

The Effect of Lameness on Milk Production in Dairy Cows

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ABSTRACT

Data were collected prospectively for 1.5 yr on two New York dairy farms to investigate the effect of lameness on milk production. The numbers of study cows (percentages treated at least once for lameness) in each herd were 1796 (52%) and 724 (40%), respectively. Lameness was identified and treated by farm employees or a professional hoof trimmer. Weekly averages of total milk production per day were recorded based on automated milk weight measurements at each milking. The effect of lameness on milk production was analyzed separately for each herd using repeated measures ANOVA. In both herds, milk production decreased significantly for cows diagnosed lame. Milk production was 1.5 kg/d lower ≥ 2 wk after lameness compared with cows that had not yet been diagnosed lame in the current lactation in the larger herd. In the second herd, milk production of lame cows was 0.8 kg/d lower in the first and second wk after lameness and 0.5 kg/d lower ≥ 3 wk after diagnosis. The decrease in milk production associated with lameness was larger for cows in second or greater lactation and for more severe cases. In one herd, the decrease in milk production was greater for cows with sole ulcers or foot abscesses than for foot rot or foot warts. Cows with abscesses or foot rot tended to have larger decreases in milk production in the other herd. The inconsistent results between farms may have resulted from differences in the relative frequencies of specific causes of lameness in the two herds and in the way lame cows were identified and defined for the study. (**Key words:** lameness, milk production, disease effects)

Abbreviation key: MPD = 1-wk average of total milk production per day, rBST = recombinant bST.

INTRODUCTION

Lameness is an important disease in dairy cattle because of economic and welfare considerations and fre-

quency of occurrence. Diseases of the foot accounted for 99% of the cases of lameness identified in cows in a prospective study of British dairy herds (Clarkson et al., 1996). Point prevalences for lameness of 8 to 15% in US studies (Warnick et al., 1995; Wells et al., 1993) and an average prevalence of 20.6% in Britain were reported (Clarkson et al., 1996). In most field investigations of lameness in dairy cattle, the frequency of lameness was highly variable among herds within the same study. Economic decisions related to the value of preventing and treating lameness in dairy cows require accurate estimates of the associated costs. Economic losses include decreased milk production, weight loss, death, culling, decreased reproductive performance, and treatment costs (Weaver, 1984). Estimates of economic losses due to lameness have varied widely (Enting et al., 1997). One of the difficulties in estimating economic losses has been obtaining accurate data on the effect of lameness on milk production. Results of studies of this question have been mixed, with some studies finding decreased production (Rajala-Schultz et al., 1999; Rowlands and Lucey, 1986; Warnick et al., 1995), others no effect (Cobo-Abreu et al., 1979; Martin et al., 1982), increased milk production for lame cows (Dohoo and Martin, 1984), or variable associations with lameness depending on specific cause or time of milk production measurement relative to diagnosis (Barkema et al., 1994; Coulon et al., 1996; Deluyker et al., 1991). The need continues for estimates of the effect of lameness on milk production in herds representative of current dairy management methods.

The objective of our study was to estimate the effect of lameness on milk production in two dairy herds with daily milk weight measurements. In addition to the overall association of lameness with milk production in each herd, we analyzed the effects of specific lameness diagnoses.

MATERIALS AND METHODS

Study Herds and On-farm Data Collection

The study was carried out in two dairy herds located near Ithaca, New York from June 1, 1997, to December 31, 1998. The herds consisted mostly of Holstein cows

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that were housed in free-stall barns and milked twice or three times daily in milking parlors. The recombinant bST (**rBST**) administration program for herd A was to inject cows every 14 d beginning after 76 DIM and continuing until 2 wk before drying off. In herd B, the rBST program was to give injections every 14 d beginning after 80 DIM until 30 d before drying off. Milk weights were recorded at each milking with automated systems [herd A used ALPRO milking equipment (DeLaval Inc., Kansas City, MO); herd B used Afimilk meters (S. A. E. Afikim, Israel)] and transferred electronically to a dairy records database (Dairy Comp 305, Valley Agricultural Software, Tulare, CA). Cows were diagnosed lame and treated by farm personnel (herd A) or foot lesions were identified by either farm personnel or by a professional hoof trimmer at monthly visits (herd B). A lameness severity score of mild, moderate, or severe was assigned in herd A, but not in the second herd.

