30% per cow-year at risk (Barkema et al., 1998; Chassigne et al., 1997; Miltenburg et al., 1996; Sargeant et al., 1998; Schukken, 1989). Bacteria that cause clinical mastitis usually enter the udder through the teat canal. The first line of defense against clinical mastitis is therefore the teat canal, and changes in teat tissue around the teat canal may favor penetration of bacteria into the udder (O'Shea, 1987).

Mechanical forces during machine milking may induce changes in teat end tissue. Teat-end callosity (TEC) builds up until approximately 4 mo of lactation and decreases thereafter (Neijenhuis et al., 2000). Cow factors like teat-end shape, teat position, teat length, milk yield, stage of lactation, and parity are associated with the degree of TEC (Bakken, 1981; Graf, 1982; Johannson, 1957; Michel et al., 1974; Neijenhuis et al., 2000; Rathore, 1977; Sieber, 1980; Sieber and Farnsworth, 1981).

An association between teat-end condition and clinical mastitis is often assumed in the field. Severe teat-end lesions (erosions or scabs) are positively associated with the prevalence of subclinical mastitis (Sieber and Farnsworth, 1981; Jackson, 1970). At the farm level, no relationship between SCC and degree of TEC was found (Shearn and Hillerton, 1996). Michel et al. (1974) stated that TEC is not a criterion for udder health based on

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SCC and bacteriology. In a cross-sectional study on 3982 teats, no positive correlation was found between TEC and the incidence of clinical mastitis (Thompson and Sieber, 1980). However, a relationship between TEC and the incidence of clinical mastitis was never examined in a longitudinal study, the month that mastitis occurred was not taken into account, and inadequate TEC classification systems were used.

The goal of this study was to examine the relationship between the occurrence of clinical mastitis and TEC in more detail. First, we determined differences in TEC between quarters within clinical mastitis cows. Second, we investigated, in a longitudinal study, the differences in TEC between clinical mastitis cows and paired herd mates without clinical mastitis, taking into account DIM and the lactation month in which clinical mastitis occurred. Third, we evaluated differences between pathogens causing the clinical mastitis with respect to TEC. Fourth, we examined the association between TEC and clinical mastitis while accounting for cow parameters (teat length, teat-end shape, and milk production).

MATERIALS AND METHODS

Study Population and Data Collection

Fifteen farms throughout The Netherlands were selected that a) participated in a three or four weekly milk recording scheme (Royal Dutch Dairy Syndicate, Arnhem, The Netherlands), b) had good cow identification in the milking parlor, and c) had milkers with known ability to detect clinical mastitis. Farms were either experimental (farm 1 to 9) or commercial (farms 10 to 15).

Between the 15 farms, 305-d milk yield ranged from 6682 to 10,069 kg. Average milk yield per cow per day ranged from 21 to 35 kg (Table 1). These ranges reflect the differences throughout The Netherlands (CR Delta, 1999).

Farms were visited monthly from August 1995 until February 1997. On average, 18 visits were made per farm. During the visit, TEC of the four teats of all cows was scored according to a previously described TEC classification system (Neijenhuis et al., 2000). In this classification system, TEC thickness (**TECT**) is scored from 1 to 5 (where 1 = no callosity ring to 5 = extremely thick callosity ring), and TEC roughness (**TECR**) is scored 0 or 1 (where 0 = smooth or no callosity ring, and 1 = rough callosity ring). Average TECT of teats was calculated by using the unit scores 1 to 5; TECR is expressed as proportion of the teats showing rough callosity rings. A total of 88,672 quarter records of TECT and TECR were obtained. Teat scoring was done by well-trained staff, and the accuracy of scoring was

checked every 6 mo to make scoring results as comparable as possible.

During the study period, the farmers took aseptic milk samples before treatment from all udder quarters that had clinical signs of mastitis before treatment. After collection, the milk samples were immediately frozen at approximately -20°C . During farm visits, the frozen milk samples were collected and taken to the laboratory for bacteriological examination. Bacteriological examination was carried out according to National Mastitis Council standards (Hogan et al., 1999).

Teat length and teat-end shape were scored twice a year. Teat-end shape was classified as round, flat, pointed, or inverted. Teat length was measured just before premilking treatment, from base to tip, with a handmade device consisting of a pipe with a diameter of 4.5 cm and length of 11 cm.

