

J. Dairy Sci. 93:4045–4053 doi:10.3168/jds.2010-3062 © American Dairy Science Association[®]. 2010.

Association between milk yield and serial locomotion score assessments in UK dairy cows

S. C. Archer,¹ M. J. Green, and J. N. Huxley

University of Nottingham, School of Veterinary Medicine and Science, Sutton Bonington Campus, Sutton Bonington, Leicestershire, LE12 5RD, United Kingdom

ABSTRACT

This study investigated the effect of lameness, measured by serial locomotion scoring over a 12-mo period, on the milk yield of UK dairy cows. The data set consisted of 11,735 records of test-day yield and locomotion scores collected monthly from 1,400 cows kept on 7 farms. The data were analyzed in a multilevel linear regression model to account for the correlation of repeated measures of milk yield within cow. Factors affecting milk yield included farm of origin, stage of lactation, parity, season, and whether cows were ever lame or ever severely lame during the study period. Cows that had been severely lame 4, 6, and 8 mo previously gave 0.51 kg/d, 0.66 kg/d, and 1.55 kg/d less milk, respectively. A severe case of lameness in the first month of lactation reduced 305-d milk yield by 350 kg; this loss may be avoidable by prompt, effective treatment. Larger reductions can be expected when cases persist or recur. Evidence-based control plans are needed to reduce the incidence and prevalence of lameness in high yielding cows to improve welfare and productivity.

Key words: locomotion score, lameness, milk yield, dairy cow

INTRODUCTION

For the last 4 decades, mastitis control has received a high profile in the developed dairy industry worldwide (LeBlanc et al., 2006). In contrast, lameness prevention has received minimal attention; the associated decrease in milk production is often not tangible at the individual cow level and historically there have been no direct financial incentives from milk plants for lameness control in the UK. Farm assurance schemes and media interest are increasing consumer awareness of dairy cow welfare (FAWC, 2009). Compared with consumer demands for welfare assurance being imposed on dairy farmers, the need for lameness monitoring and control

Received January 7, 2010.

Accepted April 23, 2010.

¹Corresponding author: simon.archer@nottingham.ac.uk

may be more readily accepted as a priority if reliable data on the financial implications of locomotion score (\mathbf{LS}) on milk production were available.

In the absence of a standard definition for a clinical case of lameness, diagnosis remains a subjective opinion and the sensitivity of detection varied between observers (Whay et al., 2003). Farmers underestimated the prevalence of lameness in their herds and accurate records were rarely kept (Mill and Ward, 1994; Whay et al., 2003). Grading the locomotion of all cows in a herd using a standardized format was used to improve the sensitivity of lameness diagnosis and several scoring systems were described (Winckler and Willen, 2001; Cook, 2003; Whay et al., 2003). Recently, a 4-point LS scale similar to that described by Whay et al. (2003) was proposed as the UK industry standard (Anon, 2007; Bell and Huxley, 2009).

Most studies evaluating the association between lameness and milk yield were based on the ad hoc diagnosis of clinical lameness by farm staff. Earlier research reported no effect (Cobo-Abreu et al., 1979), a positive association (Deluyker et al., 1991), or variable results depending on factors such as the underlying lesions, stage of lactation, and method for assessing milk loss (Barkema et al., 1994; Coulon et al., 1996). Improvements in methodology incorporating repeated test-day yield (**TDY**) measurements have shown adverse associations between lameness and milk yield; losses were 424 kg/cow per 305-d lactation in one US herd (Bicalho et al., 2008). In 2 New York dairies with free stall housing, farm-specific decreases (1.5 and 0.8 kg/d after 2 wk) in daily yields were reported following lameness diagnosis (Warnick et al., 2001). In Finnish Ayrshire cows, losses of 1.5 to 2.8 kg/d were reported during the first 2 wk following diagnosis of lameness (Rajala-Schultz et al., 1999). A study on 5 UK farms demonstrated that decreased milk yield can extend from 4 mo before until 5 mo after diagnosis of clinical lameness, resulting in a mean reduction of 357 kg (95% CI: 163 to 552) per 305-d lactation (Green et al., 2002).

Literature on the effect of lameness assessed by LS on milk yield is limited. A single farm of 2 showed a significant decrease in milk yield of 1.89 kg/d for each unit increase in LS (Juarez et al., 2003), but this was based on 1 milk recording only. A decrease in milk yield associated with increasing LS in the first 100 DIM was demonstrated for a herd in Florida through comparison of 305-d milk yields; significant effects were demonstrated for cows in parity 2 and above with the most severe cases of lameness. These cows produced 874 kg less milk than multiparous cows that were not lame (Hernandez et al., 2005).

