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The Impact of Clinical Lameness on the Milk Yield of Dairy Cows

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ABSTRACT

This paper investigates the impact of lameness on milk yield. The dataset includes approximately 8000 test-day milk yields from 900 cows on five farms in Gloucester, UK, collected over 18 mo from 1997 to 1999. The data were structured to account for repeated measures of test-day yield (1 to 10 per cow) and analyzed to account for this autocorrelation. Factors affecting milk yield included: farm of origin, stage of lactation, parity, and whether a cow ever became lame. In clinically lame cows, milk yield was reduced from up to 4 mo before a case of lameness was diagnosed and treated and for the 5 mo after treatment. The total mean estimated reduction in milk yield per 305-d lactation was approximately 360 kg. We conclude that clinical lameness has a significant impact on milk production. This is important information for assessing the economic impact of clinical lameness and its impact on cow health. It adds weight to the importance of early identification of clinical lameness and the urgency of techniques to improve the definition of this highly subjective diagnosis.
(**Key words:** milk yield, lameness, dairy cow, multilevel modeling)

Abbreviation key: TDY = test-day yield.

INTRODUCTION

Clinical lameness is of concern because of its high prevalence (Clarkson et al., 1996), association with pain (Whay et al., 1997), other diseases (Lucey et al., 1986; Barkema et al., 1994) and because of the attributed economic losses (Whitaker et al., 1983; Enting et al., 1997; Kossaibati and Esslemont, 1997).

The definition of clinical lameness in cattle is fraught with difficulty, even among specialists. Currently, cows can be "locomotion scored" (Manson and Leaver, 1988; Whay et al., 1997). These scores include a category for 'imperfect locomotion' or 'uneven gait' to define a cow that is unsound (favoring one leg) but not clinically lame. Whether these cows will become lame or are recovering from an episode of lameness or are transiently unsound is unknown. The importance of this state for the health, welfare, and production of the cow is also unknown. This clearly indicates that a gold standard (Martin et al., 1987) for 'clinical lameness, yes/no' has not been achieved. Despite this, the outcome clinical lameness is the best measure we have and is frequently used in observational research throughout the world (Whitaker et al., 1983; Miller and Dorn, 1990; Tranter and Morris, 1991; Barkema et al., 1994; Hedges et al., 2001).

The difficulty in defining clinical lameness may in part explain the high variability in the reported incidence of clinical lameness in dairy cows. Estimates of between 5 (Eddy and Scott, 1980) and 70 cases/100 cows per year (Hedges et al., 2001) have been made in the United Kingdom. This variability in incidence is reported worldwide, e.g., Harris et al., (1988) reported 0 to 50% in Australia and Barkema et al. (1994) reported 9 to 50% in the Netherlands. Part of the variation may also be attributed to the different skills of personnel responsible for identifying lame cows. Parlor workers, farm managers, veterinarians, and research workers have been used to identify lame cows both within farms (Barkema et al., 1994; Clarkson et al., 1996) and between (Lucey et al., 1986; Hedges et al., 2001). There is also clearly large variability in the incidence and types of lameness between farms (Barkema et al., 1994; Hedges et al., 2001).

The imprecise definition of lameness causes misclassification (Martin et al., 1987): Lame cows defined as nonlame and vice versa. It possibly also causes bias (Martin et al., 1987) since under-diagnosis seems empirically more likely. If there is a downward bias in identifying whether or when a cow becomes lame, the impact of lameness on health, production, and, there-

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fore, the consequential economic loss is likely to be underestimated.

Sensitivity analysis indicates that the reduction in milk volume and quality is highly influential on estimates of economic loss from clinical lameness (Enting et al., 1997). However, the evidence for the impact of lameness on milk yield is conflicting. Some authors report a decreased milk yield after diagnosis (Whitaker et al., 1983; Tranter and Morris, 1991; Warnick et al., 2001), others a decrease in milk yield before a cow was treated as well as after (Lucey et al., 1986), and others that there is no change in milk yield (Cobo-Abreu et al., 1979). However, Barkema et al. (1994) reported an increased milk yield from 100 to 270 DIM in the same lactation in cows with sole ulcer. These authors also reported an increase in the 100-d cumulative milk volume in the previous lactation for cows with any cause of lameness.

