Effects of Diseases on Test Day Milk Yield and Body Weight of Dairy Cows from Danish Research Herds

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ABSTRACT

The pre- and postdisease interrelationships of energy corrected test day milk yield and body weight of dairy cows caused by mastitis, three reproductive disorders (retained placenta, metritis, cystic ovaries), and seven metabolic disorders (milk fever, ketosis, decreased rumen motility, enteritis, left displaced abomasum, right displaced abomasum, and off feed) were quantified by using mixed models analysis with repeated measures of continuous data. The data were weekly recordings from 4414 lactations collected in three Danish research herds.

High milk yield was a risk factor for ketosis and enteritis. Heavier primiparous cows were more likely to contract mastitis.

Milk yield was decreased for a disease-specific period for all study diseases except cystic ovaries and right displaced abomasum. Metabolic disorders had a detrimental effect on body weight. The highest weight loss (69 kg) was associated with left displaced abomasum. The persistence of the weight loss differed considerably among study diseases. Almost all weight loss occurred up to and including the initial week after diagnosis, which emphasized the detrimental effect of the subclinical stage. However, weekly measured body weight seemed superior to weekly energy corrected test day milk yield for disease detection only for decreased rumen motility and left displaced abomasum. This study demonstrates the importance of the predisease level for accurate estimation of the loss of milk yield and body weight from disease.
(**Key words**: disease, milk yield, body weight, dairy cows)

Abbreviation key: **ETDM** = energy corrected test day milk yield, **LDA** = left displaced abomasum, **RDA** = right displaced abomasum, **RR** = risk ratio.

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INTRODUCTION

Production diseases in cows have different consequences in a dairy herd. Two such effects are losses of BW and milk yield; the major emphasis has been on estimating the effect on milk yield. In many studies, the effect on milk yield is measured cumulatively on a lactational basis from calving until a certain lactation stage; 305-d milk yield is commonly analyzed (8, 27, 32, 35). Another approach has been to address the short-term association between disease and milk yield (4, 5, 6, 7, 22, 28). The use of the latter approach makes it possible to explore different patterns of change in daily milk yield caused by different diseases that may not be detected by 305-d milk yield. Accordingly, an apparent lack of effect from a disease on 305-d milk yield may result from a temporary drop in milk yield combined with higher milk yield before the disease or after recovery. Short-term milk losses in a dairy herd are important because of their effects on culling. Knowledge of whether the milk loss due to disease is temporary and fairly small or more sustained and larger is important to improve culling management. In the literature, some estimates of short-term losses from diseases are based on monthly recordings of test day milk yield in commercial dairy herds. Monthly measurements may not be frequent enough to permit detection of very short-term milk losses or to properly estimate milk yields in early lactation. To estimate the true loss, this predisease milk yield is needed to predict milk yield had the cow not contracted the disease and, consequently, to obtain unbiased estimates of milk loss.

An estimate of short-term milk loss caused by a disease may also aid early detection of health problems. Some health problems that might not affect daily milk yield until later in lactation may be identified earlier by use of BW measures (29). BW loss from disease may cause yield loss directly from the sale of culled cows and indirectly from increased risk of subsequent diseases and decreased milk yield. All of these effects of BW loss call for estimates of short-term associations between diseases and BW. Only a

few studies have estimated such relationships (29), probably because available data sets with frequent weighing and proper disease recordings are rare and typically too small to estimate the effect of disease on BW loss from the low incidence of some production diseases. However, these demands have been fulfilled in the weekly data recordings on trials from Danish research herds during the last decade. Consequently, the effects of diseases on daily milk yield and BW may be studied accurately in that data set if the effects of the various trials are considered.

We used weekly recordings from three Danish research herds, which were compiled over a decade; the main objectives of this study were to quantify the pre- and postdisease interrelationships of daily milk yield and BW of dairy cows for mastitis, three reproductive disorders (retained placenta, metritis, and cystic ovaries), and seven metabolic disorders (milk fever, ketosis, decreased rumen motility, enteritis, left displaced abomasum (**LDA**), right displaced abomasum (**RDA**), and off feed).

MATERIALS AND METHODS

Data

The data for this study originated from three research dairy herds of the Danish Institute of Agricultural Sciences, Research Centre, Foulum, Denmark, from April 1985 to August 1997. The data represent a variety of controlled feeding management strategies, from total mixed ration feeding to separate feeding of each feed component. The general strategy when concentrates and roughage were fed separately was to feed roughage ad libitum and concentrates according to the feeding plan, independently of test day milk yield. All three herds had tie stalls. Herd and cow characteristics (cow identity number, breed, parity, calving date, and trial history), disease data, test day milk, fat and protein yield, and BW were available. The breeds included Danish Red, Danish Black and White, Danish Jersey, Danish Red and White, and crossbred dairy cows. Disease data included the DIM of all treatments; only the first diagnosis of each disease within a lactation was used. A standard protocol with detailed disease codes was used for all disease recordings. Milk yield and BW were recorded on a regular basis (weekly) with some supplemental recordings in specific trials such as a repeated weighing the following day. The average value was used for such recordings. Daily milk yield was measured as energy corrected test day milk yield (**ETDM**), defined as milk (kg) $\times$ (0.383 $\times$ fat (%) + 0.242 $\times$ protein (%) + 0.7832) $\div$ 3.14 (39). Fat and protein in the milk was measured in conjunction with milk yield measurement.

From these data, some recordings were excluded. The ETDM and BW recordings after 168 DIM were excluded from some analyses, for several reasons. First, the typical strategy when concentrates and roughage were fed separately was to feed roughage ad libitum; concentrates were fed according to the feeding plan independently of ETDM until 168 DIM. Second, most diseases under study occurred in early or midlactation. In addition, a uniform follow-up period for all cows was achieved by the 168 DIM cutoff. To avoid exclusion of major subclinical stages, disease recordings were included for two additional weeks (cutoff at 182 DIM). The number of cows in the study fulfilling these restrictions is given in Table 1 by herd, breed, and lactation number. The numbers of different cows and lactations were 2120 and 4414, respectively.

Cows were assigned to various trials. Trials and treatment groups within trials were identified for each lactation. Several cows were assigned to more trials within the same lactation, but only a few shifted before the cutoff at 168 DIM, and, in such cases, the later trial was ignored. If, subsequently, a cow was not assigned to any trial, then a trial value specific to herd and year was assigned afterward. The resulting numbers of trials and treatment groups were 90 and 279, respectively.

The diseases chosen for study were based on the occurrence and etiology of the diseases. Only clinical diseases were included. Accordingly, the study diseases were acute clinical mastitis (excluded was mild clinical mastitis diagnosed by milk with flakes as the only clinical sign), retained placenta (treatment because of placenta not expelled normally within 24 h after calving), metritis (inflammation of the uterus diagnosed by fetid and visible discharge), cystic ovaries (a fluid-containing enlargement of the ovary diagnosed by rectal palpation), milk fever (recumbency and other clinical signs of hypocalcemia), ketosis (lack of appetite, odor of acetone on the breath, and other clinical signs of ketosis confirmed by an abnormally high concentration of ketone bodies in the blood), decreased rumen motility (reduced rumen function without diagnosis of other metabolic disorders), enteritis (inflammation of the intestine, particularly of the small intestine, diagnosed primarily by diarrhea), LDA (abomasum enlarged with fluid or gas that is mechanically trapped in the left side of the abdominal cavity), RDA (like LDA but in the right side), and off feed (reduced feed intake without sign of other diseases).