Comparison of 305-d milk yield is a crude technique for assessing production responses because small-scale deviations from the expected lactation curve may be missed and no temporal association between a lameness event and milk yield can be observed. Use of repeated individual TDY measurements as applied to clinical lameness (Warnick et al., 2001; Green et al., 2002; Bicalho et al., 2008) is a more sensitive technique for the analysis of milk loss associated with disease events (Gröhn et al., 1999). The objective was to apply TDY measurements to lameness assessed by LS.

MATERIALS AND METHODS

Herd Selection

Dairy herds were selected on the basis of location (Midlands, UK), with a minimum of 100 cows in milk at any time, and having herd managers committed to monthly milk recording through a single company (National Milk Records, Chippenham, UK) who were willing to participate in the study; 7 dairy herds were included. Herd health history including the prevalence or incidence of lameness was not part of the selection criteria.

Herd Management

Farm Details. The 7 herds had year-round calving patterns with annual rolling mean herd sizes of 125 to 335 cows and 305-d milk yields from 7,300 to 9,400 kg/cow (National Milk Records). The cows were predominantly conventionally managed Holstein-Friesians, although a single herd (farm 1; 130 cows) was organic and about a quarter of these cows were Holstein-Friesian-Montbeliarde crosses. All cows had access to pasture between March and October according to local conditions. A single herd (farm 7; 139 cows) was entirely loose-housed in straw pens throughout winter; in all other herds, milking cows were kept in free stall housing and dry cows were kept in straw pens. Housing varied from modern free stalls bedded with sand to older style, smaller free stalls with uneven clay bases. All farms had some straw pen housing or low stocking density free stalls available for freshly calved and sick cows.

Lameness Control. A record of the existing lameness control plan on each farm was made as farms were enrolled. Routine hoof trimming was carried out on all farms; 5 of the 7 herds employed a hoof trimmer. Cows identified as lame were treated by either farm staff or the hoof trimmer. All but 1 farm had endemic digital dermatitis and used foot bathing as a control measure. Dedicated roads for access to pasture were rare. Herd managers were made aware that the study was observational; no interventions were made and they were encouraged to continue with their existing management policies. None of these included routine LS assessment of cows.

Data Collection

Assessment of LS for all milking cows on each farm was conducted at monthly intervals for 12 mo between August 2008 and July 2009 by the first author. Afternoon visits were timed to coincide with the monthly milk recording date \pm 10 d. On farms 2, 3, 5, 6, and 7 all observations were carried out as cows exited the milking parlor; this allowed their identification numbers to be recorded in advance. On the remaining farms the herd managers were in attendance as the LS assessments took place and assisted with the identification of every cow; this was achieved as the cows were brought for milking on farm 1 and as they returned to the feed bunk from a postmilking holding pen on farm 4. All cows were observed walking on flat, nonslip concrete in a well-lit location that was consistent on each farm (Whay, 2002). The proposed UK industry standard 4-point LS scale was used: 0 = good locomotion (not lame; walks with even weight bearing and rhythm on all feet, with a flat back), 1 = imperfect locomotion(not lame; steps have uneven rhythm or weight bearing, strides may be shortened, affected limb or limbs not immediately identifiable), 2 =impaired locomotion (lame; uneven weight bearing on immediately identifiable affected limb or limbs, shortened strides and often an arched back), and 3 = severely impaired locomotion (severely lame; as score 2, but cannot keep up with the healthy herd or a brisk human walking pace; Whay et al., 2003; Huxley et al., 2004; Barker et al., 2010). The first author was trained to use the locomotion scoring system by a coauthor (JH) with on-farm experience; practice locomotion scoring sessions were carried out on all 7 study farms. A check visit was conducted after 6 mo to ensure the scores had not drifted over time. Freeze brand numbers were recorded to match each cow to her score; data were spoken into a dictation recorder and transcribed later when cow flow was too rapid to record the information directly. Event data (dates of any clinical disease cases, drying off or culling for identified individual cows) that appeared in the farm records were collected.

Herd owners gave permission to access cow details (calving date and parity), TDY, and milk composition data via the Internet (www.nmr.co.uk) on a monthly basis. These data were edited in InterHerd (version 2.11.0, InterAgri, Reading, UK) and Microsoft Excel 2003 (Microsoft Corp., Redmond, WA) and then collated with LS and disease event data in Microsoft Access 2003 (Microsoft Corp.).