This finding led Barkema et al. (1994) to conclude that an estimate of milk loss by calculating the deviation from the lactation curve of daily yields was necessary to assess the impact of lameness on milk production, rather than comparison of cumulative yields. This is particularly true for cows with higher than average yield, since a reduction in total yield may bring these cows to the average, not below it, and consequently no difference in volume between lame and nonlame cows will be detected (Lucy et al., 1986).

It is therefore important to improve on current estimates of the impact of lameness on milk yield. Grohn et al. (1999) used an elegant technique with repeated measures of monthly test-day yield (**TDY**) to demonstrate that cows produced less milk immediately before and after an episode of ketosis. These cows did not produce significantly less milk than unaffected cows over 305 d of lactation, and a more simple analytical technique would not have detected the loss in milk volume.

Test-day milk yields are repeated measures from one cow. The volume of milk produced at one test is dependent in part on the volume of milk produced at the previous test day and will influence the following TDY. These repeated measures can be analyzed using hierarchically clustered mixed models with fixed and random variables (Goldstein, 1995). This paper uses this technique and presents a multilevel model of the impact of lameness on the milk yield of dairy cows.

MATERIALS AND METHODS

The data come from 900 Friesian/Holstein dairy cows on five farms in Gloucestershire, UK, from the study of Hedges et al. (2001) that investigated the effect of biotin supplementation on the incidence of lameness in dairy cows. The herds were autumn calving. Cows were at pasture in the summer months and fed on grass with concentrate ration fed in the parlor. During the winter, cows were housed in cubicles and fed a concentrate ration in the parlor and conserved forage (grass or grass and maize silage) in yards. The mean herd 305 lactation day yield ranged from 5500 to 7500 kg/cow.

The dataset includes approximately 8000 test-day milk yields (one per cow per month in milk) from 900 cows over 18 mo from 1997 to 1999. Clinical lameness was identified by the farmer and diagnosed and treated by one of six veterinarians who recorded the site of the lesion, the cause, treatment given, and date of treatment on a standard form. There was no economic cost to the farmer for this treatment (Hedges et al., 2001).

The data were structured (Table 1) so that each TDY for each cow formed one row of data. The TDY dates were repeated measures through time, and these repeated measurements were coded 1 to 10 from calving to 300 d of lactation. TDY > 10 were not used in the analysis. The number of days from lameness to/from a TDY was estimated by subtracting the date of diagnosis of lameness from the test-day date. This variable was converted to a factor variable coded as months from/to a diagnosis of lameness (–5 to +5) for each TDY. Only the first occurrence of clinical lameness in a lactation was used to estimate the impact of lameness on milk yield. Stage of lactation was modelled as DIM, and an exponential function to the power –0.05 DIM (Wilmink, 1987). The dataset also contained the following factor variables: cow identity, farm of origin (1 to 5), parity (1 to 4+), first or second lactation in the study, lame during lactation (ever-lame, 1 = yes, 0 = no) and whether the cow received a biotin supplement of 20 mg/d during the study period (1 = yes, 0 = no).

The data were analyzed in MlwiN 1.1006 (Rasbash et al., 1999). TDY was the outcome variable, the data were distributed normally. A two-level general linear model with restricted iterative generalized least squares procedure was used to analyze these hierarchically clustered data. Level two was the cow identity and grouped within this at level one were the TDY repeated measures sorted by month from calving. Each cow contributed a maximum of 20 TDY events and a minimum of one. Farm of origin and biotin supplementation were forced into the model as fixed effects.

The model was:

$$y_{ij} = \alpha_{ij} + \Sigma\beta_{ij}X_{ij} + \Sigma\Delta_j Z_j + u_j + e_{ij}$$

where y_{ij} = milk yield on test day i for cow j, α_{ij} = intercept value for test day i for cow j, β_{ij} = coefficients for X_{ij}, X_{ij} = exposure for TDY ij, Δ_j = coefficients for Z_j, Z_j = exposures for cow j, Σ = sum of 1 to n exposures,

Table 1. An example of the structure of the dataset.