The causes of lameness judged to be most significant were categorized as abscess, sole ulcer, foot rot, foot wart, or "other" according to pictures and written descriptions provided to the farms. Abscess (white line abscess, sole abscess) was defined as a pus-filled cavity of the white line or sole of the foot. Sole ulcer (pododermatitis circumscripta) included degenerative or necrotic defects in the sole near the sole-heel junction. Foot rot (interdigital phlegmon) was swelling of the soft tissues of the foot resulting in symmetrical swelling above the coronary band and spreading of the toes, in some cases with necrosis of the tissue between the claws. In herd A, cows with interdigital dermatitis or interdigital hyperplasia were coded as foot rot. Foot warts were defined as ulcerative or proliferative lesions of the digits or interdigital region with a characteristic red or gray pebbly surface. These included lesions with gray or black frond formation. Examples of other recorded conditions included heel cracks, laminitis, hoof overgrowth, stones embedded in the foot, and upper limb problems.

Lameness data were recorded on paper records that were collected on a weekly basis at herd A and were recorded on paper forms by the hoof trimmer or entered into the on-farm computer records system at herd B. The lameness information was entered into a computer database, checked for accuracy against the original records and then transferred to SAS (SAS Institute Inc., Cary, NC) for analysis. Milk production and other cow event data were collected by obtaining a backup disk from the farm computer herd records at approximately weekly intervals. Although we attempted to collect milk production values every 7 d, some backup disks were made at irregular intervals. Milk production recording dates that were less than 1 wk after the previous date

were deleted. In herd A, this resulted in 76 milk production recording dates, 85% of which were at 7-d intervals. The remaining 15% of intervals ranged from 9 to 16 d. For herd B, there were 74 milk production recording dates. Eighty-one percent were at 7-d intervals, and the remaining 19% were from 8 to 15 d apart. Data from the farm computer records system were transferred electronically from Dairy Comp 305 to SAS for analysis. The 1-wk average of total milk production per day (**MPD**) calculated and recorded by Dairy Comp 305 software was used for the analysis of lameness effect on milk production (MPD was recorded as lb/d in the Dairy Comp 305 software and then converted to kg/d for the data analysis). Cows calving from June 1, 1997, to September 30, 1998 that had at least one MPD value recorded were included in the study. For each eligible cow, only the first lactation initiated during the observation period was used for the analysis. The time of observation for cows in the study varied because of loss to follow-up due to culling, different lengths of lactation, or because the study ended before lactation was completed, particularly for cows calving towards the end of the study period.

Lameness events recorded from approximately June 1, 1997, to December 31, 1998, and that occurred on or after the date of the first observed calving and before the second observed calving date were used for the analysis of lameness on milk production. For herd A, lameness was defined as selection for lameness treatment by herd personnel. In herd B, lameness was defined as a lesion of the foot or overgrown hoof wall identified in at least one foot by the hoof trimmer at regularly scheduled visits to the farm or as a cow treated for lameness by farm employees between hoof trimming dates. In each herd, the first lameness event during the study lactation was selected for each cow for the analysis of the association between lameness and milk production.

The lameness treatment incidence rates (lameness treatment events/cow-month) were estimated for each month of lactation by counting the number of lameness treatments for study cows during the time period and dividing by the number of cows with at least one MPD value recorded during the month. For these calculations, repeated lameness treatments of the same cow on different dates were considered as separate events.

Descriptive statistics were calculated for the difference between milk production for lame cows compared with control cows in the same herd, lactation group, stage of lactation, and which remained in the herd at least until the DIM at first lameness diagnosis for the lame cows.

Statistical Analysis

The association of lameness with milk production while controlling for other explanatory factors was analyzed by repeated measures analysis of variance using PROC MIXED of SAS ver. 8. The statistical models had the following form:

$$\text{MPD}_{ijklmn} = \alpha + \text{cow}_i + \text{week}_j + \text{season}_k + \text{stage}_l + \text{lactation}_m + (\text{stage} \times \text{lactation})_{lm} + \text{lame}_n + \epsilon_{ijklmn},$$

where MPD is the 7-d average of total daily milk production (kg/d),

α = intercept,

cow_i = random effect of cow i ($i = 1, 2, \dots, 1796$ for herd A, 724 for herd B),

week_j = milk weight recording week ($j = 1, 2, \dots, 76$ for herd A, 74 for herd B),

season_k = season of calving ($k = \dots$),

stage_l = DIM category ($l = 1, 2, \dots, 31$ corresponding to 10-d intervals; DIM values > 300 were included in $l = 31$),

lactation_m = lactation number ($m = 1, 2$; cows in second or greater lactation were included in $m = 2$),

$(\text{stage} \times \text{lactation})_{lm}$ - interaction between lactation and stage,

lame_n = lameness category ($n = -1$ if the milk weight was from a date before lameness was first diagnosed or from a cow that was not lame during the study, 0 if the same week of lameness, 1 if 1 week after, 2 if 2 wk after or 3 if ≥ 3 wk after the first lameness event), and

ϵ_{ijklmn} = random residual.