Data Analyses

Before statistical analysis, observations were checked for unlikely values. No data were excluded for this reason. The analyses were carried out in two steps: 1) relationship between TEC and occurrence of clinical mastitis within cows, and 2) differences in TEC between cows with clinical mastitis and paired cows without clinical mastitis. The final regression model of step 2 was used to examine the association of specific pathogens with TEC. Effects of teat length, teat-end shape, and peak production on TEC were analyzed by including these factors in the same basic regression model in which clinical mastitis was included.

Within-Cow Analysis

Only first occurrences of clinical mastitis were eligible for inclusion into the analyses. Differences in TEC between the clinical mastitis quarter and the lateral quarter without clinical mastitis (T_DIFF) were calculated. Data from up to 4 mo before the occurrence of clinical mastitis until 4 mo after were used. Only records of cows in lactation were used. For the response variable T_DIFF , the following random regression model was fitted using the method of residual maximum likelihood (REML) (Genstat 5, 1996, Genstat 5, 1993):

$$\begin{aligned} \Delta_t Y_{1t} - Y_{2t} = & \mu_0 + \text{fixed effects} + \text{random effects} \\ & + \text{residual} = \mu_0 + PER + \text{Farm.Cow.Udder half} \\ & + \text{Farm.Cow.Udder half.Period} + \varepsilon_{rt} \end{aligned} \quad [1]$$

where Δ_t = difference in TECT or TECR between clinical mastitis quarter and the lateral quarter without clinical mastitis, where t indicates month from up to 4 mo before the occurrence of clinical mastitis until 4 mo after ($t =$

Table 1. Descriptive characteristics of herds during the study period.

Farm #	Average no. lactating cows	Mean parity	Average 305-d milk yield (kg/cow)	Average daily milk yield (kg/cow)	% Clinical mastitis ¹	Average TECT score ²	Average TECR score ³
1	114	2.8	7936	26	13	2.14	0.34
2	91	2.9	7843	25	25	2.16	0.30
3	40	2.4	9672	30	48	2.33	0.41
4	118	3.0	7743	24	16	2.20	0.41
5	72	2.8	7951	25	20	2.17	0.28
6	70	3.2	7897	25	11	2.12	0.24
7	71	2.4	7763	25	25	2.22	0.33
8	68	3.0	8135	26	38	2.24	0.44
9	37	2.8	7968	28	15	2.24	0.28
10	99	2.1	10,069	35	57	2.33	0.58
11	96	3.2	7048	23	23	2.11	0.28
12	55	3.5	7617	25	45	2.19	0.45
13	85	3.3	7980	25	22	2.09	0.22
14	110	2.7	6682	21	35	2.12	0.31
15	71	3.3	7803	26	27	2.07	0.25
Average	80	2.9	8000	26	28	2.18	0.34

¹Calculated as quarter cases per 100 cows present per year.

²TECT = Teat-end callosity thickness.

³TECR = Teat-end callosity roughness.

$-4, -3, \dots, 4$), Y_{1t} = TECT or TECR of clinical mastitis quarter, Y_{2t} = TECT or TECR of lateral quarter without clinical mastitis, μ_0 = overall mean, PER = effect of month compared with the moment of clinical mastitis (-4 to 4), $Farm.Cow.Udder\ Half$ = random effect of udder half within cow within farm, $Farm.Cow.Udder\ Half.Period$ = random effect of month within udder half within cow within farm, and ε_{rt} = residual random error.

Between-Cow Analysis

Cows that had clinical mastitis were eligible for inclusion as cases. Only data from the lactation in which the clinical mastitis occurred were used. The clinical mastitis cows were paired with control cows from the same farm and with the same parity (1, 2, 3, 4, and 5 or ≥ 6) and calving date (within 30 d), that did not have clinical mastitis during the study period. Only the scoring periods where both the clinical mastitis cow and her healthy herd mate had TEC data were used.

The response variables TECT and TECR were averaged per cow per month and analyzed by random regression models using the method of REML (Genstat 5, 1996; Genstat 5, 1993). For the response variables the consecutive measurements were shown to follow a lactation curve with a subject-specific slope and intercept (Neijenhuis et al., 2000; Wood, 1976):

$$Y = C_0 + C_1DIM + C_2 \ln(DIM) + \varepsilon_r \quad [2]$$

where Y = average TEC per cow per month, C_0 = level, C_1DIM = decreasing slope per DIM, $C_2 \ln(DIM)$ = increasing slope per DIM, ε_r = residual random error.

