Change of Milk Yield with Clinical Diseases for a High Producing Dairy Herd

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ABSTRACT

Changes in milk production associated with occurrence of clinical diseases (dystocia, stillbirth, twin births, milk fever, retained placenta, displaced abomasum, limping due to foot lesions, metritis, ketosis, and mastitis) were investigated. Data were collected daily on 388 lactation. Stepwise least squares regression was used to evaluate existence of associations between diseases and six yield measures that characterized milk production in the first 119 d postpartum. Logistic regression was used to investigate whether milk yield 1 to 5 d in milk might be of use to detect cows with early postpartum metritis (< 21 d after calving). Lower milk production to 5 d postpartum was associated with an increased risk of early postpartum metritis in the logistic regression model. Yield to 5 and to 21 d postpartum was lower in cases of stillbirth, retained placenta, and early postpartum metritis. Yield from 22 to 49 d postpartum remained lower in cows diagnosed with early postpartum metritis. Milk yield losses occurred during diagnosis and treatment of displaced abomasum and mastitis. Ketosis was associated with yield losses prior to and at treatment. Ketosis to 21 d in milk was also associated with lower production after treatment. Limping diagnosed in the first 49 d postpartum coincided with higher yield to 5 d, to 21 d, and after 49 d postpartum.
(**Key words**: metritis, clinical disease, milk production)

Abbreviation key: AVG = average daily milk yield, CMT = California Milk Test, CUM = cumulative milk yield, DIM = days in milk, DYST = dystocia, MET1 = early postpartum metritis, MF = milk fever, OR = odds ratio, PEAK = peak milk production, RP = retained placenta, SB = stillbirth, TWIN = twin births.

INTRODUCTION

Daily monitoring of milk production in dairy cows is now possible with commercially available automated milk weighing devices and transponders for individual cow identification (8, 9). Several studies found 305-d milk production to be affected in cows suffering from various clinical diseases and reproductive disorders (11, 14, 15, 16, 18). Systems for automated milk yield recording might be of use for early detection of sick cows if these milk production changes can be detected prior to or at the same time as usual clinical disease diagnosis.

We previously reported (7) that time series analysis models might be useful to discern changes in milk production associated with ketosis and mastitis. However, these models failed to demonstrate yield changes associated with metritis. The first objective of this study was to investigate occurrence of disease-associated temporal patterns in milk yield in the first 120 d in milk (DIM), when cows are at highest risk for disease (6). A second objective was to determine if yield from 1 to 5 DIM was useful for detecting early postpartum metritis (<21 DIM) (MET1).

MATERIALS AND METHODS

Data

This study was carried out on a 500-cow Holstein dairy in the San Joaquin valley in California. Cows were managed in four produc-

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tion strings (fresh cows and high, medium, and low producers) and fed a total mixed ration formulated according to 1978 NRC requirements (26). Data pertaining to the first 119 DIM were collected on lactations that lasted 119 or more d and that were initiated between August 1, 1986 and July 31, 1987.

Daily milk yield data were collected with an automated cow identification and milk yield recording system (Boumatic System 2000, Dairy Equipment Company, Madison, WI) that measured production using DHI-approved milk measurement devices (Boumatic Model M, Dairy Equipment Company, Madison, WI). During the three daily milkings occurring at 8-h intervals, information on each cow milked was captured by a microcomputer in ASCII data files and later transferred to a minicomputer database management program (Ingres, Relational Technology Inc., Alameda, CA). Missing yield data, which constituted less than 5% of the observations, were estimated using Kalman smoothed estimators (6). The PD in 305-d mature equivalent milk yield for cows' sire (sire PD milk) was obtained from DHI records. Similar dam information contained too many missing values and was not used.