All terms other than cow_i and ϵ_{ijklmn} were included in the models as fixed effects. Examples of the data and variable coding from two cows are shown in Table 1. A first-order autoregressive covariance structure was used to model the within-cow correlation of MPD. Satterthwaite's method was used to calculate the denominator degrees of freedom for F -tests on the fixed effects in the models. Separate analyses were done for all cows in each herd and for lactation = 1 and lactation ≥ 2 within herds. The lactation and lactation \times stage interaction terms were not included in analyses that were stratified by lactation group. Model assumptions were evaluated by examining the distribution of the residuals and plots of the residuals and predicted values.

The effects of specific lameness scores or diagnoses on milk production were evaluated using separate repeated measures ANOVA models for each score or diagnosis. For these analyses, only cows with lameness diagnoses or severity scores in the category of interest and cows that were never diagnosed lame were included. The effects of lameness events diagnosed at various stages of lactation (0 to 90, 91 to 180, and > 180 DIM) were analyzed by excluding postlameness MPD values for cows not belonging to the DIM category of interest. The explanatory variables for these analyses stratified by lameness diagnosis, severity, and stage of lactation of occurrence were coded as described above.

The results of models including all eligible cows in each herd were checked when controlling for milk fever, retained placenta, ketosis, left-displaced abomasum, and mastitis (dichotomous variables were created for each disease showing whether or not the milk production value was from a date ≤ 2 wk after the disease date). These diseases were diagnosed and recorded according to the usual practices on the farms—no attempt was made to standardize definitions between the two farms. No important changes were seen in the lameness effect parameter estimates when controlling for other diseases. Only the results from models without other diseases are presented.

RESULTS

Descriptive Statistics

The numbers of cows included in the study from herds A and B were 1796 and 724, respectively. Cows in first or second lactation accounted for over two-thirds of the study animals in each herd. The percentages of cows in first, second, third, or \geq fourth lactation were 39, 32, 14, and 15%, respectively, for herd A and 46, 22, 16, and 17%, respectively for herd B. The daily milk production summarized by 1-wk averages during the study period ranged from 24.7 to 38.0 kg for all milking cows on the 76 production recording dates in herd A. The 74 1-wk averages of daily milk production for all milking cows in herd B were from 23.8 to 30.6 kg.

The percentages of study cows treated for lameness at least once during the study period were 52% for herd A and 40% for herd B. Lameness occurred throughout lactation, but the incidence rate appeared to vary by days in milk (Table 2). Sixty-three percent of the lame cows in herd A were treated ≥ 2 times compared with 27% in herd B. In herd A, 821 cows were assigned lameness severity scores at the time of treatment. Thirty-six percent of these were mild, 39% moderate, and 25% severe. The distribution of first lameness events by diagnosis are shown in Tables 3 and 4. Lameness was significantly more common in older cows

Table 1. Example of data layout for two cows. Cow #1403 calved on 07/06/97 and was never diagnosed lame before being culled on 04/14/98. Cow #1427 calved on 12/24/97 and was diagnosed lame for the first time on 01/13/98.

Cow	Lactation ¹	Season ²	MPD ³	Week ⁴	Stage ⁵	Lame ⁶
1403	2	3	14.5	7/11/97	1	-1
1403	2	3	26.3	7/22/97	2	-1
1403	2	3	29.9	8/1/97	3	-1
...
...
...
1403	2	3	4.1	4/19/98	29	-1
1427	2	1	22.7	12/26/97	1	-1
1427	2	1	34.5	01/02/98	1	-1
1427	2	1	46.3	01/09/98	2	-1
1427	2	1	45.8	01/16/98	3	0
1427	2	1	51.3	01/25/98	4	1
1427	2	1	51.3	02/01/98	4	2
1427	2	1	47.6	02/08/98	5	3
1427	2	1	46.3	02/22/98	7	3
...
...
...
1427	2	1	5.4	11/03/98	31	3

¹Lactation number; 1 or ≥ 2 .

²Season of calving; December to February = 1, March to May = 2, June to August = 3, September to November = 4.

³Preceding 1-wk average of total daily milk production (kg/d); days with no milk weights were not included.

⁴Milk weight recording week identified by last day of the week.

⁵Stage of lactation category; 1, 2, 3, . . . 31 corresponding to 10-d DIM intervals, DIM > 300 included in stage = 31.

⁶Lameness category; -1 if the milk weight was from a date before lameness was first diagnosed or from a cow that was not lame during the study, 0 if the same week of lameness, 1 if 1 wk after, 2 if 2 wk after or 3 if ≥ 3 wk after the first lameness event.