Study Design

Disease frequency and distribution according to DIM of first diagnosis for each study disease were

TABLE 1. Number of cows under study by herd, breed, and parity.[1]

Breed	Parity	Number of lactations			
		Herd 1	Herd 2	Herd 3	Total
Danish Red	1	247	52		299
	2	93	39		132
	>2	38	34		72
Danish Black and White	1	236	1051	231	1518
	2	108	671	189	968
	>2	55	689	201	945
Danish Jersey	1	145			145
	2	50			50
	>2	28			28
Danish Red and White	1	34			14
	2	13			13
Crossbreed	1		101	1	102
	2		76		76
	>2		52		52
All breeds	All	1027	2765	622	4414

[1]The total number of cows and lactations was 2120 and 4414, respectively.

analyzed using descriptive statistics. Relative risks were studied by using survival analysis separately for all recorded diseases. Observations were censored [i.e., coded to reflect that the event (disease) did not occur during the study period (168 + 14 DIM)] at 182 DIM if the disease did not occur during the study period.

We assessed the relationships among ETDM, BW, and health by using observations correlated in time. The inclusion of repeated measures, rather than a single measure, allows one to study relationships between ETDM and disease according to the lactation curve and date of disease. Furthermore, relationships between BW and disease according to the BW curve can be studied by using repeated measures. To differentiate between cows without disease, cows having a disease other than the specific study disease, and cows having the specific study disease, we created a disease index for each week in milk collected. One category included cows that were free of any disease during the whole study period (until 182 DIM). Cows with a disease other than the one under study were divided into three categories according to week relative to week in lactation of the first disease diagnosis within the current lactation. These three categories were from calving to 1 wk before diagnosis, from 1 wk before diagnosis to 3 wk after diagnosis, and, finally, later than 4 wk after diagnosis. The remaining 11 categories defined the week in milk relative to the week in milk of the diagnosis of the study disease, which were ≤–4, –3, –2, –1, 0, 1, 2, 3, 4, 5, and ≥6. If a cow had the study disease, it was included in one of these latter 11 categories. For example, the –1 category refers to the week before diagnosis (i.e., recordings from 1 to 7 d before the diagnosis). Category 0 includes 0 to 6 d after diagnosis (the first week after diagnosis), and category 1 includes 7 to 13 d after diagnosis (second week after diagnosis).

Statistical Model and Method

Relative risks from exposure diseases were studied by using survival analysis separately for all recorded diseases based on the Cox proportional hazards model (semiparametric). This model is based on the order in which the events happen, not the exact times of occurrence. Only the hazard ratio, a measure analogous to the relative risk, between subjects can be estimated and compared. The model used was

$$h_i(t) = h_0(t) \times \exp(\mathbf{z}_i'\beta) \quad [1]$$

where $h_i(t)$ = hazard function assumed to describe the survival time of each observation, t = time of DIM, $h_0(t)$ = baseline hazard that is not specified because it is canceled out in the Cox model, β = vector of unknown regression parameters associated with the explanatory variables, and $\mathbf{z}_i$ = vector of measured explanatory variables for the individual i. These measured explanatory variables are the occurrence of study disease and of exposure disease; 0 and 1 represented absence and presence, respectively. Exposure disease occurring after the study disease was not modeled as a risk factor. Observations were censored at the end of the study period (182 DIM) if the study disease did not occur during the study period. Proportionality over time of the hazard of exposed and unexposed cows was assumed. Using the SAS PHREG (36) procedure, the model was fitted for each

combination of an exposure disease and a study disease if there were more than four lactations with an exposure disease prior to the study disease.

The relationships among ETDM, BW, and health were studied using mixed models analysis with repeated measures of continuous data. A separate analysis was fitted for ETDM and BW, respectively, within each of the study diseases. Primiparous cows were studied separately because of different lactation and BW curves. The patterns of change in ETDM and BW, respectively, associated with each study disease were investigated with the following model:

$$\mathbf{y} = \mathbf{X}\beta + \mathbf{Z}\gamma + \mathbf{e} \quad [2]$$

where $\mathbf{y}$ = vector representing the response variable of ETDM and BW, respectively, and β = vector of fixed effects of herd (three categories), breed (two categories: Jersey and others), parity (two categories in multiparous models: parity 2 and parity >2), lactation stage (three continuous covariates of week in milk), calving season (four categories), and disease index (15 categories). To represent the shape of the lactation curve and the BW curve, week in milk was expressed linearly, quadratically, and cubically using three continuous covariates. The 15 categories of the disease index included cows that were free of disease (one category), cows having diseases other than the study disease (three categories), and cows having the study disease (11 categories). γ = unknown vector of random effects, and $\mathbf{e}$ = unknown random error vector the elements of which are not required to be independent and homogeneous because of the random effects. $\mathbf{X}$ and $\mathbf{Z}$ = known design matrices relating the $\mathbf{y}$ vector to the β and γ vectors, respectively. The random vectors γ and $\mathbf{e}$ were normally distributed with null means and were uncorrelated. The variation in mean response among cows was accounted for by $\text{var}(\gamma) = \mathbf{I}(\sigma_\gamma)^2$. The $(\sigma_\gamma)^2$ term is the variance of the nested treatment group within trial effects.

The variation in repeated measures within cows was given by $\text{var}(\mathbf{e}) = \mathbf{R}(\sigma_e)^2$. The $(\sigma_e)^2$ is the error variance. $\mathbf{R}$ = covariance matrix that gives the autocorrelation coefficients between elements of the $\mathbf{e}$ vector. A first-order autoregressive covariance structure of $\mathbf{R}$ was used. The random effect makes the model subject specific, because it contains random terms for each treatment group nested within trial, as opposed to models in which the study population is averaged. In the subject-specific model, estimated effects describe the effect for individual treatment groups and trials; in the model that averaged the study population the effect for the entire study population is described. The effect of herd was not modeled as random because of the low number of herds.

The principles of model selection were to exclude nonsignificant effects except for parity, which is a general known confounder. The models were fitted using the SAS MIXED procedure (36) with the REML method for estimating the covariance parameters. However, the maximum likelihood method and the minimum variance quadratic unbiased method were used if there were convergence problems when REML was used.

RESULTS

Descriptive Statistics

The disease frequency and distribution according to DIM of first diagnosis for each study disease are given in Table 2. The lactational distribution of diseases is generally not normal. Therefore, the median DIM and its semi-interquartile range (range between third and first quartile of occurrence of that disease, divided by 2) were calculated.

Results from Model [1]

Risk ratios (**RR**) were estimated by survival analysis (Model [1]). The risk of acute clinical mastitis was increased by milk fever (RR = 1.5 [1.2; 1.8]). Numbers in square brackets are the 95% confidence interval. Teat injury also increased the risk of acute clinical mastitis. The risk of metritis was increased by retained placenta (RR = 1.5 [1.2; 1.8]) and dystocia (RR = 11.4 [5.3; 24.2]). The risk of ketosis was increased by milk fever (RR = 2.8 [2.0; 4.1]. The risk of decreased rumen motility was increased by retained placenta (RR = 1.8 [1.2; 2.8]) and milk fever (RR = 3.7 [2.3; 5.8]). The risk of LDA was increased by metritis (RR = 2.9 [1.1; 7.3]), milk fever (RR = 3.3 [1.4; 7.9]), ketosis (RR = 4.3 [2.0; 8.9]), and decreased rumen motility (RR = 6.2 [2.8; 14.0]). Some significant protective effects were found, the majority involving mastitis and reproductive disorders.