Data Analysis

A minimum of 1 and up to 12 recordings of TDY and LS were available for each cow. Score 0 was rarely assigned and those were grouped with LS 1 for the analysis. Categorical variables were constructed as columns for month of lactation (1 to 12+), parity (1 to 8+), season (quarter 1: Aug 08 to Oct 08; quarter 2: Nov 08 to Jan 09; quarter 3: Feb 09 to Apr 09; quarter 4: May 09 to Jul 09), and LS related to current, previous (from 1 to 11 mo before), and future (from 1 to 11 mo after) months. Binary variables were used to identify cows that were ever lame and ever severely lame during the study period (Green et al., 2002); these took the value 1 if a cow had ever been LS 2 or LS 3, otherwise it was 0. Descriptive statistical analysis was conducted in Minitab (Minitab Inc., State College, PA) and subsequent multilevel modeling was conducted in MLwiN 2.11 (Rasbash et al., 2008).

Test-day yield was the outcome variable used in multilevel models and it was distributed normally. A 2-level model was used to analyze the data to account for clustering of recordings (level 1) within cows (level 2), and model parameters were estimated using the iterative generalized least squares procedure (Goldstein, 2003). Farm of origin was forced into the model as fixed effects. The model took the form

$$\begin{split} y_{ij} &= \alpha + \Sigma \beta_{ij} \mathbf{X}_{ij} + \Sigma \Delta_j \mathbf{Z}_j + v_j + e_{ij} \\ v_j \sim & N \; (0, \; \sigma^2_{\; v}) \\ e_{ij} \sim & N \; (0, \; \sigma^2_{\; e}), \end{split}$$

where $y_{ij} = TDY$ i for cow j; $\alpha =$ intercept value; $\mathbf{X}_{ij} =$ a vector of exposure variables for TDY ij; $\beta_{ij} =$ coefficients for \mathbf{X}_{ij} ; $\mathbf{Z}_j =$ a vector of exposure variables for cow j; $\Delta_j =$ coefficients for \mathbf{Z}_j ; $\Sigma =$ sum of 1 to n exposure variables, $v_j =$ random effect to reflect re-

sidual variation between cows (assumed to be a normal distribution with mean = 0 and variance = σ_v^2); and e_{ij} = residual level 1 error (assumed to be a normal distribution with mean = 0 and variance = σ_e^2).

Variation of fixed covariates between cows was investigated, as were interactions between model fixed effects. Fixed effects and interactions were included in the model if $P \leq 0.05$. Goodness of fit was assessed using conventional plots of standardized residuals and examining the influence and leverage of data values (Rasbash et al., 2008).

RESULTS

The mean farm-level prevalence of lameness (cows with LS 2 and 3) in the study population between August 2008 and July 2009 was 62%, with a range between farms of 48 to 72%. From 11,735 records, 1.7% were LS 0, 34.6% were LS 1, 37.1% were LS 2, and 26.6% were LS 3. More than 93% of cows were LS 2 or 3 on at least 1 visit; the median number of lame scores for each cow was 5, with an interquartile range from 3 to 8. For cows assessed in consecutive months within lactation throughout the study period, approximately 50% were chronic cases (lame on both occasions), 13% became new cases, 15% recovered, and 22% were unaffected by lameness. Locomotion score data for 258 cows were available for months immediately before and after a dry period; of these, 57% were chronic cases, 16% were new cases, 18% recovered, and 9% were unaffected by lameness.

Results from the multilevel models indicated that factors affecting milk yield included farm of origin, stage of lactation, parity, and season (Table 1). Cows that were ever LS 2 had milk yields that were 2.34 ± 0.36 kg/d higher than those that were never lame, and cows that were ever LS 3 had mean milk yields that were 1.09 ± 0.35 kg/d higher than those that were never lame. Lactation curves for cows that were ever lame (LS 2 or 3) and those that were never lame are shown in Figure 1; 64% of the cows that were never lame were in parity 1 and 21% were in parity 2, and 80% of cows that were ever lame were distributed more evenly between parities 1 and 4.

Locomotion scoring did not have a significant effect on milk yield recorded within 10 d of the lameness assessment. Cows in late lactation that were LS 3 4 mo previously gave 0.51 ± 0.23 kg/d less milk, cows that were LS 3 6 mo previously gave 0.66 ± 0.31 kg/d less milk, and cows that were LS 3 8 mo previously gave 1.55 ± 0.46 kg/d less milk than cows that were not lame at these times. Of 4,945 LS assessments made 4 mo previously, 3 records were associated with test days

ARCHER ET AL.