(Cochran-Armitage trend test $P \leq 0.001$). The percentages of cows classified as lame at least once among cows in first, second, third, and fourth or greater lactations were 36, 57, 63 and 70%, respectively, for herd A, and 32, 50, 38, and 50%, respectively for herd B. The definition of lameness in herd B included cows with foot lesions found by the trimmer in addition to cows with gait abnormalities.

Lameness Effect on Milk Production

Based on descriptive results, lame cows had similar milk production to unaffected herd mates before the diagnosis of lameness (comparisons were made to control cows in the same herd, lactation group, and stage of lactation). The average lame minus control cow differences before the diagnosis of lameness were 0.3, 0.1, 0.2 and 0.2 kg/d for lactation = 1 in herd A, lactation ≥ 2 in herd A, lactation = 1 in herd B, and lactation ≥ 2 in herd B, respectively. After the diagnosis of lameness, milk production was as much as 2.6 kg/d lower on average than production of control cows (Figure 1). The decrease was larger for cows in second or greater lactation than for those in first lactation.

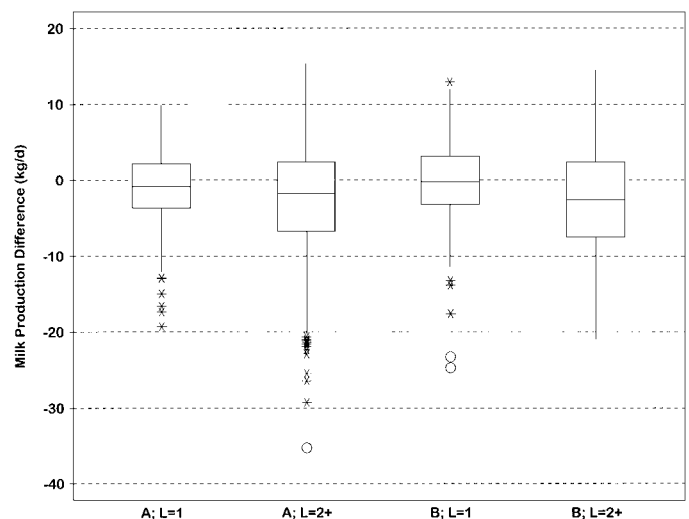


Figure 1. The distribution of average differences in milk production (kg/d) after the diagnosis of lameness for lame cows compared with cows that were not lame in the same herd, lactation group, and stage of lactation. The data are stratified by lactation = 1 in herd A (A; L = 1), lactation ≥ 2 in herd A (A; L = 2+), lactation = 1 in herd B (B; L = 1), and lactation ≥ 2 in herd B (B; L = 2+). (The box shows the interquartile range and is bisected by a line at the median value. An asterisk or O denote observations outside the quartiles by more than 1.5 and 3 times the interquartile range, respectively).

Table 2. Lameness treatment incidence rates by days in milk category for cows in two New York dairy herds.

DIM category	Herd A			Herd B		
	Lameness events	Cow-months at risk	Events/cow-month	Lameness events	Cow-months at risk	Events/cow-month
0–29	373	1796	0.21	46	724	0.06
30–59	291	1714	0.17	35	679	0.05
60–89	280	1654	0.17	28	652	0.04
90–119	253	1603	0.16	73	633	0.12
120–149	222	1513	0.15	52	600	0.09
150–179	198	1448	0.14	38	569	0.07
180–209	185	1364	0.14	26	515	0.05
210–239	132	1265	0.10	22	469	0.05
240–269	169	1153	0.15	17	407	0.04
270–299	103	1026	0.10	10	358	0.03
300–	289	896	...	47	324	...
Total study period	2495	16,489 ¹	0.15	394	6339 ¹	0.06

¹Calculated directly from total cow-months at risk for entire study period.

Herd A. Repeated measures ANOVA model results showed that cows lame at least one time during lactation had significantly lower milk production after the date of diagnosis (Table 5). Based on the analysis for all lame cows combined, milk production had decreased by an average of 1.5 kg/d by 2 wk after the lameness date compared with cows that had not been diagnosed as lame. This effect differed between lactation groups (lactation group \times lameness category interaction $P = 0.01$) and appeared to be larger for cows in second or greater lactation than for those in first lactation. The change in production was also larger for cows with higher severity scores (Table 6) and varied by diagnosis (Table 7). The results from separate models for each diagnosis suggested there was larger drop in production and longer duration of decreased milk production for cows with abscesses or sole ulcers than for foot rot, foot warts, or when no lesion was found. The effect of the stage of lactation in which lameness was first diagnosed was evaluated with separate models, as was done for the diagnosis categories. There was a statistically significant effect of lameness on milk production ($P <$

0.0001) for cows first diagnosed lame in each of three categories (0 to 90, 91 to 180, and > 180 DIM). The decreases in milk production after lameness diagnosis were similar for all three categories and were essentially the same as shown for all cows in Table 5.