Farm ID	Cow ID	Lactation in study	Parity	TD ID	TD date	Ever Lame	TD lame	lame +1 month	lame –1 month	lame –2 months
1	1	1	4	1	7/3/97	0	0	0	0	0
1	1	1	4	2	8/4/97	0	0	0	0	0
1	1	1	4	3	9/2/97	0	0	0	0	0
1	1	1	4	4	10/24/97	0	0	0	0	0
1	1	1	4	5	11/25/97	0	0	0	0	0
1	1	1	4	6	12/19/97	0	0	0	0	0
1	1	1	4	7	1/22/98	0	0	0	0	0
1	1	1	4	8	2/23/98	0	0	0	0	0
1	1	1	4	9	3/28/98	0	0	0	0	0
1	1	1	4	10	4/27/98	0	0	0	0	0
1	1	2	5	1	5/5/98	1	0	0	0	1
1	1	2	5	2	6/2/98	1	0	0	1	0
1	1	2	5	3	7/4/98	1	1	0	0	0
1	1	2	5	4	8/6/98	1	0	1	0	0
1	1	2	5	5	9/3/98	1	0	0	0	0
1	1	2	5	6	10/2/98	1	0	0	0	0
1	1	2	5	7	11/5/98	1	0	0	0	0
1	1	2	5	8	12/6/98	1	0	0	0	0
1	1	2	5	9	1/5/99	1	0	0	0	0
1	1	2	5	10	2/4/99	1	0	0	0	0

TDY = Test-day yield, ID = identity.

u_j = error term for between cow variation, e_{ij} = residual level one error.

Complex variation (where the intercept and the slope of the lactation curve varied between cows) was tested. As a consequence, u_j, between cow variation, was dependent upon $\gamma + \delta_j A_j$, where γ = intercept variance, $\delta_j A_j$ = variance function for exposure Z_j, and e_{ij} was dependent upon $\eta + \theta D_{ij}$, where η = intercept variance of repeated measures and $\theta_{ij} D_{ij}$ = variance function for exposure X_{ij}.

The occurrence of first lameness by month in milk was plotted, and the mean lactation curve for cows that were never lame and cows that were clinically lame during a lactation (ever-lame) was compared visually in Excel 97 (Microsoft Inc., Redmond, WA). The distribution of standard residuals of the multilevel model was plotted to check the model fitting.

RESULTS

Over 70% of cows became lame at least once. The four most frequent diagnoses of lameness were sole ulcer, white line disease, interdigital necrobacillosis, and digital dermatitis. These had an incidence of 9 to 11 cases/100 cows per year. The incidence of first episode of lameness peaked 3 mo after calving (Figure 1). High yielding cows were more likely to be lame and produced more milk throughout lactation than cows that were never lame (Figure 2). As a consequence, the dummy variable 'ever-lame' was put into the model to estimate the mean daily increased yield (Table 2). These cows produced a mean of 1.12 (+/–0.34) kg/d more milk than cows that were never lame: that is a mean of 342 extra kilograms of milk over 305 DIM (95% CI 135 to 549 kg). Factors affecting milk yield included: farm of origin, stage of lactation, parity, and whether this was the cow's first or second lactation (Table 2). Interactions between farm of origin and parity with stage of lactation were significant but had no impact on the estimated milk loss attributable to lameness. These have not been presented.