Occurrences of dystocia (DYST), stillbirth (SB), twin births (TWIN), milk fever (MF), retained placenta (RP), displaced abomasum, limping due to foot lesions, metritis, ketosis, and mastitis were recorded daily by the dairy producer using the following definitions. Dystocia was defined as a delivery in which assistance was necessary. The placenta was considered retained if it released more than 24 h after calving. The diagnosis of MF was based on the cow's inability to rise, with successful correction by intravenous calcium treatment, and on the event occurring between 5 d prior and 2 d after calving. Cows were screened in the early postpartum period for clinical signs of metritis such as purulent discharge and fever. Metritis was diagnosed at routine postpartum veterinary examinations, and its detection was based on an abnormally enlarged uterus or cervix. Cows with metritis in one time period could be diagnosed to be reaffected if found free at an intermediary veterinary examination. Ketosis was diagnosed by detecting depressed feed intake, a drop in milk production, no concurrent disease, and a positive response to treatment. Animals suspected of displaced abomasum were submitted to veterinary examination for confirmation and surgery. Lameness due to foot lesions included all limping due to signs of inflammation within the foot. Successive episodes of foot lameness were considered separate events if they occurred at intervals of more than 30 d. Clinical mastitis was detected by observing abnormal milk appearance and was confirmed using the California Mastitis Test (CMT). Each quarter with a CMT score of 1 or higher was cultured, and the results were interpreted according to the National Mastitis Council guidelines (25). Successive cases of clinical mastitis in a quarter were considered separate episodes if they occurred at intervals of more than 7 d. The senior investigator verified recording of clinical disease diagnoses at the visits made three times weekly.

Analysis

Six measures of milk production were calculated to characterize milk yield in the first 119 DIM for each cow in the study. Production for the entire period of interest was calculated as cumulative yield to 119 DIM (CUM119). Yield at the onset of lactation was defined as the average daily yield of 1 to 5 DIM (AVG1–5); d 1 was the day of calving. Following calving, there was a period of rapid production increase that we characterized as yield to 21 DIM (CUM21) (5). After that period, a more moderate increase occurred until peak yield was reached after 49 DIM, on average (5). Production in this second subperiod was characterized as cumulative yield from 22 to 49 DIM (CUM22–49). Peak production (PEAK) was defined as a cow's highest average yield during any 7-d period in the first 119 DIM. Production following peak yield was represented by a third subperiod and defined as cumulative yield from 50 to 119 DIM (CUM50–119).

The patterns of change of milk production associated with diseases were investigated using six regression models. Because analysis was for direct effects only, use of multiple linear regression, rather than path analysis (4, 11, 15), was considered appropriate. In each model, one of the defined milk yield measures was the dependent variable, and variables indicating disease occurrence, season, parity, and genetic milk potential were used as independent variables. A forward stepwise linear regression

TABLE 1. Independent variables included as covariates in the linear models of six milk yield measures characterizing milk production in the first 119 d postpartum.

Covariates	Milk yield measure[1]					
	AVG1–5	CUM21	CUM22–49	CUM50–119	CUM119	PEAK
Calving season[2]	–[3]	+[4]	+	+	+	+
Heifer[5]	+	+	+	+	+	+
Parity group[6]	+	–	–	–	–	–
PD milk	–	–	–	–	–	–

[1]AVG = Average milk yield for indicated days in milk; CUM = cumulative milk yield for indicated days in milk; PEAK = peak milk production.

[2]Three indicator variables representing four seasons by comparing winter, spring, and summer (=1) with the three other seasons (=0).

[3]Not included as a covariate in original model but tested for significance in the stepwise regression.

[4]Included as a covariate in original and successive models.

[5]One indicator variable representing heifers (=0) versus multiparous cows (=1).

[6]Two indicator variables representing third (=1) and fourth and higher parity cows (=1) versus other parity animals (=0).

algorithm (BMDP 2R, BMDP Statistical Software, Los Angeles, CA) was used to select significant independent variables. The final model selected for each dependent variable was the one with largest adjusted R^2 and which, with regard to disease occurrence, contained only independent variables whose coefficients were ($P<.05$).

Previous analyses of the data (5) have shown that the six measures of milk yield differed by parity and calving season. Frequencies of occurrence of retained placenta, metritis, and ketosis also differed significantly by parity and season. To avoid attributing effects of parity or season to diseases, the stepwise selection started from a model that already included season and parity effects. Representation of parity and calving season in the models is shown in Table 1. Because significant differences had been found (5) among the three groups of multiparous cows for PEAK and for AVG1–5, three indicator variables representing four parity groups (first, second, third, and fourth and higher) were used instead of only one (heifers versus multiparous cows). For AVG1–5, the three indicator variables representing season were not included in the original model because no seasonal differences had been found (5). Sire PD milk was used as a continuous covariate. Rather than demonstrate differences in average susceptibility to disease of cows with high or low milk yield potential, a measure of genetic potential allows investigation of a cow's inability to achieve its milk yield potential (28).