Herd B. Lameness caused a significant decrease in milk production in herd B (-0.8 kg per day), but the decrease was smaller than in herd A. Although the interaction between lactation group and lameness category was not statistically significant ($P = 0.4$), the production loss followed the same pattern as herd A in that the decrease was greater in older cows (Table 8). In fact, the effect of lameness was not statistically significant in the analysis, of only first-lactation cows ($P = 0.6$). A large decrease in milk production (-4.7 kg/d) occurred for cows treated for lameness by farm employees on dates other than scheduled visits by the hoof trimmer (Table 9; lame between trimming dates). In contrast to the analysis of individual diagnoses in herd A, foot rot tended to have a longer and larger effect on production than sole ulcers or abscesses in herd B. For example, the estimated loss ≥ 3 wk after the diagnosis of foot rot in herd B was -2.0 kg/d compared with 0.8 and -1.0 kg/d for sole ulcers and abscesses, respectively. The effect of lameness was not statistically significant when analyzed separately for

Table 3. The distribution of herd A first lameness events by diagnosis.

Diagnosis	Number of cows affected	% of lame cows
No lesion found	333	36.0%
Sole ulcer	186	20.1%
Foot wart	117	12.6%
Abscess	103	11.1%
Foot rot ¹	76	8.2%
Laminitis	18	1.9%
Other conditions	35	3.8%
Multiple diagnoses	23	2.5%
Missing diagnosis	34	3.7%
Total	925	100.0%

¹Includes interdigital dermatitis and interdigital hyperplasia.

Table 4. The distribution of herd B first lameness events by diagnosis.

Diagnosis	Number of cows affected	% of lame cows
Foot wart	145	50.5%
Sole ulcer	50	17.4%
Foot rot	39	13.6%
Abscess	17	5.9%
Overgrown	11	3.8%
Other conditions	25	8.7%
Total	287	100.0%

Table 5. The effect of lameness on milk production (kg/d) for each lactation group and for all cows combined in herd A.

	First lactation ^{1†}		Second lactation ^{1†}		All cows ^{2†}	
Total cows	697		1099		1796	
Number lame	249		676		925	
Total observations	23,378		35,693		59,071	
Lame category level	β Estimate	(SE)	β Estimate	(SE)	β Estimate	(SE)
Before or never lame ³	0	...	0	...	0	...
Same week	-0.6	(0.2)	-1.0	(0.2)	-0.9	(0.1)
1 wk after	-0.8	(0.3)	-1.7	(0.2)	-1.4	(0.2)
2 wk after	-0.8	(0.3)	-1.8	(0.2)	-1.5	(0.2)
≥3 wk after	-0.7	(0.3)	-1.9	(0.3)	-1.5	(0.2)

¹Controlling for stage of lactation, week of milk weight measurement, and season of calving.

²Controlling for stage of lactation, lactation number, stage × lactation interaction, week of milk weight measurement, and season of calving.

³Parameter estimates represent differences in milk production at various times after lameness relative to the baseline level consisting of production for cows before lameness diagnosis and for cows that were never lame.

†Lame category $P < 0.05$.

three categories of DIM at first lameness, but milk production tended to decrease more for cows diagnosed lame during later stages of lactation.

DISCUSSION

The incidence rates of lameness treatment in the two study herds were higher than usually reported in the literature. The approximate number of lameness treatment events/cow-month were 0.15 and 0.06 for herds A and B, respectively. These incidence rates would fall in the top 25% of herd incidence rates reported from a prospective study in Britain (Clarkson et al., 1996). It should be recognized that, unlike the British study, we did not exclude repeated lameness treatments of the

same limb within 28 d from our estimates of incidence rates. The difference in incidence between the herds is partially due to the different ways in which lame cows were identified, treated, and defined for the study. In herd A, farm employees identified and treated the majority of lame cows, while in herd B, only a few individual cows were selected for treatment by regular farm employees. Most lame cows in that herd were treated during regularly scheduled visits by a professional hoof trimmer. We analyzed the effect of lameness on milk production separately for each herd because of these differences.