Clinically lame cows had a reduced milk yield from up to 4 mo before a case of lameness was diagnosed and treated and for 5 mo after treatment (Table 3). The

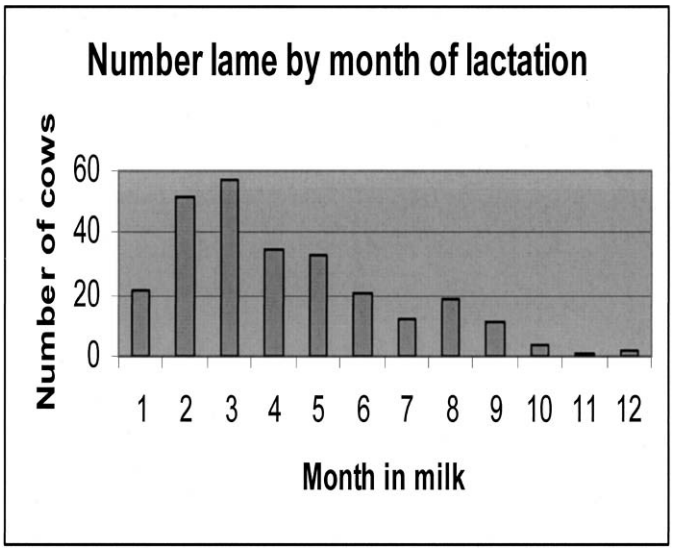

Figure 1. Bar chart of number of cows lame by month in milk.

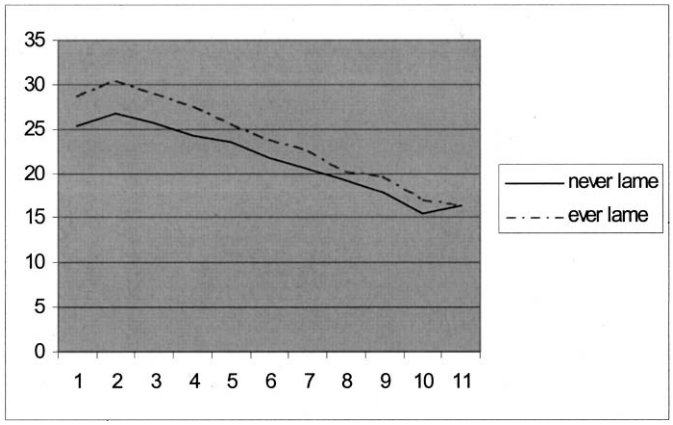

Figure 2. Mean lactation curves for cows that were ever-lame versus those that were never-lame. X axis = repeated measures of test day yield, Y axis = estimated kg of milk per day

total mean estimated reduction in milk yield for a cow lame in the fifth month of lactation onwards was 357 kg (95% CI 163 to 552) per 305-d lactation (Table 3). Figure 3 illustrates the impact on milk yield for a cow lame 2 mo after calving, together with the estimated milk yield had the cow not become lame, compared with the mean yield of a cow that was not lame.

There was complex variation in the random structure of the model with DIM and whether the cow was in her first or second lactation in the study, accounting for some of the between-cow variation (Table 4). This indicates that the slopes of the lactation curves varied between cows because of these exposures. The residual plots indicated that the model assumptions were correct (Figure 4).

DISCUSSION

This analysis has identified a higher mean lactation yield in cows that were lame during a lactation (ever-lame) versus those that were not lame during a lactation as postulated by Lucey et al. (1986), Barkema et al. (1994), and Hansen et al. (1978). As a consequence, a level 2 dummy variable that coded cows that were lame during lactation as 'ever-lame' was created and put into the model. Cows that were lame produced a mean increased milk yield of 1.12 kg/d during lactation on the days where lameness did not cause reduced milk production. This has implications for the health of high yielding dairy cows. They are at greater risk of ketosis (Grohn et al., 1999) and other health disorders (Hansen et al., 1979) and we can now confirm that they are at greater risk of lameness. This increased risk may arise because their nutritional demands are not met. Even where there is an adequate quantity and quality of food, high yielding cows must stand for long periods to eat, and this too may increase their risk of lameness. However, it may also be that these cattle are at greater risk of lameness innately. Genetic studies indicate that high milk yield is negatively correlated with low incidence of lameness (Hansen et al., 1979).

This information makes decisions on culling for lameness more complex. Total yield needs to be considered before lame cows are culled. This may be why there is not always a positive association between culling and

Table 2. The impact of fixed effects on milk yield (kg).