Table 1. Final model of repeated test-day milk yield (kg) within cow (response); effect of the fixed and random	m
effects ¹	

Item	Mean effect	SE	Lower 95% CI	Upper 95% CI
Fixed effect				
Intercept	23.422	0.789	21.876	24.968
Farm				
2	1.853	0.650	0.579	3.127
3	-0.983	0.618	-2.194	0.228
4	8.400	0.646	7.134	9.666
5	1.602	0.547	0.530	2.674
6	3.300	0.651	2.024	4.576
7	3.918	0.641	2.662	5.174
Parity				
2	0.809	0.275	0.270	1.348
3	3.365	0.347	2.685	4.045
4	5.910	0.416	5.095	6.725
5	6.349	0.476	5.416	7.282
6	4.717	0.568	3.604	5.830
$\ddot{7}$	4.491	0.69	3.139	5.843
8+	3.555	0.767	2.052	5.058
Month of lactation	0.000	0.101	2.002	0.000
2	3.476	0.238	3.010	3.942
3	2.350	0.400	1.566	3.134
4	0.614	0.243	0.138	1.090
5	-1.169	0.249	-1.683	-0.655
6	-2.935	0.202 0.274	-3.472	-2.398
7	-4.892	0.214	-5.445	-4.339
8	-7.041	0.282	-7.609	-6.473
9	-9.291	0.290	-9.863	-8.719
10	-11.922	0.304	-12.518	-11.326
10	-11.322 -14.265	0.328	-14.908	-13.622
$11 \\ 12+$	-14.203 -16.296	0.318	-16.919	-15.673
Quarter of study	-10.230	0.516	-10.313	-15.075
Nov 08 to Jan 09	0.465	0.158	0.155	0.775
Feb 09 to Apr 09	2.864	0.108	2.470	3.258
May 09 to Jul 09	3.241	0.239	2.470 2.773	3.709
Ever locomotion score 2	2.336	0.239 0.357	1.636	3.036
Ever locomotion score 3	2.330	0.357 0.348	0.409	5.050 1.773
	1.091	0.548	0.409	1.775
Locomotion score -4 mo 2	-0.245	0.184	-0.606	0.116
2 3	-0.245 -0.505	$0.184 \\ 0.226$	-0.948	-0.062
-	-0.303	0.220	-0.948	=0.002
Locomotion score -6 mo 2	0.42	0.941	0.002	0.042
2 3	-0.43	0.241	-0.902	
~	-0.663	0.309	-1.269	-0.057
Locomotion score -8 mo	0.710	0.970	1 459	0.099
2 3	-0.710	0.379	-1.453	0.033
9	-1.552	0.464	-2.461	-0.643
Random effect	Varianc	e		SE
Level 2 (cow)	28.551			1.234
Level 1 (test day)	25.440)	(0.364

¹Total log-likelihood from model = -35,448.999; null model = -40,224.849.

in the first 3 mo of lactation; of 2,689 LS assessments made 6 mo previously, 3 records were associated with test days in the first 5 mo of lactation; and of 1,121 LS assessments made 8 mo previously, 2 records were associated with test days in the first 7 mo of lactation, indicating a negligible contribution of data from previous lactations spanning the dry period. Cows that were assessed as LS 3 after a particular test day gave significantly more milk at that test day than those that were sound. These terms were confounded by the ever lame and ever severely lame parameters and have not been included to maintain model parsimony.

Variation in the shape of lactation curves between cows was evident as interactions between parity and stage of lactation, parity and farm of origin, and stage of lactation and farm of origin ($P \le 0.05$). Cows that were LS 3 on 2 particular farms had significantly higher test-day yields than the baseline. One of these herds was the highest yielding (farm 4: mean TDY = 33 kg; 25 to 29 kg for the other herds); the other was the largest

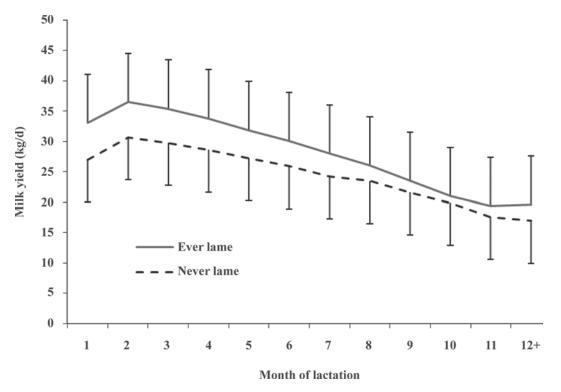


Figure 1. Lactation curves of daily milk yield with positive and negative standard deviation for cows that were ever lame (locomotion score 2 or 3; 80% of cows that were ever lame were distributed evenly between parities 1 and 4) and cows that were never lame (64% of cows that were never lame were in parity 1 and 21% were in parity 2).

herd (farm 5: mean number of cows scored per month = 275; 102 to 133 for the other herds). These interactions did not influence the fixed effects of interest and were not included in the final model. Diagnostics from the developed model indicated that it was a good fit to the data (Figure 2).