Results from Model [2]

In Model [2], the fixed effects of herd and calving season were generally small. Because of convergence problems, the fixed effects of herd and calving season were excluded from all the final models of the effect of study disease on ETDM and BW (Model [2]).

The effects on ETDM and BW estimated by Model [2] for primiparous and multiparous cows separately are presented for acute clinical mastitis (Table 3), retained placenta (Table 4), metritis (Table 5), milk fever (Table 6), ketosis (Table 7), decreased rumen motility (Table 8), enteritis (Table 9), LDA (Table

TABLE 2. Frequency, cumulative incidence, and distribution according to DIM of first diagnosis for each study disease.[1]

Study disease	Parity	Frequency	Incidence	Mean DIM	Median DIM	SIR[2]
Acute clinical mastitis	1	486	0.234	44	9	39.0
	>1	767	0.328	51	37	43.0
Retained placenta	1	191	0.092	1	1	0.5
	>1	287	0.123	1	1	0.5
Metritis	1	129	0.062	19	8	5.5
	>1	74	0.032	29	11	16.0
Cystic ovaries	1	196	0.094	76	74	24.5
	>1	246	0.105	73	68	34.5
Milk fever	1	6	0.003	1	1	0.5
	>1	208	0.089	1	1	0.5
Ketosis	1	42	0.020	34	28	14.0
	>1	233	0.100	27	24	9.0
Decreased rumen motility	1	43	0.021	45	22	30.5
	>1	102	0.044	49	22	43.0
Enteritis	1	89	0.043	61	40	47.5
	>1	161	0.069	48	22	32.0
LDA[3]	1	13	0.006	38	20	16.0
	>1	29	0.012	19	17	8.0
RDA[3]	1	2	0.001	66	66	59.5
	>1	3	0.001	64	50	65.0
Off feed	1	15	0.007	46	11	38.0
	>1	13	0.006	48	22	39.0

[1]Data include 2078, 1239, and 1097 lactations from parities 1, 2, and >2, respectively. Observations later than 182 DIM were excluded.

[2]Semi-interquartile range is calculated as the range between the third and the first quartile of occurrence of the disease divided by two.

[3]LDA = Left displaced abomasum; RDA = right displaced abomasum.

10), and off feed (Table 11). For cystic ovaries and RDA, no significant effect of disease was found.

The estimates in Tables 3 through 11 are relative to baseline estimates of cows without diseases, which are the same cows, regardless of the study disease. Because these baseline estimates are alike, they are only presented in the following text. The estimated average ETDM for non-Jersey cows without disease was 23.8 kg (SE = 0.4). The effect of the Jersey breed was –1.8 kg (SE = 0.3). In the multiparous model, the estimated average ETDM for non-Jersey cows with parity >2 and without disease was 31.2 kg (SE = 0.5). The effect of the Jersey breed was –4.3 kg (SE = 0.5). The effect of parity 2 was –1.7 kg (SE = 0.2). The estimated BW at calving for non-Jersey cows without disease was 505.5 kg (SE = 5.4). The effect of Jersey breed was –157.4 kg (SE = 4.9). In the multiparous model, the estimated BW at calving for non-Jersey cows with parity higher than 2 and without disease was 631.3 kg (SE = 7.0). The effect of Jersey breed was –183.1 kg (SE = 6.4). The effect of parity 2 was –48.7 kg (SE = 2.3).

The estimates of the three disease categories for cows having diseases other than the study disease were alike among the study diseases in Tables 3 through 11 and are summarized in the following text only. Both ETDM and BW were generally higher in the predisease category of cows having other diseases. For this category, earlier than 1 wk before the diagnosis of other diseases, ETDM estimates were 0.4 to 0.5 (SE = 0.2) and 0.7 to 1.0 (SE = 0.2 to 0.3) for primiparous and multiparous cows, respectively. These estimates for BW, which were generally not significant, were 1.6 to 5.0 (SE = 2.0 to 2.4) and 1.8 to 3.5 (SE = 2.6 to 2.9) for primiparous and multiparous cows, respectively. In the succeeding category, from 1 wk before to 3 wk after diagnosis of other diseases, ETDM and BW were generally lower than for cows without disease. For this latter category, ETDM estimates were –0.4 to –0.1 (SE = 0.1 to 0.2) and –0.7 to –0.6 (SE = 0.2) for primiparous and multiparous cows, respectively. These estimates for BW, which were generally not significant for primiparous cows, were –4.8 to –0.2

TABLE 3. Effect of acute clinical mastitis on energy corrected test day milk yield (ETDM) and BW in the weeks preceding and succeeding the day of disease diagnosis estimated by Model [2].

Week after diagnosis	ETDM, kg				BW, kg			
	Primiparous		Multiparous		Primiparous		Multiparous	
	β	SE	β	SE	β	SE	β	SE
≤–4	0.1^{NS}	0.3	1.1***	0.3	10.0***	3.0	3.1^{NS}	3.2
–3	0.0^{NS}	0.3	0.6^{NS}	0.3	10.5	3.0	4.4^{NS}	3.1
–2			0.7*	0.3	9.8***	2.9		
–1	–1.0***	0.3	–0.1^{NS}	0.3	6.6*	2.9	1.4^{NS}	3.1
0	–2.5***	0.2	–2.3***	0.3	3.8^{NS}	2.8	0.0^{NS}	3.1
1	–0.9***	0.2	–0.7*	0.3	3.1^{NS}	2.8	–2.2^{NS}	3.1
2			–0.8***	0.3	2.2^{NS}	2.8		
3			–1.3***	0.3	3.9^{NS}	2.8		
4	–1.1***	0.2	–1.8***	0.3	4.6^{NS}	2.8	–0.1^{NS}	3.1
5			–1.6***	0.3	7.1*	2.9		
≥6	–1.3***	0.2	–1.8***	0.2	7.7**	2.8	2.1^{NS}	3.1

*P < 0.05.

**P < 0.01.

***P < 0.001.

NS = Not significant.

(SE = 1.6 to 2.3) and –7.9 to –3.8 (SE = 2.5 to 2.8) for primiparous and multiparous cows, respectively. In the final category from later than 3 wk after diagnosis of 'other diseases', ETDM was generally lower than cows without any disease. For this category, ETDM estimates were –0.7 to –0.3 (SE = 0.1 to 0.2) and –0.7 to –1.0 (SE = 0.2) for primiparous and multiparous cows, respectively. These estimates for BW, which were generally not significant, were –0.7 to 3.8 (SE = 1.5 to 2.3) and –5.1 to 0.4 (SE = 2.5 to 2.8) for primiparous and multiparous cows, respectively.

The effect of the study disease on ETDM for primiparous and multiparous cows was lower ETDM compared with that of healthy cows for a disease-specific number of weeks after the diagnosis of each study disease except for metritis (primiparous), cystic ovaries, and RDA. Cows contracting acute clinical mastitis (multiparous; Table 3), ketosis (Table 7), and enteritis (Table 9) had significantly higher milk yield before the disease that did healthy cows. The

TABLE 4. Effect of retained placenta on energy corrected test day milk yield (ETDM) and body weight in the weeks preceding and succeeding the day of disease diagnosis estimated by Model [2].