Where level is modeled as:

$$C_0 = \beta_0 + PAR + MAST + Farm + Farm.CC + Farm.CC.Cow + Farm.CC.Per + Farm.CC.Cow.Per \quad [2a]$$

where β_0 = overall mean, PAR = effect of parity (PAR 1 = parity 1, 2 = 2, 3 = 3 and 4 and, 4 ≥ 5), $MAST$ = effect of whether the cow belongs to the paired animals without clinical mastitis or to the clinical mastitis group, and if so, in which month the clinical mastitis occurred ($MAST$ is 0 = no clinical mastitis during the lactation and clinical mastitis did occur in 1 = first month of lactation, 2 = second month of lactation, 3 = third month of lactation, 4 = fourth and fifth months of lactation and 5 = later than 5th mo of lactation), $Farm$ = random effect of farm (where farm range from 1 to 15), $Farm.CC$ = random effect of couples of paired animals within farm (where couples range from 1 to 199), $Farm.CC.Cow$ = random effect of cow within couples of paired animals within farm (where animal is 1 or 2), $Farm.CC.Cow.Per$ = random effect of month within couples of paired animals within farm (where month ranges from 1 to 18) and, $Farm.CC.Cow.Per$ = random effect of month within cow within couples of paired animals within farm where decreasing slope is modeled as:

$$C_1DIM = \{\beta_1 + PAR + MAST + Farm.CC + Farm.CC.Cow\}DIM \quad [2b]$$

where parameters are as in model [2a], and increasing slope is modeled as:

Table 2. Number of clinical mastitis cases by pathogen and lactation month.

	Lactation stage (month)					Total
	1	2	3	4 & 5	≥6	
<i>Escherichia coli</i>	13	6	10	11	8	48
<i>Staphylococcus aureus</i>	17	8	0	8	2	35
Coagulase-negative staphylococci	13	1	1	3	4	22
<i>Streptococcus dysgalactiae</i>	12	5	1	4	2	24
Culture negative	8	3	2	6	2	21
<i>Streptococcus uberis</i>	6	1	2	1	1	11
Other	10	4	6	4	1	25
Not sampled	6	1	3	0	3	13
Total	85	29	25	37	23	199

$$\underline{C}_2DIM = \{\beta_2 + PAR + MAST + Farm.CC + Farm.CC.Cow\} \ln(DIM) \quad [2c]$$

where parameters are as in model [2a].

Pathogens

Model 2 was also a basis to examine whether TEC of cows that had clinical mastitis caused by a specific pathogen differed from the average TEC of cows with clinical mastitis. A pathogen was studied if that specific pathogen was identified in more than 10 mastitis cases during the study period. The remainder pathogens were grouped as 'other.' Because specific pathogen cases were not orthogonally spread over the lactation months in which clinical mastitis occurred (Table 2), only TEC curves were fitted if more than two cows had clinical mastitis with a specific pathogen in a specific lactation month. *Escherichia coli*, *Staphylococcus aureus*, *Streptococcus dysgalactiae*, coagulase-negative staphylococci, and *Streptococcus uberis* were the major pathogens involved. For each pathogen, a separate model was built. These models consisted of model 2, extended with terms for level, decrease, and increase of the pathogen of interest. The use of level, decrease, and increase allows for a subject-specific slope and intercept (Neijenhuis et al., 2000; Wood, 1976). For example, the *E. coli* model looks as follows:

$$Y = C_0 + C_1DIM + C_2 \ln(DIM) + ECO + ECODIM + ECO \ln(DIM) + \varepsilon_r \quad [3]$$

where parameters are as in model [2] and ECO is a binary variable with value 1 when a cow had *E. coli* mastitis in a month or 0 when a cow had no *E. coli* mastitis in that specific month.

The differences between the effect estimates for TEC as given by the pathogen-specific models (such as model 3) and general mastitis model (model 2) were calculated. These differences were used to evaluate the effect of a specific mastitis pathogen on TEC.