To summarize the estimates of milk loss associated with severe lameness, TDY results from Table 1 were scaled up to assess their effect on 305-d lactation yield (Table 2). In addition to the associations at 4, 6, and 8 mo following the observation of severe lameness from the model ($P \leq 0.05$), it is assumed adverse effects on TDY occurred 5, 7, 9, and 10 mo after initial diagnosis. The assumed TDY effects were constant within each 30.5-d month and were equal to those in the preceding month with a significant result. Regardless of previous lameness history, Table 2 shows that when severe lameness does not persist beyond a single monthly locomotion assessment, the additive effect of TDY on 305-d yield decreases as the month of lactation when LS 3 is recorded advances. In an extreme case, cows observed severely lame in the first month of lactation could have their potential 305-d milk yield reduced by 350 kg (95%) CI: 81 to 620); this applied to 26% of cows that were assessed in the first month of lactation in the current data set.

Table 3 demonstrates that the TDY effects have a cumulative effect on potential 305-d yield for persistent or recurrent severe lameness cases that increase with the length of time for which cows are severely lame. For example, if a cow is severely lame in previous months, TDY was reduced according to the additive effect of the associations that occur throughout the entire period of time affected. If a cow was recorded as LS 3 for the first 2 consecutive months of lactation, her mean potential 305-d milk yield could be reduced by 620 kg (95% CI: 140 to 1,100); this applied to 63% of those cows that were severely lame in the first month of lactation.

DISCUSSION

This study demonstrated that at the level of the individual cow, severe lameness (LS 3) in early lactation had a negative influence on TDY; however, this does not occur until at least 4 mo after the identification of lameness takes place. A reduction in daily milk yield was not observed within 10 d of a lameness event and, thus, TDY is a meager predictor of locomotion impairment. As a result, the need for treatment of lame cows may not be perceived as urgent by farmers or their advisors.

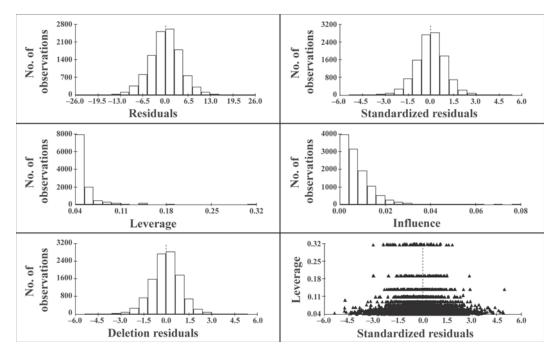


Figure 2. Plots of residuals including the influence and leverage of data values for the developed model.

Because significant adverse effects on TDY were demonstrated 4, 6, and 8 mo after the observation of severe lameness, it is plausible that a trend exists in UK dairy cows that there are adverse effects on milk yield 5, 7, 9, and 10 mo later. Incidence of clinical lameness peaked in early lactation (Green et al., 2002); the current study indicated that the largest reductions in 305-d yield were associated with cases of severe lameness early in lactation (Table 2) and their persistence (Table 3).

As was shown previously for cases of clinical lameness (Deluyker et al., 1991; Green et al., 2002; Bicalho et al., 2008), cows that were ever lame in this study had higher mean TDY throughout lactation than those that were never lame (Figure 1); lameness is unequivocally a production disease associated with high yield, which is more likely observed in multiparous cows. Figure 1 should be interpreted with care because it is part of the descriptive results and not derived from the model; therefore, it does not account for the influences of parity, season, and farm of origin.

Those cows ever severely lame (LS 3) belong to a population that produced an additional 330 kg (95% CI: 120 to 540) per 305-d lactation compared with cows that were never lame. Eighty percent of cows that were ever severely lame were LS 2 on at least one occasion; this was associated with the further mean production of more than twice this amount of milk again [710 kg (95% CI: 500 to 930) per 305-d lactation], and therefore, these cows produced an additional 1,040 kg (95%

CI: 620 to 1,470) per 305-d lactation compared with cows that were never lame. Risk factors that confound the relationship between lameness and high milk yield require careful management for dairy herds to operate efficiently and minimize lameness incidence. Further research is required to fully identify critical control points for lameness in the management of high genetic merit cows. Areas for consideration include lying times (Cook and Nordlund, 2009), control of intercurrent disease, DMI, and associated changes in BCS after calving.