Week after diagnosis	ETDM, kg				BW, kg			
	Primiparous		Multiparous		Primiparous		Multiparous	
	β	SE	β	SE	β	SE	β	SE
0	–2.4***	0.4	–5.7***	0.5	0.7^{NS}	3.8	–8.9*	4.1
1	0.0^{NS}	0.4	–1.0*	0.5	–7.6*	3.9	–12.5**	4.2
2	0.6^{NS}	0.4	0.2^{NS}	0.4	–3.7^{NS}	3.8	–8.9*	4.1
3	0.4^{NS}	0.3	0.3^{NS}	0.4	–5.4^{NS}	3.9	–9.1*	4.2
4	0.3^{NS}	0.3	–0.6^{NS}	0.4	2.0^{NS}	3.8	–2.8^{NS}	4.1
5	–0.4^{NS}	0.3	–0.3^{NS}	0.4	2.2^{NS}	3.9	0.1^{NS}	4.1
≥6	–0.7**	0.3	–0.7*	0.3	6.3^{NS}	3.7	4.9^{NS}	4.0

*P < 0.05.

**P < 0.01.

***P < 0.001.

NS = Not significant.

TABLE 5. Effect of metritis on energy corrected test day milk yield (ETDM) and BW in weeks preceding and succeeding the day of disease diagnosis estimated by Model [2].

Week after diagnosis	ETDM, kg				BW, kg			
	Primiparous		Multiparous		Primiparous		Multiparous	
	β	SE	β	SE	β	SE	β	SE
≤–4	–0.6^{NS}	0.7	–0.8^{NS}	1.0	5.7^{NS}	5.6	3.9^{NS}	8.3
–3	–1.3*	0.6	–2.7*	1.1	0.5^{NS}	5.3	–0.1^{NS}	7.5
–2			–4.8***	1.0	6.6^{NS}	4.8		
–1	–0.5^{NS}	0.5	–5.4***	0.9	–4.9^{NS}	4.6	–11.8^{NS}	7.4
0	–0.3^{NS}	0.4	–4.9***	0.9	–14.8**	4.6	–11.9^{NS}	7.5
1	0.0^{NS}	0.4	–3.5***	0.8	–13.8**	4.6	–8.3^{NS}	7.2
2			–3.8***	0.8	–17.8***	4.6		
3	–0.3^{NS}	0.3	–2.9***	0.8	–11.8**	4.6	–6.6^{NS}	7.2
4			–3.0***	0.8	–10.2*	4.6		
5			–3.2***	0.7	–9.8*	4.6		
≥6	–0.9**	0.3	–1.9***	0.6	–8.2^{NS}	4.5	1.9^{NS}	7.2

*P < 0.05.
**P < 0.01.
***P < 0.001.
NS = Not significant.

opposite situation of lower milk yield before the disease was found only for multiparous cows contracting LDA (Table 10).

The body weight of cows with retained placenta, metritis (primiparous), ketosis, decreased rumen motility (multiparous), enteritis (multiparous), and LDA was lower after diagnosis than was that of healthy cows. Primiparous cows contracting acute clinical mastitis had significantly higher BW in all predisease categories than did healthy cows. Based on the few nonparturient milk fever cases, multiparous

TABLE 6. Effect of milk fever on energy corrected test day milk yield (ETDM) and BW in the weeks preceding and succeeding the day of disease diagnosis estimated by Model [2].

Week after diagnosis	ETDM, kg				BW, kg			
	Primiparous		Multiparous		Primiparous		Multiparous	
	β	SE	β	SE	β	SE	β	SE
–1	NED[1]		NED		NED		11.9^{NS}	6.8
0	10.7	2.0	–0.6^{NS}	0.7	80.8***	19.7	1.7^{NS}	5.2
1	1.4^{NS}	1.6	–0.4^{NS}	0.6	NED		–0.0^{NS}	5.2
2			–0.3^{NS}	0.6	68.3**	20.7	–1.7^{NS}	5.2
3	–1.9^{NS}	1.5	–1.1*	0.6	80.1***	21.6	–6.5^{NS}	5.2
4			–0.3^{NS}	0.5	55.2**	20.2	–4.2^{NS}	5.1
5			–1.2*	0.5	81.5***	21.6	–2.4^{NS}	5.1
≥6	–4.49***	1.3	–1.5***	0.4	54.5**	19.7	3.2^{NS}	4.8

[1]Not enough data.
*P < 0.05.
**P < 0.01.
***P < 0.001.
NS = not significant.

TABLE 7. Effect of ketosis on energy corrected test day milk yield (ETDM) and BW in the weeks preceding and succeeding the day of disease diagnosis estimated by Model [2].

Week after diagnosis	ETDM, kg				BW, kg			
	Primiparous		Multiparous		Primiparous		Multiparous	
	β	SE	β	SE	β	SE	β	SE
≤–4	1.5^{NS}	0.8	2.5***	0.6	–5.0^{NS}	7.9	5.2^{NS}	4.9
–3	1.8**	0.7	2.4***	0.5	–8.7^{NS}	8.0	0.8^{NS}	4.8
–2					–7.6^{NS}	7.7	–4.0^{NS}	4.9
–1	0.8^{NS}	0.7	–0.2^{NS}	0.5	–29.8***	7.7	–13.4**	4.7
0	–3.1***	0.7	–4.2***	0.5	–32.5***	7.7	–30.5***	4.8
1	–0.3^{NS}	0.6	–1.7***	0.4	–26.7***	7.7	–25.0***	4.8
2					–29.1***	7.7	–27.7***	4.9
3	–1.0^{NS}	0.6	–1.5***	0.4	–31.3***	7.8	–27.6***	4.8
4					–27.6***	7.8	–24.4***	4.8
5					–28.7***	7.9	–25.0***	4.8
≥6	–0.3^{NS}	0.5	–0.6^{NS}	0.4	–31.0***	7.6	–25.0***	4.6

*$P < 0.05$.

**$P < 0.01$.

***$P < 0.001$.

NS = Not significant.

TABLE 8. Effect of decreased rumen motility on energy corrected test day milk yield (ETDM) and BW in the weeks preceding and succeeding the day of disease diagnosis estimated by Model [2].

Week after diagnosis	ETDM, kg				BW, kg			
	Primiparous		Multiparous		Primiparous		Multiparous	
	β	SE	β	SE	β	SE	β	SE
≤–4	0.1^{NS}	0.8	–0.4^{NS}	0.7	11.5^{NS}	8.0	2.9^{NS}	7.0
–3	0.4^{NS}	0.8	–0.1^{NS}	0.7	10.3^{NS}	8.1	–3.2^{NS}	6.9
–2	1.1^{NS}	0.8			8.9^{NS}	7.8	–14.3*	6.8
–1	–0.1^{NS}	0.8	0.3^{NS}	0.7	–4.3^{NS}	7.7	–12.3^{NS}	6.8
0	–3.2***	0.7	–4.2***	0.7	–12.7^{NS}	7.7	–34.7***	6.9
1	–2.3**	0.7	–2.5***	0.6	–12.7^{NS}	7.7	–25.9***	7.0
2	–2.5***	0.7			–7.6^{NS}	7.8	–27.9***	7.0
3	–2.3**	0.7			–9.9^{NS}	7.8	–25.4***	7.2
4	–2.9***	0.7	–1.6**	0.6	–5.1^{NS}	7.8	–20.7**	7.1
5	–2.5***	0.6			–4.4^{NS}	7.9	–22.0**	7.3
≥6	–2.7***	0.6	–1.1*	0.5	–4.3^{NS}	7.6	–18.9**	6.8

*$P < 0.05$.

**$P < 0.01$.

***$P < 0.001$.

NS = Not significant.

TABLE 9. Effect of enteritis on energy corrected test day milk yield (ETDM) and BW in the weeks preceding and succeeding the day of disease diagnosis estimated by Model [2].