Other Cow Parameters

On top of model [2] teat-end shape, teat length, and peak milk yield were examined for their relationship with TEC.

$$Y = C_0 + \underline{C}_1DIM + \underline{C}_2 \ln(DIM) + TES + TL + KG + \varepsilon_r \quad [4]$$

where Y = average TEC, C_0 = level, \underline{C}_1DIM = decreasing slope, per DIM, $\underline{C}_2 \ln(DIM)$ = increasing slope, per DIM, TES = effect of teat end shape (where 1 = pointed, 2 = round, 3 = flat, and 4 = inverted); TL = effect of teat length (where 1 < 4.5 cm, 2 = 4.5 to 5.0 cm, 3 = 5.0 to 5.5 cm, and 4 = > 5.5 cm); KG = effect of peak milk production per day between 10 and 20 wk of lactation (where milk yield ranges from 14 to 63 kg), and ε_r = residual random error.

RESULTS

Study Population and Descriptive Results

During the 1.5-yr study period, 2157 different animals were observed during the farm visits. Teat-end callosity of 2051 animals was scored; 123 animals were only scored during the dry period or as nonlactating pregnant heifers. From the 1928 lactating animals, 103 had only one TEC score during lactation. On average, TEC was scored 8.6 times per animal during lactation. Average TECT score was 2.18, ranging per farm from 2.07 to 2.33 (Table 1). For lactating cows, the average score was 2.22, ranging from 2.10 to 2.41 per farm. On average, 34% of the udder quarters had rough callous rings, ranging from 22 to 58% among farms. On average, 38% of the udder quarters of lactating cows had rough callous rings, ranging from 24 to 65% among farms.

Table 3. Differences in teat-end callosity thickness (TECT) and roughness (TECR) of clinical mastitis quarters and lateral quarters without clinical mastitis on average (μ), and 4 mo before the clinical mastitis occurred to 4 mo after the clinical mastitis ($X[-4] - X[4]$ and $X[0]$ = month of clinical mastitis), calculated from the final regression model.

Variable	TECT			TECR		
	Coefficient	SE(β)	P-value	Coefficient	SE(β)	P-value
μ	0.03188	0.02107	0.065	0.007861	0.01370	0.28
X[-4]	0.05667	0.05133	0.13	0.02719	0.03619	0.23
X[-3]	0.1347	0.04383	0.0011	0.02311	0.03086	0.23
X[-2]	0.008781	0.03917	0.41	-0.02623	0.02757	0.17
X[-1]	0.01991	0.03556	0.29	0.02869	0.02501	0.13
X[0]	0.07543	0.03108	0.0077	0.007737	0.02187	0.36
X[1]	0.05976	0.03163	0.030	-0.02640	0.02230	0.12
X[2]	0.05826	0.03256	0.037	0.004219	0.02301	0.43
X[3]	0.04913	0.03405	0.075	-0.02522	0.02411	0.15
X[4]	0.02560	0.03666	0.24	0.02493	0.02601	0.17

Within-Cow Analysis

In the within-cow analysis, 491 cows and 505 udder halves were included. Teat-end callosity thickness of the clinical mastitis quarters was on average 0.03 ($P = 0.07$) higher than lateral quarters without clinical mastitis (Table 3). Clinical mastitis quarters had 0.8% ($P = 0.28$) more rough callosity rings than healthy quarters. Before and after the clinical mastitis occurred, TECT was higher on the clinical mastitis quarters than in lateral quarters without clinical mastitis. Three months before the clinical mastitis occurred, TECT was 0.13 higher ($P < 0.05$). In the month clinical mastitis occurred, TECT was 0.08 higher ($P = 0.008$), and 1 mo after the clinical mastitis, TECT was 0.06 higher ($P = 0.03$). Two months after clinical mastitis, the TECT was 0.06 higher ($P = 0.04$). Clinical mastitis quarters and lateral quarters without clinical mastitis did not differ significantly in TECE in any of the months during the period of up to 4 mo before to 4 mo after clinical mastitis occurred.