The net effect of nonpersistent lameness in early lactation at the herd level is that 305-d yield decreases toward that for average cows that are never lame, so the full potential benefit of high yield is partially lost. Cows experiencing chronic or recurrent severe lameness commencing in the first, second, and third months of lactation more likely have a tangible decrease in 305-d milk yield at the herd level compared with cows that are never lame (Table 3). Despite welfare concerns, this may be one of the reasons farmers are unlikely to perceive lame cows as uneconomical when culling decisions are influenced by milk yield. The halving of the apparent production response associated with cows that are ever LS 2 compared with those ever LS 3 indicated that under conventional management lame cows had the ability to increase TDY further still. This augments welfare concerns if it contributes to the retention of lame cows in our dairy herds.

Alongside a trend of increasing annual milk yield per cow (Anon, 2009), scoring locomotion of UK cows

Month of lactation in which a cow is locomotion score 3	Month of lactation in which reduction in milk yield occurs							
	4	5	6	7	8	9	10	Total
1	-15.40	-15.40	-35.62	-35.62	-82.96	-82.96	-82.96	-350.9
2	(-1.89 to -28.91)	(-1.89 to -28.91) -15.40) $(-3.64 \text{ to } -67.61)$ -15.40	(-23.24 to -67.61) -35.62	(-23.24 to -142.68) -35.62	(-23.24 to -142.68) -82.96	(-23.24 to -142.68) -82.96	(-80.8 to -621.1) -268.0
3		(-1.89 to -28.91)	-15.40	-15.40	(-3.64 to -67.61) -35.62	(-23.24 to -142.68) -35.62	(-23.24 to -142.68) -82.96	-185.0
4			(-1.89 to -28.91)	(-1.89 to -28.91) -15.40 (-1.80 to -28.01)	-15.40	(-3.64 to -67.61) -35.62 (-2.64 to -67.61)	(-23.24 to -142.68) -35.62 (-2.64 to -67.61)	(-34.3 to -335.7) -102.1 (-11.1 to -102.0)
5				(-1.89 to -28.91)	(-1.89 to -28.91) -15.40 (-1.89 to -28.91)	(-3.64 to -67.61) -15.40 (-1.89 to -28.91)	(-3.64 to -67.61) -35.62 (-3.64 to -67.61)	(-11.1 to -193.0) -66.4 (-7.4 to -125.4)
6					(-1.89 to -28.91)	$(-1.89\ t0\ -28.91)$ -15.40	$(-3.04\ 10\ -07.01)$ -15.40	$(-7.4\ 10\ -125.4)$ -30.8
7						(-1.89 to -28.91)	(-1.89 to -28.91) -15.40 (-1.89 to -28.91)	(-3.8 to -57.8) -15.4 (-1.9 to -28.9)

Table 2. Estimated mean (95% CI) effect of severe lameness¹ (locomotion score 3) on potential 305-d milk yield (kg) based on model predictions and assumptions²

¹If severe lameness does not persist.

 2 Significant associations from the model occurred 4, 6, and 8 mo following the observation of severe lameness; based on these, additional adverse effects on milk yield have been assumed to occur after 5, 7, 9, and 10 mo.

Month of lactation in which locomotion score 3 is initially identified	No. of months of lactation spent chronically lame							
	1	2	3	4	5	6	7	
1	-350.9	-618.9	-803.9	-906.0	-972.4	-1003.2	-1,018.6	
0	(-80.8 to -621.1)	(-138.3 to -1,099.5)	(-172.6 to -1,435.2)	(-183.3 to -1,628.2)	(-191.1 to -1,753.6)	(-194.9 to -1,811.4)	(196.8 to -1,840.3)	
2	-268.0 (-57.5 to -478.4)	-453.0 (-91.8 to -814.1)	-555.0 (-102.9 to -1,007.1)	-621.5 (-110.3 to -1,132.5)	-652.3 (-114.1 to -1,190.3)	-667.7 (-116.0 to -1.219.2)		
3	-185.0	-287.1	-353.5	-384.3	-399.7	(11010 00 1,21012)		
	(-34.3 to -335.7)	(-45.4 to -528.7)	(-52.8 to -654.1)	(-56.6 to -711.9)	(-58.5 to -740.8)			
4	-102.1 (-11.1 to -193.0)	-168.5 (-18.5 to -318.4)	-199.3 (-22.3 to -376.2)	-214.7 (-24.2 to -405.1)				

Table 3. Cumulative reduction in potential 305-d milk yield (95% CI) associated with chronic¹ severe lameness (locomotion score 3) for cases commencing at the start of the first, second, third, and fourth months² of lactation based on model predictions and assumptions³

¹Includes both persistent and recurrent cases of lameness.