Week after diagnosis	ETDM, kg				BW, kg			
	Primiparous		Multiparous		Primiparous		Multiparous	
	β	SE	β	SE	β	SE	β	SE
≤–4	0.5^{NS}	0.5	2.0***	0.6	1.1^{NS}	5.0	–0.9^{NS}	5.3
–3	1.0*	0.5	2.4***	0.6	1.9^{NS}	6.4	–0.8^{NS}	5.1
–2			2.5***	0.6	6.1^{NS}	6.1		
–1	1.1*	0.5	1.2*	0.6	7.0^{NS}	5.8	–5.7^{NS}	5.1
0	–2.2***	0.5	–2.4***	0.5	–5.6^{NS}	6.2	–20.7***	5.2
1	–0.5^{NS}	0.4	–1.3*	0.5	–9.7^{NS}	6.1	–14.5**	5.0
2			–0.8^{NS}	0.5	–1.8^{NS}	7.3		
3	0.0^{NS}	0.4	–0.6^{NS}	0.5	–5.4^{NS}	6.5	–10.0*	5.0
4			–0.7^{NS}	0.5	6.1^{NS}	6.9		
5			0.0^{NS}	0.5	–0.7^{NS}	6.1		
≥6	0.3^{NS}	0.4	–0.4^{NS}	0.4	3.7^{NS}	4.6	–5.5^{NS}	5.1

*P < 0.05.
**P < 0.01.
***P < 0.001.
NS = Not significant.

cows contracting milk fever had significantly higher BW before the disease than did healthy cows. Cows contracting ketosis, decreased rumen motility, or LDA (multiparous) had significantly lower BW in wk 1 or 2 before the disease than did healthy cows, but no significant effect was found before that. For the remaining situations, no significant effect on BW before diagnosis of a study disease compared with healthy cows was found.

DISCUSSION

The data included five different breeds; Danish Black and White was the major breed (Table 1). The

TABLE 10. Effect of LDA on test day milk yield and body weight in weeks preceding and succeeding the day of disease diagnosis estimated by Model [2].

Week after diagnosis	Test day milk yield				BW			
	Primiparous		Multiparous		Primiparous		Multiparous	
	β	SE	β	SE	β	SE	β	SE
≤–4	–1.5^{NS}	1.5	–3.1^{NS}	2.0	8.3^{NS}	14.2	–4.9^{NS}	12.7
–3	–0.0^{NS}	1.6	–4.3*	1.7	14.2^{NS}	13.3	7.2^{NS}	11.8
–2	2.7^{NS}	1.4	–3.7*	1.5			–32.0**	11.8
–1	0.5^{NS}	1.3	–4.8***	1.4	–16.0^{NS}	13.6	–28.7***	11.3
0	–9.1***	1.3	–7.8***	1.2	–22.8^{NS}	13.3	–73.7*	11.4
1	–5.7***	1.3	–6.8***	1.2	–30.2*	13.3	–51.7***	11.3
2	–4.1**	1.3	–5.0***	1.2			–62.5***	11.4
3	–4.4***	1.3	–3.9**	1.2			–53.4***	11.3
4	–3.5**	1.4	–3.0*	1.2	–27.5*	13.5	–50.1***	11.9
5	–4.7***	1.3	–1.9^{NS}	1.1			–43.6***	11.4
≥6	–4.7***	1.2	–1.3^{NS}	0.9	–34.2*	13.7	–38.1***	11.2

*P < 0.05.
**P < 0.01.
***P < 0.001.
NS = Not significant.

TABLE 11. Effect of off feed on energy corrected test day milk yield (ETDM) and BW in the weeks preceding and succeeding the day of disease diagnosis estimated by Model [2].

Week after diagnosis	ETDM, kg				BW, kg			
	Primiparous		Multiparous		Primiparous		Multiparous	
	β	SE	β	SE	β	SE	β	SE
≤–4	–1.5^{NS}	1.4	–2.3^{NS}	1.9	21.1^{NS}	13.6	2.4^{NS}	17.5
–3	–1.4^{NS}	1.5	–3.4^{NS}	1.9	21.6^{NS}	14.7	11.7^{NS}	16.6
–2	–2.8^{NS}	1.4	–2.9^{NS}	2.1	18.5^{NS}	13.3	–7.5^{NS}	16.8
–1	–1.8^{NS}	1.3	–4.1*	1.8	15.8^{NS}	13.5	–8.2^{NS}	16.4
0	–3.3**	1.2	–7.2***	1.9	1.1^{NS}	12.5	–24.4^{NS}	16.4
1	–1.7^{NS}	1.2	–5.9***	1.8	2.7^{NS}	13.4	–13.7^{NS}	16.3
2	–2.6*	1.2	–6.4***	1.9	6.0^{NS}	13.0	–12.9^{NS}	16.4
3	–1.7^{NS}	1.2	–5.7**	1.8	3.6^{NS}	13.8	–6.7^{NS}	16.6
4	–1.4^{NS}	1.1	–4.3*	1.9	12.1^{NS}	12.8	–9.3^{NS}	16.6
5	–2.9**	1.1	–3.6*	1.8	6.6^{NS}	13.4	1.2^{NS}	16.5
≥6	–2.1*	0.9	–1.8^{NS}	1.5	11.6^{NS}	12.4	1.0^{NS}	16.5

*$P < 0.05$.

**$P < 0.01$.

***$P < 0.001$.

NS = Not significant.

lactational incidence rates in Table 2 are in general agreement with results of other studies for clinical mastitis (3, 5, 26, 28), retained placenta (3, 5, 10, 16, 23, 24, 30), metritis (3, 5, 10, 16), cystic ovaries (10, 16), milk fever (5, 7, 10), displaced abomasum (LDA and RDA) (38), and ketosis (7, 10, 20, 16, 23). Some studies reported higher incidences for metritis (23, 30), milk fever (23), and ketosis (5). Some studies reported lower incidences for clinical mastitis (10, 16, 22), milk fever (16), and ketosis (30). For RDA and off feed, we found no difference in incidence for primiparous versus multiparous cows. However, for all other diseases, incidences were higher for multiparous cows than for primiparous cows, which generally agrees with results of other studies (14, 30). The diagnosis of enteritis (inflammation of the intestine, particularly of the small intestine), paralysis of the rumen (reduced rumen function), or off feed (reduced feed intake) refers to symptoms rather than to specific diseases. Consequently, comparisons with other studies are difficult. However, comparison may be made with the study of Dohoo et al. (10), who categorized miscellaneous digestive tract disorders, which had a lactational incidence rate of 2.4% and median days to first diagnosis of 76 d. Rumen acidosis may be one of the underlying diseases of enteritis and rumen paralysis diagnosis.

The median DIM of first occurrence of each study disease in Table 2 are similar to those reported in the studies just cited. Consequently, the bias from loss of follow-up from the cutoff at 182 DIM seems unimportant. Within disease interrelationships, our findings that retained placenta and milk fever were risk factors for other diseases agree with results of most other studies (3, 9, 14, 19, 23). Overall, the incidence, lactational distribution of the diseases, and disease interrelationships in the three herds studied did not differ significantly from general findings in other studies.

Herd effects have been suggested to be important with regard to disease (8, 12, 23, 33). Our study included only three research herds. Within these three herds, no significant herd effects were found, probably because the management of these three research herds was more alike than for herds in general. The three study herds were similar to commercial herds regarding breed and management. However, the attention paid to disease and number of treatments may have been relatively high, which would tend to underestimate relationships because better treatment of diseases may reduce the effects of the diseases on production.