For each disease condition, indicator variables representing disease occurrence or absence (1 or 0, respectively) were used as independent variables. One indicator variable was used for DYST, SB, MF, RP, TWIN, and displaced abomasum. For diseases that could be diagnosed more than once in the period of interest, i.e., mastitis, limping due to foot lesions, metritis, and ketosis, three indicator variables were used for each disease. They indicated occurrence or absence (1 or 0, respectively) of the condition between 1 and 21 DIM (DIM1–21), 22 and 49 DIM (DIM22–49), and 50 and 119 DIM (DIM50–119). Only four cows were diagnosed with limping DIM1–21 after a median of 17 DIM. These cases were represented by the same variable as for limping DIM22–49.

Logistic regression (Statistix II, NH Analytical Software, Roseville, MN) was used to model risk of MET1. Backward stepwise regression was carried out from a model containing factors identified in previous reports (4, 5, 10, 12, 14, 15, 21, 30) to increase (directly or indirectly) the risk of metritis and which are known at the time of detection of MET1:

$$\ln(\mathrm{OR}) = A + B(\mathrm{DYST}) + C(\mathrm{RP}) + D(\mathrm{TWIN}) + E(\mathrm{MF}) + F(\mathrm{SB}) + G(\mathrm{HFR}) + H(\mathrm{SMMR}) \quad [1]$$

TABLE 2. Descriptive statistics of six measures of milk yield characterizing production in the first 119 d postpartum (n = 388).

Yield measure[1]	Mean	SD	Maximum	Minimum
AVG1–5	24.4	6.1	42.6	9.3
CUM21	671.6	129.6	976.9	329.6
CUM22–49	1095.9	190.8	1518.6	597.6
CUM50–119	2813.3	444.5	3841.3	1727.6
CUM119	4580.8	730.0	6271.2	2768.4
PEAK	43.5	7.0	50.0	25.0

[1]AVG = Average milk yield for indicated days in milk; CUM = cumulative milk yield for indicated days in milk; PEAK = peak milk production.

where

$$OR = P(MET1)/[1 - P(MET1)].$$

At each step, a variable was deleted if its deletion did not affect model fit, which was ascertained using a chi-square test (1 df) to test for significant change of the log-likelihood ratio ($P<.1$). When the final model was obtained, AVG1–5 was included, and improvement of model fit was similarly tested. Existence of significant interactions between parameters of this final model was determined similarly. Goodness of fit of the final model was also tested with a chi-square test.

Forward stepwise regression using the independent variables of model [1] and AVG1–5 was carried out in the same way as for the backward stepwise regression. Potential use of the final model as a tool to screen for MET1 was determined by calculating sensitivity, specificity, and predictive value for several cutpoints of the odds ratio (OR) (22).

RESULTS AND DISCUSSION

During the period of observation, data on 408 lactations were collected. Twenty animals were removed from the herd in the first 119 d postpartum because of low production associated with mastitis or injury of the mammary gland (5 cows), displaced abomasum (5 cows), SB (3 cows), ketosis (2 cows), and conditions not considered in this study. Data on these 20 lactations were excluded from the analysis. This has probably resulted in an underestimation of the milk production losses associated with mastitis, displaced abomasum, DYST, and ketosis described below. Descriptive statistics of the milk yield data are provided in Table 2. Indicative cumulative lactational disease incidence rates for animals studied are listed in Table 3 (5).

Adjusted R^2 varied between .52 and .60, except for AVG1–5, for which it was only .37. Significant changes in milk production variables associated with clinical disease variables are shown in Table 3.

Occurrence of TWIN, DYST, and MF were not directly associated with any of the measures of milk production examined. Several studies found no direct association of TWIN (17), DYST (11, 14, 15, 28) or MF (11, 27) with short-term, 305-d or similar measures of milk yield. However, Thompson et al. (30) reported a decrease in 30-d, but not in 90-d or 305-d mature equivalent production in heifers with DYST. They did not distinguish between DYST cases with or without metritis. Their findings may have been confounded with metritis, which has been shown to be associated directly (4, 11, 15) or indirectly (14, 15) (via retained placenta) with occurrence of DYST.

Stillbirth, also a risk factor for metritis (20), was associated with lower AVG1–5 (9.2%) and CUM21 (5.1%). Retained placenta was only associated with lower AVG1–5 (8.8%). When entered in the final model on CUM21, however, RP was associated with a 20.7-kg (2.8%) nonsignificant ($P = .1$) reduction in milk yield. Lucey et al. (19) similarly reported RP to be associated with reduced short-term yield in early lactation. Although several studies found no significant direct association of RP with 305-d production (3, 14, 15, 21) or similar yield measures (11, 16), Rowlands and Lucey (28) reported that cows with RP had lower 305-d and PEAK. Their findings also may have

TABLE 3. Effect[1] of diseases at different stages of the lactation on six measures of milk yield in the first 119 d postpartum (n = 388).