 2 Cases of severe lameness that commence later than the third month of lactation were unlikely associated with a net reduction in 305-d milk yield at the herd level given that cows that are ever locomotion score 3 produce a mean additional 330 (95% CI: 120 to 540) kg per 305-d lactation.

³Significant associations from the model occurred 4, 6, and 8 mo following the observation of severe lameness; based on these, additional adverse effects on milk yield have been assumed to occur after 5, 7, 9, and 10 mo.

by independent observers has provided mean (range) prevalence estimates of 20.6% (2.0 to 53.9; Clarkson et al., 1996), 22.1% (0 to 50.0; (Whay et al., 2003), and 24.2% (6.8 to 55.6; Huxley et al., 2004) to 36.8%(0 to 79.2; Barker et al., 2010). Although considerably higher in comparison, the mean (range) prevalence of lameness (LS 2 and 3) in the current study [62%] (48 to 72) is within the wide ranges previously reported. Because lameness prevalence and incidence were not selection criteria for inclusion, farmers that were willing to participate could more likely have problems with lameness in their herds. Because the 7 farms were a geographically clustered convenience sample and were under the care of a single veterinary practice, it may not be possible to generalize the results from this study to all UK dairy farms or those with a lower prevalence of lameness.

Even with a single observer, consistent serial locomotion scoring is difficult to achieve (Channon et al., 2009). In this study cows were observed walking in their normal routine on each farm. Despite this, the observer's presence evoked suspicion in some cows, leading to an alteration in their gait. Because the cows were managed and moved around in groups, the number of strides observed for every cow was not consistent, nor was the observer's view of each individual animal. Misidentification of moving cows was possible where brand marks were not easy to read, although recording cow identity in the parlor before observing locomotion as they exited minimized this source of error. On farms where this method was not used, only experienced farm staff assisted in the identification of moving cows.

It is possible that not all confounding factors have been identified; records of intercurrent disease were not complete for all farms and have not been included in the analysis. If the effect of lameness on TDY is indeed delayed as suggested here, further studies focusing on the effect of LS on lifetime production would help to fully evaluate the economic impact of lameness in dairy herds.

CONCLUSIONS

In the 7 dairy herds, severely lame cows failed to achieve their potential 305-d milk yields as a result of a progressive decrease in TDY from 4 mo after the occurrence. Regular LS assessment of cows to facilitate the early identification of lameness cases for treatment is a strategy to mitigate this effect. Because cows that are ever lame are the highest yielding animals, the net effect at the herd level may only be that the milk yield of lame cows is reduced toward that for average cows that are never lame. Where culling decisions are influenced by milk yield, the retention of lame cows in dairy herds is a current welfare concern.

ACKNOWLEDGMENTS

Simon Archer is a Resident in Production Animal Medicine part funded by the RCVS Trust (London, UK). Martin Green is supported by a Wellcome Trust (London, UK) Intermediate Clinical Fellowship. The authors gratefully acknowledge their funding bodies and the help and cooperation of the farmers and herdspersons involved in the study.