The disease index made it possible to estimate weekly ETDM loss and weekly BW change before and after diagnosis of each study disease and to compare those estimates with values for baseline cows. Our estimates are average losses and do not consider when a disease actually occurred. However, the independence of DIM could be questioned for diseases with widely distributed occurrences such as mastitis (1, 5, 25) and metritis (8). The potential of losing more milk from higher yielding cows, as demonstrated by the effect of 305-d milk yield of previous lactation on LDA (6) and milk yield at disease onset for mastitis (25), was not addressed in our study because of the limited data.

The baseline cows were free of diseases. In some cases, however, different baseline cows might have been more appropriate for estimating only the direct effect of the study disease. If the predisease level of ETDM or BW is a risk factor for the study disease, baseline cows should be adjusted toward this predisease level. However, this adjustment may be complicated by the effect of subclinical stage and study diseases occurring soon after calving. This latter fact is most obvious for retained placenta and milk fever because there are no milk yield measurements before these diseases occur. For diseases occurring sufficiently late after calving, it may be relevant to adjust to an above-zero estimate of the first disease index category because the potential bias from an early subclinical level is toward zero, which is more accurate than using zero itself. If the first predisease estimates are below zero, adjustment should be made only if the level of the first estimates appears stable, indicating a true below zero predisease level rather than the effect of early subclinical stage.

Additionally, the disease interrelationships address the question of whether to use cows without the study disease rather than cows without any diseases as the baseline, especially for addressing the relationships between effects from different categories within the disease index. From these estimates of the three disease categories, which includes cows with diseases other than the study disease, the magnitude of the difference can be found, and the study disease effects can be adjusted according to different stages of other diseases. Because of the potential bias from diseases that are not associated with the study disease, we decided to use cows that were free of any diseases as the baseline.

Mastitis

For all nine mastitis cases studied, Maltz et al. (29) found severe decreases in BW that preceded a drop in milk yield. Compared with the BW >3 wk before diagnosis, our results also showed that mastitis caused BW to decrease, but only by <8 kg; the loss was only temporary, and recovery occurred about 5 wk after diagnosis (Table 3). Both this recovery and reduced milk yield could explain some milk loss. There is no simple explanation for why mastitic primiparous cows were heavier before mastitis than healthy cows, unless heavier cows produce more milk and are more susceptible to mastitis. Perhaps fat cows have decreased immune responses in the mammary gland than cows of average condition.

The higher risk of mastitis for higher yielding cows, as indicated for multiparous cows in our study, agrees with results of some studies (11, 18, 16, 22, 26) but not of others (5, 13). A comparison of the estimate of ETDM >3 wk before diagnosis of mastitis with the succeeding nine estimates shows an aggregated milk loss of 65 kg for primiparous cows and 117 kg for multiparous cows. These losses underestimate the true losses because they do not include the loss occurring >5 wk after diagnosis. Of these 9-wk losses, 57 (1.4 kg/d) and 103 kg (2.5 kg/d), respectively, were from the 6-wk period after diagnosis. The magnitude of the drop in milk yield caused by mastitis is within the range found in the literature (1, 5, 8, 22, 25, 27, 28). The wide range of estimates in the literature may be due to differing estimation methods and origin of data (37). Within estimation methods, control cows were healthy cows (25), cows without mastitis (5, 22, 27), or cows at production levels preceding mastitis (1, 28). Some studies (8, 27) estimated only 305-d milk loss. In a study (1) using predisease milk yield to estimate temporary milk loss, mastitis occurring before 150 DIM caused a 113-kg (1.9-kg/d) loss within the first 60 d after diagnosis. Our results of depressed milk yield for the remainder of the lactation caused by mastitis is supported in some short-term analysis (25), but not in others (5, 22). Recurrence of mastitis within the same lactation (9, 34) could explain some of the persistence of the milk drop; in this study, we addressed only the first mastitis diagnosis. A tendency to a more temporary loss pattern during lactation has been suggested (5, 25). The diagnosis of acute clinical mastitis covers a variety of agents. Bartlett et al. (1) isolated these agents from clinical cases and found no strong association with milk yield in the 60 d following clinical onset, but other studies (27) found contradictory results. Still, the average loss from mastitis may cover a variety of loss patterns (25). Our finding of higher loss from mastitis in multiparous cows is supported by Bartlett et al. (1). Similar to results of some other studies (1, 25, 28), our results suggest a potential of ETDM detection of subclinical mastitis the week before its clinical appearance.

Reproductive Disorders

Within the reproductive disorders, BW decreased as a result of retained placenta (Table 4) and metritis (Table 5), but not from cystic ovaries. Because retained placenta and some cases of metritis occur very early after calving, we have no clear estimate of the BW prior to disease diagnosis. A high body condition score at calving has been found to have a protective effect on the incidence of retained placenta and metritis (30). This relationship agrees with our estimates of lower BW in the first week after diagnosis of retained placenta, but no proper comparison can be made with metritis because of its occurrence some days after calving. Primiparous cows with retained

placenta lost 8 kg of BW with recovery within a few weeks. The BW loss from retained placenta, compared with that of healthy cows, could be explained by the lower BW at calving, which is a risk factor for retained placenta for multiparous cows. Compared with the BW >3 wk before diagnosis, metritis caused a temporary BW loss of 23 (primiparous) and 16 kg (multiparous), and some weeks of recovery were needed before BW was regained. Compared with healthy cows, the loss because of metritis was smaller and nonsignificant for multiparous cows. In the literature, we found no estimates of BW loss from reproductive disorders.

High milk yield has generally not been found to be a risk factor for reproductive disorders (9, 13, 16), but Deluyker et al. (5) found increased risks of metritis and retained placenta from lower milk yield from 1 to 5 DIM.

We found no effect of cystic ovaries on ETDM. Previous studies have estimated increased milk yield associated with cystic ovaries (2, 8, 15), but a 2.4% detrimental effect was found when yield per day of life was used instead of 305-d milk yield (8).

For retained placenta (Table 4), ETDM decreased only temporarily compared with that of healthy cows. For primiparous cows, the ETDM was decreased only for the first week after diagnosis, causing a milk loss of 17 kg (2.4 kg/d). For multiparous cows, the ETDM was decreased the first and second week after diagnosis causing an aggregated milk loss of 47 kg (3.4 kg/d). A temporary milk loss has been found previously (28) but not consistently (5). For a 305-d milk loss, estimates vary from a 7% persistent loss (35) to 0.4% loss (8). The latter included retained placentas that did not require veterinary attention. In a review (24), milk loss from retained placenta was suggested to be evident only at the herd level and not at the cow level.

For metritis, we found no loss in milk yield for primiparous cows, either when comparing them with healthy cows or ETDM >3 wk before diagnosis. Compared with healthy cows, multiparous cows had an extended period, beginning 3 wk before diagnosis, of decreased milk yield. A comparison of the estimate of ETDM >3 wk before diagnosis of metritis with the succeeding nine estimates yielded an aggregated milk loss of 192 kg, of which 74 kg was lost before diagnosis. Compared with healthy cows, these losses were 239 (5.7 kg/d) and 90 kg, respectively, and underestimated the true losses because the loss occurring >5 wk after diagnosis was not included. In a previous study (5), metritis occurring <22 DIM decreased milk yield by 112 kg (2.2 kg/d) from calving until 49 DIM, compared with yield of cows without metritis. In that study (5), the loss was not differentiated between primiparous and multiparous cows, which may explain some of the difference compared with the high losses we found for multiparous cows with metritis. Other studies (28, 35) found no loss of milk yield from metritis. By inclusion of DIM of diagnosis, an increasing detrimental effect of metritis from 0 to 4.6% loss for 305-d milk has been found (8).