Between-Cow Analysis

Between farms, the incidence of clinical mastitis ranged from 11 to 57% per 100 cows per year (Table 1). During the study period, 827 quarter cases of clinical mastitis were diagnosed. These clinical mastitis cases involved 674 different quarters of 509 different animals. From these clinical mastitis cases, 42 animals had no or just one record on TEC. From the cows with clinical mastitis, 467 were eligible for inclusion into the analyses with their first clinical mastitis case. All of them had TEC scores during lactation. On average, 10.8 records on TEC during lactation were recorded for the animals that had clinical mastitis. The 2570 records of TEC from 199 couples of paired animals were brought into the analysis.

Days in milk, parity, and whether and when clinical mastitis occurred were associated with the course of TECT and TECE. Teat-end callosity increased after parturition until approximately 120 DIM and decreased thereafter. Clinical mastitis cows had thicker, and more frequently rough, callous rings on their teat ends than cows that did not have clinical mastitis, both before and after the clinical mastitis occurred, at least if the clinical mastitis occurred after the first and before the sixth month of lactation (Figures 1 and 2). On the other hand, cows with clinical mastitis in the first month of lactation showed less TECT and TECE during lactation than other cows.

First-parity cows had the least TECT followed by the higher-parity cows (parity ≥ 5). Cows with parities 3 and 4 had the highest TECT followed by parity 2 cows (Figure 3). First-parity cows had the least TECE fol-

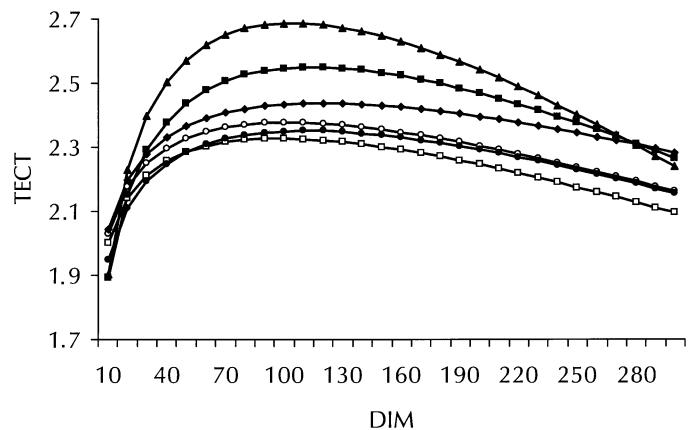


Figure 1. Teat-end callosity thickness (TECT) during lactation for second-parity cows without clinical mastitis ($-\square-$), or with clinical mastitis in the first ($-\square-$), second ($-\blacktriangle-$), third ($-\blacksquare-$), fourth and fifth ($-\blacklozenge-$), and ≥ 6 ($-\bullet-$) months of lactation, calculated from the final regression model.

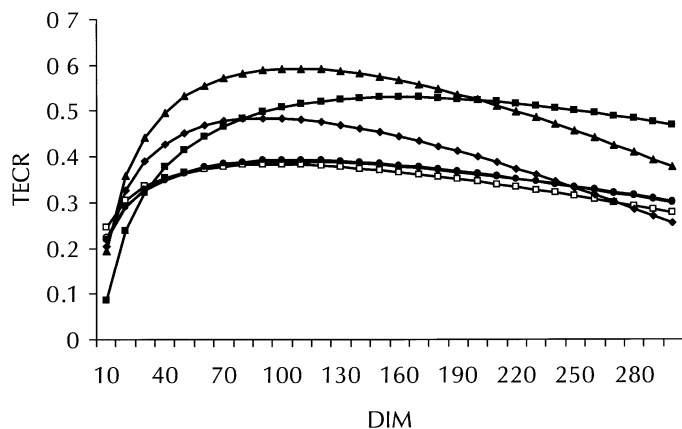


Figure 2. Teat-end callosity roughness (TECR) during lactation for second-parity cows without clinical mastitis (—○—), or with clinical mastitis in the first (—□—), second (—▲—), third (—■—), fourth and fifth (—◆—), and ≥ 6 (—●—) months of lactation, calculated from the final regression model.