We found that lameness was more common in early lactation and was more likely to occur in older cows. The association with parity was consistent with prevalence

Table 6. The effect of lameness on milk production (kg/d) for cows with different lameness severity scores in herd A.¹

	Mild [†]		Moderate [†]		Severe [†]	
Total cows	1163		1194		1077	
Number lame	292		323		206	
Total observations	36,865		36,425		31,697	
Lame category level	β Estimate	(SE)	β Estimate	(SE)	β Estimate	(SE)
Before or never lame ²	0	...	0	...	0	...
Same week	-0.1	(0.2)	-1.1	(0.2)	-1.7	(0.3)
1 wk after	-0.8	(0.3)	-1.6	(0.3)	-2.4	(0.3)
2 wk after	-0.7	(0.3)	-1.6	(0.3)	-2.8	(0.4)
≥3 wk after	-0.9	(0.3)	-1.3	(0.3)	-2.8	(0.4)

¹Controlling for stage of lactation, lactation number, stage × lactation interaction, week of milk weight measurement, and season of calving. Separate models were used for each severity score, but cows never diagnosed lame were included in all three models.

²Parameter estimates represent differences in milk production at various times after lameness relative to the baseline level consisting of production for cows before lameness diagnosis and for cows that were never lame.

†Lame category $P < 0.05$.

Table 7. The effect of specific lameness diagnoses on milk production (kg/d) in herd A.¹

	Abscess [†]		Sole ulcer [†]		Foot rot [†]		Foot wart		No lesion [†]	
Total cows	974		1057		947		988		1204	
Number lame	103		186		76		117		333	
Total observations	28,847		31,422		27,549		29,204		38,193	
Lame category level	β	(SE)	β	(SE)	β	(SE)	β	(SE)	β	(SE)
Before or never lame ²	0	...	0	...	0	...	0	...	0	...
0 wk	-1.2	(0.4)	-0.9	(0.3)	-2.3	(0.5)	-0.4	(0.4)	-0.6	(0.2)
1 wk	-1.7	(0.5)	-2.2	(0.4)	-0.9	(0.6)	-1.2	(0.4)	-0.9	(0.3)
2 wk	-1.4	(0.5)	-2.5	(0.4)	-0.7	(0.6)	-0.8	(0.5)	-1.1	(0.3)
≥3 wk	-1.7	(0.5)	-2.3	(0.4)	-0.8	(0.6)	-0.6	(0.5)	-1.2	(0.3)

¹Controlling for stage of lactation, lactation number, stage × lactation interaction, week of milk weight measurement and season of calving. Separate models were used for each lameness diagnosis, but cows never diagnosed lame were included in all models.

²Parameter estimates represent differences in milk production at various times after lameness relative to the baseline level consisting of production for cows before lameness diagnosis and for cows that were never lame.

[†]Lame category $P < 0.05$.

studies in the United States (Warnick et al., 1995; Wells et al., 1993) and with a cohort study in Dutch dairy herds (Barkema et al., 1994). The Dutch study also reported higher incidence rates in early lactation as we observed (Barkema et al., 1994). The distribution of diagnoses in herd A was similar to previous studies; sole ulcer was the most common lesion identified (Barkema et al., 1994; Clarkson et al., 1996), but herd B had an unusually high incidence of digital dermatitis.

Lameness was associated with a significant decrease in weekly average milk production in both of the dairy herds participating in our study. Previous investigations of lameness effects on milk production have produced inconsistent results. The use of different measures of milk production, discrepant lameness definitions, varying susceptibility to bias due to culling,

variation in herd management, and analysis of milk loss by different statistical methods may have contributed to inconsistent results among studies. Contrary to expectations, associations of lameness with increased production have been identified (Dohoo and Martin, 1984; Rowlands and Lucey, 1986). Others found no significant effect of lameness on milk production (Cobo-Abreu et al., 1979; Martin et al., 1982). Culling bias may in part account for these results because cows with both lameness and low production would be expected to be culled more often than cows with lameness and high production. Culling bias is especially problematic in studying diseases such as lameness, which occur throughout lactation. This is because cows with higher milk production remain in the herd longer and have an increased chance of experiencing the disease at least

Table 8. The effect of lameness on milk production (kg/d) for each lactation group and for all cows combined in herd B.

	First lactation ¹		≥ Second lactation ^{1†}		All cows ^{2†}	
Total cows	330		394		724	
Number lame	104		183		287	
Total observations	10,540		12,594		23,134	
Lame category level	β Estimate	(SE)	β Estimate	(SE)	β Estimate	(SE)
Before or never lame ³	0	...	0	...	0	...
Same wk	-0.3	(0.3)	-0.4	(0.3)	-0.4	(0.2)
1 wk after	-0.4	(0.3)	-0.9	(0.3)	-0.8	(0.2)
2 wk after	-0.2	(0.4)	-1.1	(0.4)	-0.8	(0.3)
≥3 wk after	-0.1	(0.4)	-0.6	(0.5)	-0.5	(0.3)

¹Controlling for stage of lactation, week of milk weight measurement, and season of calving.