Model parameter	Postpartum period[3]	Cumulative incidence rate[4]	Milk yield measure[2]					
			AVG1–5	CUM21	CUM22–49	CUM50–119	CUM119	PEAK
Intercept[5]			27.2[6] (.6)[7]	741.1 (10.3)	1233.3 (14.8)	3103.4 (36.3)	5093.4 (56.3)	47.3 (.6)
Disease variables								
Milk fever		6.7	NS[a]	NS	NS	NS	NS	NS
Twin calvings		4.2	NS	NS	NS	NS	NS	NS
Dystocia		6.9	NS	NS	NS	NS	NS	NS
Stillbirth		6.3	–2.4 (1.1)	–37.7 (19.5)	NS	NS	NS	NS
Retained placenta		16.2	–2.4 (.7)	NS	NS	NS	NS	NS
Metritis	1– 21 d	5.6	–2.6 (1.1)	–50.1 (20.8)	–62.0 (30.0)	NS	–265.8 (110.7)	NS
	22– 49 d	9.7	NS	NS	NS	NS	NS	NS
	50–119 d		NS	NS	NS	NS	NS	NS
Displaced abomasum		2.0	NS	–202.0 (53.1)	–200.0 (77.0)	NS	NS	NS
Limping		15.2						
	1– 49 d		1.7 (.9)	36.7 (16.3)	NS	113.3 (56.5)	NS	NS
	50–119 d[8]		NS	NS	NS	NS	NS	NS
Mastitis		26.1						
	1– 21 d		–2.5 (1.2)	–53.8 (20.5)	–73.8 (29.7)	NS	NS	NS
	22– 49 d		NS	NS	NS	NS	NS	NS
	50–119 d		NS	NS	NS	–250.1 (53.1)	–281.0 (83.8)	NS

Ketosis		18.3						
	1– 21 d		NS	–66.0	–92.6	NS	–253.4	–2.7
				(20.5)	(32.6)		(123.6)	
	22– 49 d		NS	–44.3	–156.5	NS	–260.1	NS
			(18.4)	(26.6)		(100.8)		
	50–119 d		NS	NS	NS	–285.4	–336.8	NS
						(110.4)	(174.1)	
Covariates								
Heifer			–7.5	–196.2	–303.0	–682.1	–1185.9	–10.4
			(.7)	(10.2)	(14.8)	(34.9)	(55.8)	(.6)
Third parity			.9	NS	NS	NS	NS	1.9
			(.7)					(.7)
Fourth parity			–1.5	NS	NS	NS	NS	1.9
			(.8)					(.7)
Winter			NS	–6.8	–16.7	–64.0	–79.4	–1.3
				(12.2)	(17.7)	(42.4)	(67.1)	(.6)
Spring			NS	–4.2	–37.5	–108.6	–137.9	–1.8
				(12.2)	(17.7)	(42.0)	(67.0)	(.6)
Summer			NS	–18.6	–59.3	–239.7	–291.5	–3.2
				(16.7)	(24.3)	(55.8)	(92.2)	(.8)
Sire PD Milk			.001	.029	.039	.106	.167	.002
			(.0005)	(.0009)	(.001)	(.031)	(.05)	(.0004)

[a]NS = not significant ($P>.05$).

[1]Effects are values of significant parameters ($P<.05$) of a stepwise multiple linear regression model of milk production on disease, season, parity, and PD milk.

[2]AVG = Average milk yield for indicated days in milk; CUM = cumulative milk yield for indicated days in milk; PEAK = peak milk production. Effects are expressed as differences in kilograms of milk produced by affected animals.

[3]Days in milk when diagnosed.

[4]Lactational disease incidence rate (%).

[5]Milk yield when independent variables all equal 0 (no disease).

[6]Average.

[7]SE.

[8]Includes four cases of limping DIM1–21.

TABLE 4. Effect[1] of disease, season, and yield on the natural logarithm of the odds of early postpartum metritis (n = 388).