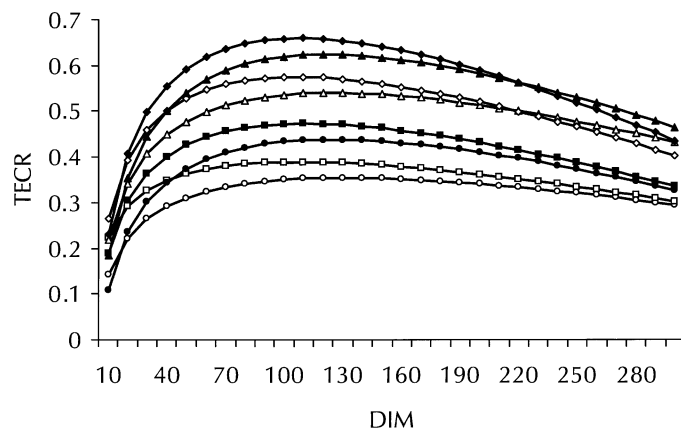


Figure 4. Teat-end callosity roughness (TECR) during lactation for cows with (solid) and without clinical mastitis (open) per parity (● = parity 1, ■ = parity 2, ▲ = parity 3 and 4, and ◆ = parity ≥ 5), calculated from the final regression model.

lowed by the second-parity cows. Cows with parities 3 and 4 had the highest TECR followed by higher-parity cows (parity ≥ 5) (Figure 4). Within-parity groups, clinical mastitis cows had more TEC than nonmastitis cows.

Pathogens

The pathogens most frequently isolated from the 827 quarter cases of clinical mastitis were *Staph. aureus* (24%), *E. coli* (21%), *Strep. dysgalactiae* (11%), coagulase-negative staphylococci (11%), and *Strep. uberis* (7%). From 11% of the milk samples no pathogen was isolated.

From the 199 clinical mastitis cases selected for the analysis, 186 were examined bacteriologically. Patho-

gens most frequently isolated in this subset were *E. coli* (26%), *Staph. aureus* (19%), *Strep. dysgalactiae* (13%), coagulase-negative staphylococci (12%), and *Strep. uberis* (6%). From 11% of the milk samples, no pathogen was isolated.

Compared with cases of clinical mastitis in the same month of lactation, some pathogens were associated with a small difference in TECT or TECR. Clinical *E. coli* mastitis in the second- (level $P = 0.023$) or third-lactation month (level $P = 0.014$ and increase $P < 0.001$) occurred in cows with less TECT during the complete lactation. Cows with clinical mastitis caused by other pathogens in the second (increase $P = 0.0073$) or third lactation month (increase $P = 0.001$) had more TECT, the TECR was also more severe in cows with clinical mastitis in the third mo of lactation (level $P = 0.014$). The clinical mastitis cases that were culture negative had more TECT if clinical mastitis occurred in the second-lactation month (level $P = 0.032$), fourth- or fifth-lactation month (decrease $P = 0.0065$) as did the cases after the fifth month (decrease $P = 0.012$), the TECR was also more severe in cows with clinical mastitis in the fourth- or fifth-lactation month (decrease $P = 0.018$).

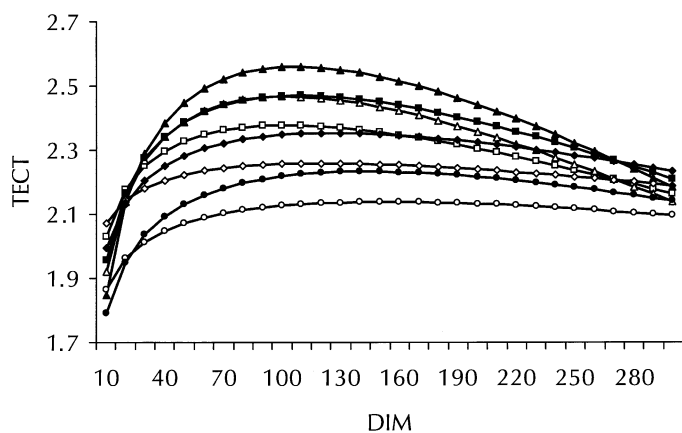


Figure 3. Teat-end callosity thickness (TECT) during lactation for cows with (solid) and without clinical mastitis (open) per parity (● = parity 1, ■ = parity 2, ▲ = parity 3 and 4, and ◆ = parity ≥ 5), calculated from the final regression model.