²Controlling for stage of lactation, lactation number, stage × lactation interaction, week of milk weight measurement and season of calving.

³Parameter estimates represent differences in milk production at various times after lameness relative to the baseline level consisting of production for cows before lameness diagnosis and for cows that were never lame.

[†]Lame category $P < 0.05$.

Table 9. The effect of specific lameness diagnoses on milk production (kg/d) in herd B.¹

	Abscess		Sole ulcer		Foot rot		Foot wart		Lame between trimming dates ^{2†}	
Total cows	454		487		476		582		465	
Number lame	17		50		39		145		28	
Total observations	13,124		14,367		13,833		17,980		13,434	
Category	β	(SE)	β	(SE)	β	(SE)	β	(SE)	β	(SE)
Before or never lame ³	0	...	0	...	0	...	0	...	0	...
0 wk	-1.6	(0.8)	-0.3	(0.5)	-1.0	(0.5)	0.1	(0.3)	-4.1	(0.7)
1 wk	-1.3	(1.0)	-0.3	(0.6)	-1.3	(0.6)	-0.3	(0.3)	-4.7	(0.9)
2 wk	-1.7	(1.2)	0.5	(0.7)	-2.0	(0.8)	-0.2	(0.4)	-4.6	(1.0)
≥3 wk	-1.0	(1.3)	0.8	(0.8)	-2.0	(0.9)	-0.3	(0.5)	-0.9	(1.1)

¹Controlling for stage of lactation, lactation number, stage × lactation interaction, week of milk weight measurement, and season of calving. Separate models were used for each lameness diagnosis, but cows never diagnosed lame were included in all models.

²Treated for lameness by farm employees on dates other than regularly scheduled hoof trimmer visit dates.

³Parameter estimates represent differences in milk production at various times after lameness relative to the baseline level consisting of production for cows before lameness diagnosis and for cows that were never lame.

†Lame category $P < 0.05$.

once. It may also be more difficult to detect changes in milk production using lactation-summary measures of production, particularly when the period of decreased production is short or if higher producing cows are more susceptible to lameness. The relationship between high milk production and lameness in some investigations supports the possibility that increased milk production is a risk factor for lameness (Barkema et al., 1994; Deluyker et al., 1991; Rowlands and Lucey, 1986).

A detrimental effect of lameness on milk production was observed in a few previous studies. A 350-kg decrease in milk yield per lactation was observed for cows with heel lesions, but was attributed to a decrease in the length of lactation (Rowlands and Lucey, 1986). In a study of lameness prevalence in a sample of Virginia dairy herds, cows classified as clinically lame had lower 305-d mature equivalent production (-320 kg; 95% CI -667, 27) than unaffected herdmates (Warnick et al., 1995). However, the effect of lameness on milk production may have been underestimated because of incorrect classification of cows that became lame before or after study herd visits. A prospective study in three French research herds found milk loss occurred following about 25% of cases of lameness with median losses of 440 and 270 kg for early lactation and mid-to late lactation, respectively (Coulon et al., 1996). Cows in these research herds were housed in tie-stall barns in the winter and were on pasture in summer, which limits the application of the results to cows in free-stall housing throughout the year. An economic analysis of data from 21 Dutch dairy farms estimated that cows culled for lameness had 3.3 kg/d lower milk production

than other cows, but found no effect on production among cows that were not culled for lameness (Enting et al., 1997). Our results were similar to estimated milk production losses of 1.5 to 2.8 kg/d within 2 wk after veterinary-diagnosed lameness in Finnish dairy cows (Rajala-Schultz et al., 1999).

We coded weekly average milk weights according to when they were recorded relative to the date of the first lameness event observed during lactation. The interpretation of the lameness category parameter estimates is the difference in daily milk production of lame cows during the same week of lameness, 1 wk after, 2 wk after, or 3 or more wk after lameness compared with milk production in the same calendar date, from cows in the same stage of lactation, season of calving, and lactation number, but before lameness was diagnosed. This comparison category included milk weights from cows that were never diagnosed lame during the study period. The parameter estimates for all cows and diagnoses in herd A were -0.9, -1.4, -1.5, and -1.5 for 0, 1, 2, and ≥3 wk after lameness, respectively. For example, these differences would result in a total lactation loss in milk production associated with lameness of about 295 kg for a cow that became lame at 100 DIM and continued to milk another 200 d. The analogous loss for a cow in herd B was 104 kg, which was lower than herd A because both the maximum decrease and the average decrease for the period ≥3 wk after lameness were smaller. This may be a result of differences in how lame cows were identified and treated or differences in the relative frequencies of specific causes of lameness between the herds. The changes in milk production

observed occurred in spite of treatment of lame cows with typical therapies.