Exposure	Mean effect	s.e.	lower 95% CI[1]	upper 95% CI
Intercept	26.578	0.697	25.184	27.972
			0	0
Ever lame	1.123	0.343	0.437	1.809
			0	0
DIM	–0.49	0.002	–0.053	–0.045
Wilmink function	–9.169	0.424	–10.017	–8.321
			0	0
Second study lactation	2.777	0.312	2.153	3.401
Farm 2	4.219	0.631	2.957	5.481
Farm 3	0.983	0.578	–0.173	2.139
Farm 4	5.907	0.510	4.887	6.927
Farm 5	–2.143	0.769	–3.681	–0.605
Parity 2	0.493	0.368	–0.243	1.229
Parity 3	1.022	0.426	0.176	3.314
Parity 4+	2.462	0.413	1.636	3.288
April–June	0.179	0.732	–1.285	1.643
July–Sept	0.866	0.666	–0.466	2.198
Oct–Dec	0.737	0.546	–0.355	1.829
Biotin—yes	–0.008	0.0.302	–0.612	0.596

CI = Confidence interval.

Table 3. Mean daily reduction in milk yield (kg) in lame cows in the months before and after diagnosis.[1]

Exposure	Mean effect	s.e.	Lower 95% CI	Upper 95% CI	Cumulative mean loss in yield*	Cumulative lower 95% CI loss in yield	Cumulative upper 95% CI loss in yield
Months before diagnosis							
5	–0.255	0.352	0.449	–0.959	–7.65	13.47	–28.77
4	–1.065	0.353	–0.359	–1.771	–31.95	–10.77	–53.13
3	–0.85	0.355	–0.14	–1.56	–25.5	–4.2	–46.8
2	–1.598	0.374	–0.85	–2.346	–47.94	–25.5	–70.38
1	–1.729	0.363	–1.003	–2.455	–51.87	–30.09	–73.65
Months after diagnosis							
1	–1.706	0.394	–0.918	–2.494	–51.18	–27.54	–74.82
2	–1.885	0.422	–1.041	–2.729	–56.55	–31.23	–81.87
3	–1.228	0.466	–0.296	–2.16	–36.84	–8.88	–64.8
4	–1.847	0.514	–0.819	–2.875	–55.41	–24.57	–86.25
5	–2.028	0.563	–0.902	–3.154	–60.84	–27.06	–94.62
Total#					–357.24	–162.78	–551.7

[1]Assuming 30 d per month. # excluding 5 mo before diagnosis because confidence level (CI) include unity.

lameness (Barkema et al., 1994). It appears that farmers are already aware of this positive association between milk yield and lameness when they decide whether or not to cull a cow (Barkema et al., 1994). However, this in no way indicates that clinical lameness is acceptable or even tolerable in these cows. The estimates from this paper indicate that lame cows fail to produce an average of approximately 350 kg of milk; therefore, this advantage of higher yield is lost. The conclusion is that to benefit from high yielding cows extra management care is required. If this is not possible then rather than aiming for maximum milk yields farmers should define an optimum yield suitable for their system that maximizes cow health and productivity and use appropriate genetic stock.

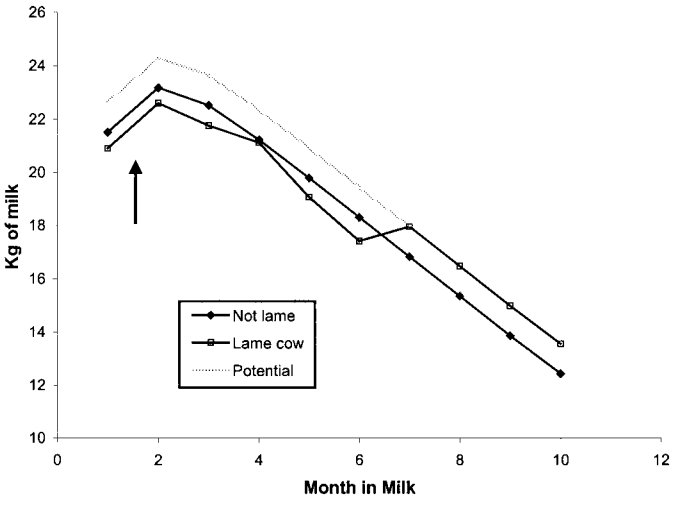

Figure 3. Predicted milk yields of a nonlame cow and a cow with a case of lameness in the second month of lactation. The arrow indicates the time of diagnosis. The dotted line indicates the production potential of the lame cow (estimated from the 'ever-lame' parameter).