Metabolic Disorders

Because only six primiparous cows contracted milk fever (Table 6), these ETDM and BW estimates are not reliable. The higher BW of multiparous cows in the week before milk fever diagnosis suggest that high BW increases the risk of milk fever. Compared with the BW of healthy cows or the BW in the initial week after milk fever diagnosis, no significant effect of milk fever on BW was found. Compared with the BW in the week preceding diagnosis of milk fever, a temporary 18-kg loss was found. The fact that this predisease estimate is based on a relatively large number of observations offers some reliability to this latter estimate of BW loss from milk fever.

There were some similarities within the BW results for the other metabolic diseases: ketosis, decreased rumen motility, enteritis, LDA, and off feed. Compared with mastitis and reproductive disorders, the BW losses were significantly higher. A high body condition score has been associated with increased risk of ketosis (30) and LDA (38). Our results did not demonstrate predisease BW as a risk factor for either ketosis, decreased rumen motility, enteritis, LDA, or off feed (Tables 6 through 11). Compared with the BW >3 wk before diagnosis, ketosis, decreased rumen motility, enteritis, and off feed caused a BW loss for multiparous cows of between 20 and 32 kg; loss from LDA was 69 kg. Except for ketosis, the losses tended to be slightly lower for primiparous cows. The loss from ketosis was persistent, but recovery within 5 wk after diagnosis occurred after enteritis and off feed, and some recovery occurred after decreased rumen motility and LDA. After LDA and off feed, primiparous cows tended to recover less fully than multiparous cows. Almost all BW loss had occurred up to and including the first week after diagnosis, which emphasizes the detrimental effect of the subclinical stage.

High milk yield has generally not been found to be a risk factor for metabolic diseases, except for milk fever (9, 13, 17, 16) and ketosis (7, 11, 17). Our results of milk yield >3 wk before diagnosis indicate high milk yield as a risk factor for ketosis and enteritis. In contrast, as in another study (31), our results indicate low milk yield as a risk factor for LDA. Rather than low milk yield itself being a risk factor for LDA, low milk yield may result from the subclinical phase of LDA (or from other diseases) before the

diagnosis is made. Predisease milk yield cannot be evaluated as a risk factor for milk fever in our study because of its early occurrence, but, if high milk yield is a risk factor, we may underestimate the loss when comparing it with the loss experienced by healthy cows. The persistent milk loss for multiparous cows with milk fever could be due to its importance as a risk factor for other diseases (9, 14, 23). Previous studies (5, 8) found either temporary or persistent loss of milk yield from milk fever.

Comparison of the estimate of ETDM >3 wk before diagnosis with the successive nine estimates resulted in an aggregated measure of milk loss; the total milk losses were 205, 106, 29, 107, and 42 kg for primiparous cows and 211, 73, 125, 92, and 157 kg for multiparous cows from ketosis, decreased rumen motility, enteritis, LDA, and off feed, respectively. These losses underestimate the total loss in cases of persistent milk loss, which we found for primiparous cows with ketosis, decreased rumen motility, and LDA and for multiparous cows with ketosis and enteritis. The milk yield did not drop before diagnosis, as determined by the estimate of ETDM >3 wk before diagnosis, except for multiparous cows with ketosis and off feed, for which the milk yield decreased the week before diagnosis.

Compared with healthy cows, the average milk loss from LDA within the first 6 wk after diagnosis was 4.6 and 5.2 kg/d for primiparous and multiparous cows, respectively. In a previous study (7) using corresponding estimates except for including the first 60 d after LDA diagnosis, a similar loss was found for primiparous cows (4.4 kg/d), but a higher loss was found for multiparous cows (7.3 for parities 2, 3, and 4). However, the pattern of milk loss was similar in their study (6) and in ours. In another study (5), milk yield of cows with displaced abomasum did not differ from that of healthy cows from 1 to 5 DIM, but a temporary milk loss of 402 kg occurred until 49 DIM. This loss is even higher than the difference between cows with LDA and healthy cows in our results, partly because only eight cases of displaced abomasum were included in their (5) study. Additionally, in both of those previous studies (5, 6), the average milk yield was significantly higher than in the herds we studied. Studies using 305-d milk yield have estimated losses from LDA of 11% (32) and 7.5% (8).

Our estimates of loss from ketosis are within the range of estimates in other studies (5, 7, 8, 21, 35). In one of these studies (7), ketosis caused a loss of 2.6 kg/d of milk for 17 d after diagnosis. They (7) had no recordings of milk losses before diagnosis, which may explain why we found higher milk loss in that period. In another study (5), cows with ketosis were found not to differ in 1 to 5 DIM milk yield, but they had a temporary milk loss until 49 DIM compared with cows without ketosis. This loss was 158.6 kg if ketosis occurred between 1 and 21 DIM and 200.8 kg if ketosis occurred between 22 and 49 DIM; the 119-d milk loss for all ketosis cases ranged between 253.4 and 336.8 kg (5). In a study of milk loss from hyperketonemia, a 233.4 kg (8.5%) 100-d milk loss and a 328 kg (6.5%) 200-d milk loss were found (21). In studies using 305-d milk yield, the estimates of milk loss from ketosis ranged from no loss (8, 35) to 141.1 kg (7). One of those studies (8) found a 2.5% increased milk yield per day of life from ketosis.

We found no comparable studies of production loss from decreased rumen motility, enteritis, or off feed.

CONCLUSIONS

By using data from Danish research herds, it was possible for us to quantify some short-term effects of mastitis, three reproductive disorders (retained placenta, metritis, and cystic ovaries), and seven metabolic disorders (milk fever, ketosis, decreased rumen motility, enteritis, LDA, RDA, and off feed) on weekly ETDM and BW. For study diseases with sufficient recordings before diagnosis, which excludes retained placenta and milk fever, high milk yield was indicated as a risk factor for ketosis and enteritis, and heavier primiparous cows were more likely to contract mastitis.

The ETDM was decreased for a disease-specific period for all study diseases except cystic ovaries and RDA. Metabolic diseases had a more significant detrimental effect on BW loss than did reproductive disorders and mastitis. The highest BW loss (69 kg) was caused by LDA. The persistence of the BW loss differed considerably among study diseases. Almost all BW loss occurred up to and including the initial week after diagnosis, which emphasized the detrimental effect of the subclinical stage. However, weekly measured BW seemed superior to weekly ETDM for disease detection only for decreased rumen motility and LDA. This study demonstrates the importance of the predisease level for accurate estimation of the loss of ETDM and BW caused by disease.

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REFERENCES

1 Bartlett, P. C., J. Van Wijk, D. J. Wilson, C. D. Green, G. Y. Miller, G. A. Majewski, and L. E. Heider. 1991. Temporal patterns of lost milk production following clinical mastitis in a large Michigan Holstein herd. J. Dairy Sci. 74:1561–1572.

2 Bigras-Poulin, M., A. H. Meek, and S. W. Martin. 1990. Interrelationships of health problems and age on milk production in selected Ontario Holstein cows. Prev. Vet. Med. 8:3–13.

3 Calavas, D., B. Faye, F. Bugnard, C. Ducrot, and F. Raymond. 1996. Analysis of associations among diseases in French dairy cows in two consecutive lactations. Prev. Vet. Med. 27:43–55.