The decrease in production was less in first-lactation cows than for those in greater than or equal to second lactation in both herds. This difference between lactation groups may result in part because older cows have higher milk production on average, and therefore greater decreases in production are possible. The herds were also similar in that cows diagnosed with digital dermatitis did not have decreased milk production when analyzed separately from other diagnoses. In herd A, sole ulcers were associated with the largest drop in milk production, followed by abscesses. Foot rot appeared to cause a significant short-term loss. This pattern of diagnosis-specific losses is consistent with the finding that cows with foot warts or foot rot returned to a normal pain response more quickly than those with sole ulcers or white line disease (Whay et al., 1998). It is plausible that an important mechanism for the decrease in milk production following lameness is lower feed intake associated with pain and reluctance to move to and stand at the feed bunk. Decreased feed intake associated with foot lesions has been described (Bareille et al., 2000).

Herd B differed from herd A in that sole ulcer and abscesses were not significantly associated with milk production when analyzed individually, possibly because of the smaller sample size and different lameness definitions. The milk loss was larger for more severe lameness in herd A and for cows treated between scheduled hoof trimmer visits in herd B. Treatment between scheduled hoof care dates most likely occurred for more severe cases in need of immediate treatment.

We encountered several problems frequently associated with the collection of data in commercial dairy herds. A comparison of studies with only owner-reported cases of lameness with studies of owner- and investigator-diagnosed cases shows that relying on owner reports alone may result in underestimating the occurrence of lameness. Wells et al. (1993) found that herd managers underestimated the prevalence of lameness relative to observations by study personnel. Nevertheless, prospective epidemiologic studies frequently rely on owner-diagnosed lameness because not all cases are treated by veterinarians (Whitaker et al., 1983) and the need to evaluate cows frequently over long observation periods. Studies based on owner- or farm worker-diagnosed disease also have the advantage of measuring diseases that are recognizable and of importance to dairy farmers (Waltner-Toews et al., 1986). This is particularly appropriate for diseases such as lameness, for which many cases are not diagnosed or treated by veterinarians.

Some differences in categorization occurred in spite of our attempts to standardize the definition of specific causes of lameness. For example, herd A personnel coded interdigital dermatitis and interdigital hyperplasia as foot rot. It is also likely that some lame cows were not treated and that some cows with normal gaits were coded as lame. The latter is particularly likely in herd B, where lameness was defined by lesions found by the hoof trimmer during scheduled visits when some of the cows were examined during routine corrective trimming. Variation in methods used to treat lame cows may have affected subsequent recovery and milk production. There also may have been errors in milk recording when cow identification numbers were not read correctly from transponders on entry in the milking parlor. We used weekly average milk production in an attempt to minimize the effect of missing or incorrect individual milk weights. Both errors in milk weight recording and misclassification of lame cows were likely to be independent of each other and of other study variables. Therefore, these types of errors would be expected to result in lameness effect estimates closer to zero, making our results somewhat conservative.

Another potential problem was that the intervals between weekly milk average recording were occasionally irregular. The first-order autoregressive covariance structure assumes that correlations decrease exponentially as the distance between measurements increases. This is most applicable when there are equal time intervals between repeated measurements from the same subject (Littell et al., 1996). However, it is unlikely that having some intervals from 8 to 16 d rather than 7 d seriously affected this assumption. Furthermore, the choice between first-order autoregressive and unstructured covariance structures for repeated measures ANOVA models similar to ours did not have a large effect on the point estimates for fixed effects (Gröhn et al., 1999).

The way we coded milk production values relative to the date of diagnosis of lameness did not allow a statistical analysis of the milk production of lame cows before diagnosis. This possibility was evaluated descriptively by calculating mean differences between milk production for lame cows with various DIM at onset of lameness compared with nonlame cows in the same stage of lactation and that remained in the herd at least as long as the lame cows. This showed that lame cows tended to have slightly higher milk production before the date of diagnosis than unaffected herd-mates. We found that excluding the lame cows from the baseline comparison group (as would be necessary to model prelameness milk production for lame cows) caused bias in the estimates of the association of lameness category with milk production. Therefore, weekly

milk production from both lame cows before the onset of lameness and cows that were not diagnosed lame were used as the comparison group in the repeated measures ANOVA models.

CONCLUSIONS

We found that milk production decreased significantly following diagnosis of lameness and in spite of treatment of affected cows in two dairy herds. The decrease was largest for cows in greater than or equal to second lactation and when the degree of lameness was judged by farm employees to be more severe. Foot rot and foot warts were associated with smaller decreases in production than sole ulcers or abscesses in one herd. In the other herd, cows with foot abscesses and foot rot tended to have larger decreases in milk production.

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