REFERENCES

- Anon. 2007. DairyCo mobility score. http://cattle-lameness.org.uk/ contentdocs/DairyCoMobilityScore.pdf Accessed Jun. 13, 2010.
- Anon. 2009. Dairy statistics. An insider's guide 2009. http://www. dairyco.org.uk/library/market-information/dairy-statistics/dairystatistics-an-insider's-guide-2009.aspx Accessed Jun. 13, 2010.
- Barkema, H. W., J. D. Westrik, K. A. S. van Keulen, Y. H. Schukken, and A. Brand. 1994. The effects of lameness on reproductive performance, milk production and culling in Dutch dairy farms. Prev. Vet. Med. 20:249–259.
- Barker, Z. E., K. A. Leach, H. R. Whay, N. J. Bell, and D. C. J. Main. 2010. Assessment of lameness prevalence and associated risk factors in dairy herds in England and Wales. J. Dairy Sci. 93:932–941.
- Bell, N., and J. N. Huxley. 2009. Locomotion, lameness and mobility in dairy cows. Vet. Rec. 164:726.
- Bicalho, R. C., L. D. Warnick, and C. L. Guard. 2008. Strategies to analyze milk losses caused by diseases with potential incidence throughout the lactation: A lameness example. J. Dairy Sci. 91:2653–2661.
- Channon, A. J., A. M. Walker, T. Pfau, I. M. Sheldon, and A. M. Wilson. 2009. Variability of Manson and Leaver locomotion scores assigned to dairy cows by different observers. Vet. Rec. 164:388– 392.
- Clarkson, M. J., W. B. Faull, J. W. Hughes, F. J. Manson, J. B. Merritt, R. D. Murray, J. E. Sutherst, W. R. Ward, D. Y. Downham, and W. B. Russell. 1996. Incidence and prevalence of lameness in dairy cattle. Vet. Rec. 138:563–567.
- Cobo-Abreu, R., S. W. Martin, R. A. Willoughby, and J. B. Stone. 1979. The association between disease, production and culling in a university dairy herd. Can. Vet. J. 20:191–195.
- Cook, N. B. 2003. Prevalence of lameness among dairy cattle in Wisconsin as a function of housing type and stall surface. J. Am. Vet. Med. Assoc. 223:1324–1328.
- Cook, N. B., and K. V. Nordlund. 2009. The influence of the environment on dairy cow behavior, claw health and herd lameness dynamics. Vet. J. 179:360–369.
- 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.
- 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 dairy herd. J. Dairy Sci. 74:436–445.
- FAWC. 2009. Farm animal welfare council opinion on the welfare of the dairy cow. http://www.fawc.org.uk/pdf/dcwelfar-091022.pdf Accessed Dec. 10, 2009.
- Goldstein, H. 2003. Multilevel Statistical Models. 3rd ed. Arnold, London, UK.
- Green, L. E., V. J. Hedges, Y. H. Schukken, R. W. Blowey, and A. J. Packington. 2002. The impact of clinical lameness on the milk yield of dairy cows. J. Dairy Sci. 85:2250–2256.
- Gröhn, Y. T., J. J. McDermott, Y. H. Schukken, J. A. Hertl, and S. W. Eicker. 1999. Analysis of correlated continuous repeated

observations: Modelling the effect of ketosis on milk yield in dairy cows. Prev. Vet. Med. 39:137–153.

- Hernandez, J. A., E. J. Garbarino, J. K. Shearer, C. A. Risco, and W. W. Thatcher. 2005. Comparison of milk yield in dairy cows with different degrees of lameness. J. Am. Vet. Med. Assoc. 227:1292– 1296.
- Huxley, J. N., J. Burke, S. Roderick, D. C. J. Main, and H. R. Whay. 2004. Animal welfare assessment benchmarking as a tool for health and welfare planning in organic dairy herds. Vet. Rec. 155:237– 239.
- Juarez, S. T., P. H. Robinson, E. J. DePeters, and E. O. Price. 2003. Impact of lameness on behavior and productivity of lactating Holstein cows. Appl. Anim. Behav. Sci. 83:1–14.
- LeBlanc, S. J., K. D. Lissemore, D. F. Kelton, T. F. Duffield, and K. E. Leslie. 2006. Major advances in disease prevention in dairy cattle. J. Dairy Sci. 89:1267–1279.
- Mill, J. M., and W. R. Ward. 1994. Lameness in dairy cows and farmers' knowledge, training and awareness. Vet. Rec. 134:162–164.

- Rajala-Schultz, P. J., Y. T. Gröhn, and C. E. McCulloch. 1999. Effects of milk fever, ketosis, and lameness on milk yield in dairy cows. J. Dairy Sci. 82:288–294.
- Rasbash, J., F. Steele, W. Browne, and H. Goldstein. 2008. A User's Guide to MLwiN Version 2.10. Centre for Multilevel Modelling, University of Bristol, Bristol, UK.
- Warnick, L. D., D. Janssen, C. L. Guard, and Y. T. Gröhn. 2001. The effect of lameness on milk production in dairy cows. J. Dairy Sci. 84:1988–1997.
- Whay, H. 2002. Locomotion scoring and lameness detection in dairy cattle. In Pract. 24:444–449.
- Whay, H. R., D. C. J. Main, A. J. F. Webster, and L. E. Green. 2003. Assessment of the welfare of dairy cattle using animal-based measurements: Direct observations and investigation of farm records. Vet. Rec. 153:197–202.
- Winckler, C., and S. Willen. 2001. The reliability and repeatability of a lameness scoring system for use as an indicator of welfare in dairy cattle. Acta Agric. Scand. Anim. Sci. 30:103–107.