4 Coulon, J. B., F. Lescourret, and A. Fonty. 1996. Effect of foot lesions on milk production by dairy cows. J. Dairy Sci. 79:44–49.

5 Deluyker, H. A., J. M. Gay, L. D. Weaver, and A. S. Azari. 1991. Change of milk yield with clinical diseases for a high producing herd. J. Dairy Sci. 74:436–445.

6 Detilleux, J. C., Y. T. Gröhn, S. W. Eicker, and R. L. Quaas. 1997. Effects of left displaced abomasum on test day milk yields of Holstein cows. J. Dairy Sci. 80:121–126.

7 Detilleux, J. C., Y. T. Gröhn, and R. L. Quaas. 1994. Effects of clinical ketosis on test day milk yields in Finnish Ayrshire cattle. J. Dairy Sci. 77:3316–3323.

8 Dohoo, I. R., and S. W. Martin. 1984. Disease, production and culling in Holstein-Friesian cows. IV. Effects of disease on production. Prev. Vet. Med. 2:755–770.

9 Dohoo, I. R., and S. W. Martin. 1984. Disease, production and culling in Holstein-Friesian cows. III. Disease and production as determinants of diseases. Prev. Vet. Med. 2:671–690.

10 Dohoo, I. R., S. W. Martin, A. H. Meek, and W. C. D. Sandals. 1983. Disease, production and culling in holstein-friesian cows. I. The data. Prev. Vet. Med. 1:321–334.

11 Emanuelson, U. 1988. Recording of production diseases in cattle and possibilities for genetic improvements: a review. Livest. Prod. Sci. 20:89–106.

12 Enevoldsen, C., and Y. T. Gröhn. 1994. Disease effects on milk yield in dairy cows: Development and reliability assessment of a simplified within herd and within test day approach for estimation. The Kenya Veterinarian 18:418–420.

13 Erb, H. N. 1987. Interrelationships among production and clinical disease in dairy cattle: a review. Can. Vet. J. 28:326–329.

14 Erb, H. N., and Y. T. Gröhn. 1988. Epidemiology of metabolic disorders in the periparturient dairy cow. J. Dairy Sci. 71: 2557–2571.

15 Erb, H. N., R. D. Smith, P. A. Oltenacu, C. L. Guard, R. B. Hillman, P. A. Powers, M. C. Smith, and M. E. White. 1985. Path model of reproductive disorders and performance, milk fever, mastitis, milk yield, and culling in Holstein cows. J. Dairy Sci. 68:3337–3349.

16 Gröhn, Y. T., S. W. Eicker, and J. A. Hertl. 1995. The association between previous 305-day milk yield and disease in New York State dairy cows. J. Dairy Sci. 78:1693–1702.

17 Gröhn, Y. T., H. N. Erb, C. E. McCulloch, and H. S. Saloniemi. 1989. Epidemiology of metabolic disorders in dairy cattle: association among host characteristics, disease, and production. J. Dairy Sci. 72:1876–1885.

18 Gröhn, Y. T., H. N. Erb, C. E. McCulloch, and H. S. Saloniemi. 1990. Epidemiology of mammary gland disorders in multiparous Finnish Ayrshire cows. Prev. Vet. Med. 8:241–252.

19 Gröhn, Y. T., H. N. Erb, C. E. McCulloch, and H. S. Saloniemi. 1990. Epidemiology of reproductive disorders in dairy cattle: association among host characteristics, disease and production. Prev. Vet. Med. 8:25–39.

20 Gröhn, Y. T., J. R. Thompson, and M. L. Bruss. 1984. Epidemiology and genetic basis of ketosis in Finish Ayrshire cattle. Prev. Vet. Med. 3:65–77.

21 Gustafsson, A. H., L. Anderson, and U. Emanuelson. 1993. Effect of hyperketonaemia, feeding frequency and intake of concentrate and energy on milk yield in dairy cows. Anim. Prod. 56:51–60.

22 Houben, E. H. P., A. A. Dijkhuizen, J.A.M. van Arendonk, and R.B.M. Huirne. 1993. Short- and long-term production losses and repeatability of clinical mastitis in dairy cattle. J. Dairy Sci. 76:2561–2578.

23 Klerx, H. J., and E.A.A. Smolders. 1997. Herd and cow random variation in models of interrelationships between metabolic and reproductive disorders in high yielding multiparous Holstein dairy cattle in The Netherlands. Livest. Prod. Sci. 52:21–29.

24 Laven, R. A., and A. R. Peters. 1996. Bovine retained placenta: aetiology, pathogenesis and economic loss. Vet. Rec. 139: 465–471.

25 Lescourret, F., and J. B. Coulon. 1994. Modelling the impact of mastitis on milk production by dairy cows. J. Dairy Sci. 77: 2289–2301.

26 Lescourret, F., J. B. Coulon, and B. Faye. 1995. Predictive model of mastitis occurrence in the dairy cow. J. Dairy Sci. 78: 2167–2177.

27 Lucey, S., and G. J. Rowlands. 1984. The association between clinical mastitis and milk yield in dairy cows. Anim. Prod. 39: 165–175.

28 Lucey, S., G. J. Rowlands, and A. M. Russel. 1986. Short-term associations between disease and milk yield of dairy cows. J. Dairy Res. 53:7–15.

29 Maltz, E., S. Devir, J.H.M. Metz, and H. Hogeveen. 1997. The body weight of the dairy cow. 1. Introductory study into body weight changes in dairy cows as a management aid. Livest. Prod. Sci. 48:175–186.

30 Markusfeld, O., N. Galon, and E. Ezra. 1997. Body condition score, health, yield and fertility in dairy cows. Vet. Rec. 141: 67–72.

31 Markusfeld, O., N. Nahari, and H. Adler. 1988. Traits associated with the "fat cow syndrome" in dairy cattle. A combined clinical, epidemiological and biochemical study of a multifactorial disease syndrome. Isr. J. Vet. Med. 44:176–182.

32 Martin, S. W., K. L. Kirby, and R. A. Curtis. 1978. Left abomasal displacement in dairy cows: its relationship to production. J. Dairy Sci. 19:250–253.

33 Morris, R. S. 1988. The effects of disease on productivity and profitability of livestock: how should it be assessed? Proc. N.Z. Soc. Anim. Prod. 48:117–125.

34 Morse, D., M. A. Delorenzo, C. J. Wilcox, R. P. Natzke, and D. R. Bray. 1987. Occurrence and recurrence of clinical mastitis. J. Dairy Sci. 70:2168–2175.

35 Rowlands, G. J., and S. Lucey. 1986. Changes in milk yield in dairy cows associated with metabolic and reproductive disease and lameness. Prev. Vet. Med. 4:205–221.

36 SAS® Institute Inc. SAS/STAT Software: Changes and Enhancements through Release 6.11, Cary, NC: SAS Inst. Inc., 1996. 1104 pp.

37 Schepers, J., and A. A. Dijkhuizen. 1991. The economics of mastitis and mastitis control in dairy cattle: a critical review of estimates published since 1970. Prev. Vet. Med. 10:213–224.

38 Shaver, R. D. 1997. Nutritional risk factors in the etiology of left displaced abomasum in dairy cows: a review. J. Dairy Sci. 80:2449–2453.

39 Sjaunja, L. O., L. Baevre, L. Junkkarinen, J. Pedersen, and J. Setälä. 1990. A Nordic Proposal For an Energy Corrected Milk (ECM) formula. 27th session, Int. Committee for Anim. Recording, Paris, France.