Teat-End Shape and Length and Milk Production

Cows with clinical mastitis more frequently had pointed teat ends than nonmastitis cows (6 versus 2%; $P = 0.019$). In both groups, most of the teat ends were round (58%). No difference in teat length between the clinical mastitis and the nonmastitis cows was found ($P = 0.23$); teat length was on average 4.8 cm. Milk yield at top lactation was on average 33.5 kg/d and did not differ between the clinical mastitis and the nonmastitis cows ($P = 0.28$).

Table 4. Association between teat-end shape and maximum milk yield per day between 10 to 20 wk in lactation, and teat-end callosity thickness (TECT), and teat-end callosity roughness (TECR) on top of the complete model (199 couples of matched cows).

	TECT		TECR	
	Coefficient	<i>P</i> -value	Coefficient	<i>P</i> -value
Teat end shape				
Round vs. pointed	-0.1120	0.16	-0.1197	0.048
Flat vs. pointed	-0.2433	0.018	-0.2941	<0.001
Inverted vs. pointed	-0.4069	<0.001	-0.4513	<0.001
Flat vs. round	-0.1313	0.0050	-0.1744	<0.001
Inverted vs. round	-0.2949	0.0089	-0.3316	<0.001
Inverted vs. flat	-0.1636	0.099	-0.1572	0.020
Peak milk yield (per 10 kg)	0.1043	<0.001		

¹Empty cells indicate that factor is not significant and left out of the model.

When adding the cow factors, teat length, teat-end shape, and peak yield to model [2], teat length did not account for any variance and was left out of the models for TECT and TECR. Peak yield did also not account for any variance in the model for TECR and was left out. The addition of teat-end shape and peak yield resulted in almost the same estimations for the coefficients of the other variables in the basic model [2]. Pointed teat ends had higher TECT and TECR than flat or inverted teat ends (Table 4). Round teats had more TECT and TECR than inverted teat ends. Flat teats had more TECR than inverted teat ends. Pointed teats had more TECR than round teat ends. With increasing peak yield, TECT increased. For each 10-kg increase in milk yield, the TECT was 0.1 higher ($P < 0.001$; Table 4).

DISCUSSION

Forty percent of the quarters had clinical mastitis during the 1.5-yr study period; on average, 28% quarter cases per 100 cows were present per year. This falls within the range of other research (Barkema et al., 1998; Miltenburg et al., 1996; Schukken, 1989; Chasagne et al., 1997; Sargeant et al., 1998).

In contrast to previously published work (Sieber and Farnsworth, 1981; Thompson and Sieber, 1980), this study has shown a small but consistent and significant relationship between TEC and clinical mastitis within mastitis cows, and between clinical mastitis cows and paired herd mates without clinical mastitis. Before, and particularly after, the clinical mastitis occurred, TECT was higher in the clinical mastitis quarters than in lateral quarters without clinical mastitis of the same cow. It is assumed that clinical mastitis causes milk flow changes in the affected quarter. These changes may lead to more stress on the teat by milking, and may result in more TECT after the clinical mastitis had occurred. Higher levels of TECT before clinical mastitis

indicates a relationship between TECT and risk of clinical mastitis. The difference of TECT 3 mo before the clinical mastitis occurred, between the clinical mastitis quarter compared with the healthy lateral quarter, was 0.13-unit scores. On a scale of 5, this is not a large difference and the biological importance of this finding may be questioned. However, the difference of TECT between farms is 0.26-unit scores, which shows that the difference between mastitis and nonmastitis udder quarters is relatively high. Moreover, this difference in TECT between farms may be part of an explanation for the large differences in mastitis incidence between farms. However, this should be confirmed by further research. The small difference in TECT between quarters with and without mastitis can therefore be considered as biologically important. TECR did not differ significantly between the clinical mastitis quarters compared with the lateral quarters without clinical mastitis of the same cow. This may be evidence that the development of TECT and TECR are not the same process and should be used as complementary parameters (Neijenhuis et al., 2000).

Differences were found in TEC between clinical mastitis cases and paired healthy cows. Part of the observed differences in TEC between cows could be explained by differences in stage of lactation, milk yield, parity, and teat-end shape. This is in accordance with other research (Graf, 1982; Johannsson, 1957; Michel et al., 1974; Neijenhuis et al., 2000; Rathore, 1977; Sieber, 1980; Sieber and Farnsworth, 1981).