The mean range of milk lost per affected cow was 160 to 550 kg. The wide range in predicted loss occurred because all causes of lameness were included in the analysis, and some causes may have impacted on milk yield more than others. It will also have occurred because lameness occurred in each month of lactation, and so not all lame cows contributed to all months where milk yield was reduced, e.g., a cow lame in mo 2, as in Figure 3, would not contribute a milk yield for 3 or 4 mo before she was lame. Variability in the cause of lameness and the time of lameness during lactation will also have led to variability in the mean estimate of milk lost by month (Table 3). The 95% range in milk lost per month is also wide, and the importance of variability in mean monthly loss is unknown; lesion specific causes of lameness may explain this variability. Unfortunately, in our data, there were not enough cases of each individual cause of lameness to test their impact on milk yield in this study. However, it is an important issue and should be the subject of future research. To make this possible without resorting to expensive prospective studies, current recording systems used in herd health programs need to move away from recording 'lameness' as a single entity and towards recording individual causes of lameness.

The reduced milk yield before a diagnosis of lameness could arise from a confounder that was associated with lameness later in lactation and reduced milk yield. For example, it is possible that these cows had an insult early in lactation (e.g., ketosis) that reduced their milk yield that was correlated with or increased their likelihood of becoming lame later in lactation. In which case, this early reduction in milk yield was caused by a sepa-

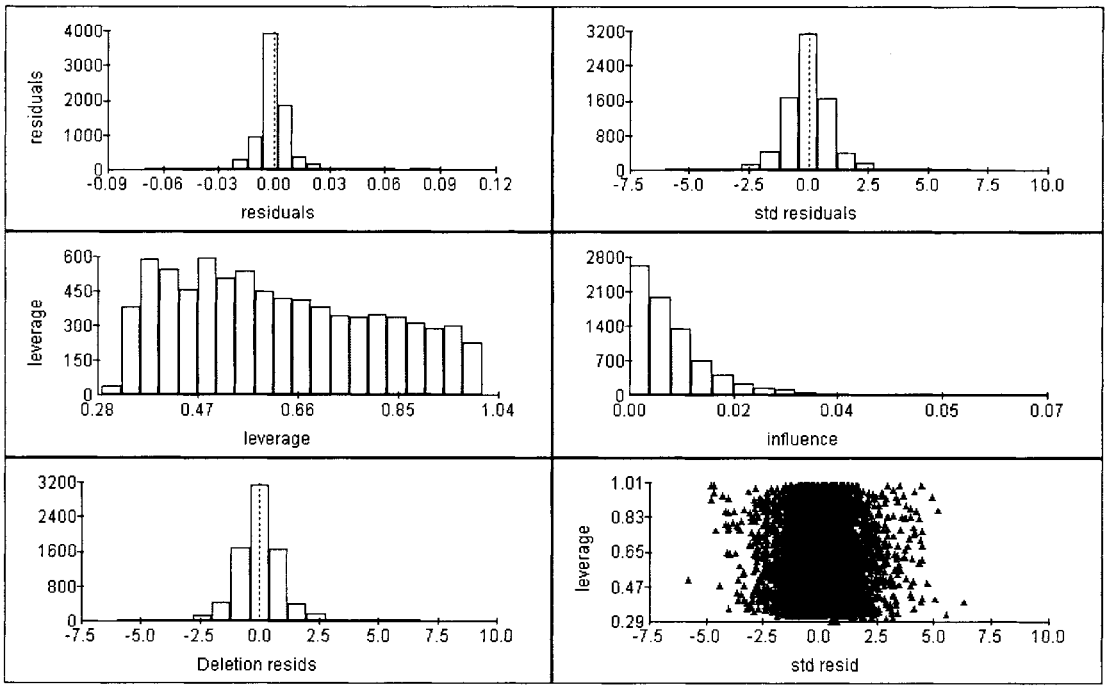

Figure 4. Plots of residuals and leverage from the developed model. std resid = Standardized residuals.