Model parameter[2]	$\overline{X}$	SE
DYST	1.630	.571
TWIN	1.735	.772
SMMR	1.071	.581
AVG1–5	–.153	.014

[1]Effects are values of significant parameters ($P<.1$) of a stepwise logistic regression model of early postpartum metritis on different parameters.

[2]DYST = Dystocia, TWIN = twin calving, SMMR = summer calving, and AVG1–5 = average yield in the first 5 d postpartum.

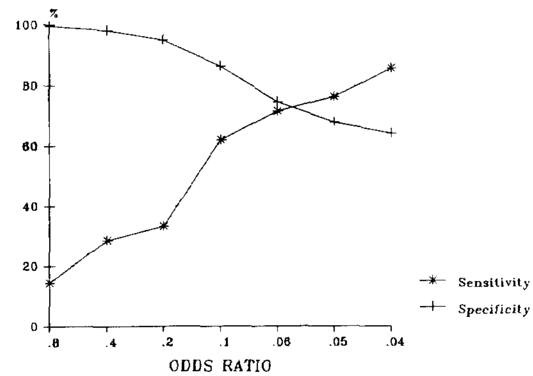

Figure 1. Sensitivities and specificities for detection of early postpartum metritis at various cutoff points of the odds ratio, using the logistic regression model in Table 4.

been confounded by metritis, for which RP is a risk factor (4, 10, 14, 15).

The milk yield reduction associated with MET1 (DIM1–21) represented 9.6% of AVG1–5, 6.9% of CUM21, and 4.9% of CUM119, indicating that the proportion of milk lost was largest at the onset of the lactation. A direct negative long-term effect on production appeared to exist, because the reduction in CUM119 associated with MET1 could not entirely be attributed to losses in DIM1–21 (Table 3). This is in disagreement with several previous reports (3, 11, 15, 27) that did not use daily measurements to estimate milk yield. Differences from previous studies could also be partly due to differences in the clinical signs used to detect the disease as well as the distinction made with regard to the postpartum period when the diagnosis was made.

The backward and the forward stepwise logistic regression algorithm resulted in the model shown in Table 4. The chi-square goodness-of-fit test indicated good model fit ($P>.999$). The intercept for this model was not significant and therefore was deleted. The OR for MET1 was increased in case of DYST (5.1), TWIN (5.7), and summer calving (2.9). A .9 decrease in the OR occurred for every kilogram of increase in average milk yield in the first 5 d postpartum. Figure 1 shows sensitivities and specificities obtained for several cutoff points of the OR of MET1. At the .04 cutoff, sensitivity and specificity were .84 and .61, and predictive value of a positive and a negative test were .11 and .98, respectively. These results do not support this model as a useful tool to screen for animals affected by MET1, as defined in the present study. Confirmation of the present results based on signs that closely reflect the inflammation status of the uterus (2) would be useful.

Occurrence of displaced abomasum was associated with large significant losses in CUM21 (27.3%) and CUM22–49 (16.2%). All cases were diagnosed in DIM2–26, indicating that these direct production losses occurred in the period of diagnosis and treatment. Previous studies (3, 11, 22) found displaced abomasum to be associated with lower 305-d yield. Displaced abomasum was not associated with CUM119 ($P>.05$), but when entered in the final model on CUM119, a nonsignificant ($P = .18$) loss was noted in production of 378.4 kg (7.4%), similar to the losses in CUM21 plus CUM22–49.

Lameness was associated with a significant increase in AVG1–5 (6.3%) and CUM21 (5.0%). Rowlands and Lucey (28) found PEAK to be higher in cows with lesions of the interdigital cleft and the sole or white line. In our study, lameness was mostly due to sole and white line lesions. The positive association of limping DIM1–49 with early postpartum yield suggests that high production was a risk factor for lameness. Higher production is associated with higher feed intake, which might increase the risk of rumen acidosis and cause limping as a result of the histamine release (1). Increased feed intake may also result in more time spent walking and standing and increased risk of feet problems. CUM22–49 was not associated with limping DIM1–49, but CUM50–119 coincided with a 3.9% yield increase. This suggests that during diagnosis and treatment of limping DIM1–49, production was reduced and increased again thereafter. Several studies (3, 23) found no significant direct relationship with 305-d yield milk yield, but Dohoo and Martin (11) reported higher average lifetime yield in cows with foot problems. In this study, limping DIM1–49 was associated with a nonsignificant (P = .12) yield increase (140.9 kg) when entered in the final model on CUM119.