Cows that presented with clinical mastitis in the second to the fifth month of lactation showed more TEC than the paired healthy cows. Teat-end callosity develops in the first month up to fourth month in lactation and decreases thereafter. The association with clinical mastitis and TEC was found within this period, suggesting a biological relationship between development of TEC and clinical mastitis.

Cows with clinical mastitis cases in the first month of lactation showed no difference in TEC compared with paired cows without clinical mastitis. The distribution of cases of clinical mastitis during lactation is skewed, with most cases in the beginning of lactation (43%), which is similar to other research (Miltenburg et al., 1996; Rajala-Schultz et al., 1999). The cause of these cases may be the depressed immune system at parturition and smooth muscle contraction, which is vital to closure of the teat sphincter after milking, and is suspected to be impaired by hypocalcemia (Goff and Horst, 1997).

Specific pathogens may differ in opportune use of TEC to multiply or to enter the teat canal. The only groups of pathogens of clinical mastitis cases occurring in the second or third months of lactation that had an association with increased TECT were the group 'other'. Because of the small incidence, the group 'other' contained 10 different pathogens. Within this group clinical mastitis cases with yeast (two cows), *Klebsiella pneumoniae* (one cow), and *Enterobacter aerogenes* (two cows) had higher TEC than other cows. Barkema et al. (1999) reported an association between milking machine factors and clinical *E. coli* mastitis. Some of these milking machine factors are associated with increasing TEC. In contrast, in this research, an association of clinical *E. coli* mastitis with less TECT was found.

Teat-end shape was mostly round, similar to that found in other research (Chrystal et al., 1999; Bakken, 1981; Johansson, 1957). The finding that round or rather pointed teat ends were more prone to have higher TECT and TECR than flat or inverted teat ends was in agreement with other research (Bakken, 1981; Johansson, 1957; Neijenhuis et al., 2000; Rathore, 1977). Cows with clinical mastitis frequently had more pointed teat ends than their paired healthy herd mates. Natzke et al. (1978) found that cows with pointed teat ends had the highest rate of new infections. In contrast, other studies (Hodgson and Murdock, 1980; Seykora and McDaniel, 1985) reported that as teat-end shape varied from pointed toward flat and inverted, SCC increased. However, a more recent study (Chrystal et al., 1999) showed no relationship between teat-end shape and SCC. Adding teat-end shape to the model in this study did not alter the relationship between clinical mastitis and TEC.

The average teat length found was 4.8 cm, within the range of other research (Hamann, 1987; Bakken, 1981). Teat length did not account for differences in TEC, similar as found by Johansson (1957). With increasing peak yield, TECT increased, similar to that found in other research (Bakken, 1981; Sieber, 1980). Higher milk yield will be accompanied by longer machine-on time, which results in more TEC (Neijenhuis et al.,

2000). When milkflow rate was adjusted for milk yield, Seykora and McDaniel (1985) found a negative association with TEC. Dohoo and Martin (1984) stated that the level of milk production was not significantly related to the risk of mastitis. Woolford (1985) found that there is milk loss around the occurrence of clinical mastitis but compensation takes place in older cows. Therefore, the influence of peak milk yield can be underestimated within first-parity cows. Adding peak yield to the model in this study did not alter the relationship between clinical mastitis and TEC.

CONCLUSIONS

The level of TECT was higher in the clinical mastitis quarter than in the lateral healthy quarter within a cow, before and particularly during and after occurrence of clinical mastitis. Quarters with or without clinical mastitis did not differ significantly in roughness of the callosity ring.

Teat-end callosity of cows with clinical mastitis in the second to fourth months of lactation was higher before and after the occurrence of clinical mastitis occurred compared with paired healthy herd mates. However, cows with clinical mastitis in the first or after the fifth months of lactation, had less TECT and TECR.

Clinical mastitis cases which were culture-negative or caused by less frequently found pathogens like yeast, *Kl. pneumoniae* and *E. aerogenes* were associated with higher TEC. Clinical *E. coli* mastitis was associated with less TECT.

Teat-end shape was associated with the degree of TECT and TECR. With increasing peak yield, TECT increased. Teat length was not associated with TEC.

From this study, it is clear that clinical mastitis does have a relationship with TEC. Further research should focus on TEC as risk condition for mastitis. If TEC is a risk factor for clinical mastitis, it could be used as a predictor for clinical mastitis in milking machine research.

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