rate insult, and not by the later occurring lameness. It is also possible that since Collick et al. (1989) estimated that 66% of sole ulcers occurred by 100 DIM and Leach et al. (1997) estimated that white line lesions were most severe at 63 DIM, that this reduction in milk yield is a result of undetected clinical lameness. These animals may have been undiagnosed until later in lactation. Recent work indicates that farmers underestimate the prevalence of lameness in their cows considerably; in a study of 53 herds, the mean estimate of lame cows was 5% by farmers versus 25% by the researcher (Whay et al., 2002).

Another more likely possibility for the delay in treatment may be that these cows were unsound but not clinically lame in early lactation but eventually became clinically lame. This highlights the important issue of case definition. There is clearly a need to improve the detection of clinical lameness and to remove the subjective assessment of the human observer, whether farmer or veterinarian or agricultural consultant. There have been attempts to do this using rising position and limb placing but none has reached commercial development. A reliable and repeatable objective assessment of lameness is required. Until we have such a measure, all estimates of the impact of lameness will be imprecise and will therefore affect estimates of its effect on milk yield despite the improved precision of estimates of milk lost.

Lucey et al. (1986) reported a reduction in yield from 9 wk before an episode of sole ulcer or white line separation lameness; this was the maximum time that milk loss was investigated in this study. Both sole ulcers and white line lesions result from an insult to the corium. The defective horn which is produced as a consequence of this insult may be visible (on the sole surface) 2 to 3 mo later (Lischer et al., 2000) and therefore may affect milk production over a period of time before any signs of injury. Warnick et al. (2001), using daily milk recording, reported that acute and severe lameness cases were quickly resolved, e.g., interdigital necrobacillosis had a short impact on milk production. In the present study, TDY were recorded each month and such short-duration changes in milk production may have been missed.

Table 4. Random effects.[1]

Exposure	Variance	s.e.
Level 2		
Intercept	42.385	2.575
DIM	0.001	0.000
Covariance DIM/Intercept	–0.177	0.013
Second study lactation	29.025	2.669
Covariance lact./Intercept covariance	–14.629	2.228
Second lact./DIM	0.053	0.012
Level 1		
Intercept	15.655	0.287

[1]Total log likelihood from model (Tables 2–4) = 47,412.3, null model 52,434.4.

In this study, the veterinarians diagnosed the cause of lameness and locomotion scored the herds every 2 mo and identified all lame cows not under treatment; there were rarely any. This may have been because the cows were treated at no cost to the farmer. The very high incidence rate of 70 cases per 100 cows per year indicates that lameness detection rates were high. Assuming that we may have included relatively mild cases of lameness, as well as more severe, our data would underestimate milk loss per cow compared with herds where only more severe cases are diagnosed.

Because of the small number of farms in this study, the results are not likely to be generalizable to all herds. The exact quantity of milk lost is unlikely to be precise for all farms. Similarly, the range of months of reduced milk production may vary. However, the general principle, that lame cows do produce less milk than their potential and do so before and after they are diagnosed as lame is likely to be true in herds with cows of good genetic merit.

CONCLUSIONS

We conclude that some of the potential of high yielding cows in a herd may be lost if they become lame. In this study, decreased milk yield occurred from 4 mo before until 5 mo after a cow was diagnosed as clinically lame and caused up to 360 kg (range 160 to 550 kg) milk loss over a lactation. This is important information for assessing the economic impact of lameness and also its impact on cow health. These results add weight to the importance of early identification of clinical lameness and the urgency of techniques to improve this highly subjective diagnosis.

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