Significant reductions in AVG1–5 (9.2%), CUM21 (7.3%), and CUM22–49 (6.0%) were associated with mastitis in DIM1–21. A 35.3-kg (2.9%), nonsignificant (P = .2) yield reduction was found when mastitis DIM22–49 was entered in the final model on CUM22–49. Mastitis DIM50–119 was associated with significantly lower (8.1%) CUM50–119. No indications of production decreases were found to occur outside the period in which clinical mastitis was diagnosed. This is in disagreement with Lucey et al. (19), who reported yield decreases to precede onset of clinical disease. This discrepancy in results could be due to the predominant type of microorganism involved. In our study, only bacteria classified as environmental in origin were isolated from quarters with clinical mastitis, whereas in the Lucey et al. (19) study both primary udder pathogens and environmental microorganisms were involved. Dohoo and Martin (11) reported a small beneficial effect of clinical mastitis on average lifetime production, but the benef it was accompanied by a large negative effect associated with subclinical mastitis, suggesting again that significant losses occurred outside the period in which clinical mastitis was diagnosed and treated. These authors provided no indication on the type of microorganisms involved. Other studies (3, 15, 18) reported direct negative associations between clinical mastitis and 305-d yield. We found mastitis DIM50–119 to significantly reduce CUM119.

Cows treated for ketosis DIM22–49 had a 6.1% lower CUM21. Similarly, when losses in CUM50–119 and CUM119 attributable to ketosis DIM50–119 were compared (Table 3), a 51.4-kg yield (4.1%) reduction appeared to have occurred in DIM1–49. This supports data from Lucey et al. (19) and a previous report (7) on the present data, which showed decreased milk production up to several weeks prior to treatment in cows with clinical ketosis. In agreement with other reports (19, 27) milk production was significantly decreased during episodes of clinical ketosis: 8.8% of CUM21 for ketosis DIM1–21, 12.7% of CUM22–49 for ketosis DIM22–49, and 8.2% of CUM50–119 for ketosis DIM50–119. Different clinical signs have been used in the literature to define ketosis (13). Our definition includes reduced milk yield (as perceived by the dairy operator) at the time of diagnosis. This explains at least in part the negative association between ketosis and production at the time of diagnosis. Confirmation of the present results using a definition of ketosis based on existence of ketonemia or ketolactia is therefore necessary. Despite treatment, ketosis DIM1–21 was associated with significant losses (7.5%) in CUM22–49. Only nonsignificant yield reductions in CUM50–119 were found when ketosis DIM1–21 [84.9 kg, (P = .28)] or ketosis DIM22–49 [60.1 kg, (P = .35)] were entered in the final model on CUM50–119. Such persistence of milk yield losses, following treatment, has not previously been reported. Peak yield was reduced in cows with ketosis DIM1–21. Rowlands and Lucey (28) also reported PEAK reduced in case of prior ketosis. Positive direct associations have been reported between clinical ketosis and 305-d (16, 23) or average lifetime (11) production. Rowlands and Lucey (28) reported a lack of significant difference in 305-d production in cows with ketosis due to a compensatory higher persistency later in lactation. In our study, compensation did not appear to have started by 119 DIM.

Sire PD milk was associated (P<.01) with all the milk production variables considered. Other studies have adjusted for genetic potential by comparing yield in the lactation in which the disease occurred with yield in the previous lactation (28). Our adjustment for genetic potential was incomplete because it was limited to genetic potential of the cows' sire.

CONCLUSIONS

From this and a previous report (7), we conclude that milk production in this herd was significantly reduced at the time of diagnosis and treatment of displaced abomasum and clinical mastitis. With MET1 and ketosis, daily milk yield was already reduced prior to diagnosis and remained lower thereafter. Data for this observational study were generated in a single well-managed operation with high milk production (10,900 kg DHI rolling herd average) and extensive use of disease prevention schemes. Confirmation of these results in other herds is necessary before results can be generalized.

More research is needed to determine whether milk yield measures in addition to use of available clinical signs, can improve the capability to screen for diseases. Some of these signs might also be measured automatically, e.g., elevated milk temperature as an indicator of elevated body temperature in case of metritis. For example, Schlunsen et al. (29) proposed a model to identify cows in estrus using automatically recorded data such as milk temperature, activity, and milk yield. Such models to screen for diseases also could use information on preceding occurrence or absence of occurrence of risk factors, which themselves often represent a clinical condition (4).

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