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Association between somatic cell count and serial locomotion score assessments in UK dairy cows

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ABSTRACT

This research investigated the effect of lameness, measured by locomotion score (LS) on the somatic cell count (SCC) of UK dairy cows. The data set consisted of 11,141 records of SCC and LS collected monthly on 12 occasions from 1,397 cows kept on 7 farms. The data were analyzed to account for the correlation of repeated measures of SCC within cow. Results were controlled for farm of origin, stage of lactation, parity, season, and test-day milk yield. Compared with the geometric mean SCC for cows with LS 1 on each farm, cows on farm 3 with LS 2 produced milk with 28,000 fewer somatic cells/mL, and cows with LS 2 on farm 6 produced milk with 30,000 fewer somatic cells/mL at a test day within 10 d. Cows that would have LS 3 six months later produced milk with 16,000 fewer somatic cells/mL compared with the geometric mean SCC for cows that would have LS 1 in 6 mo time. These results illustrate differences in disease dynamics between farms, highlight potential conflict between lameness and mastitis control measures, and emphasize the importance of developing farm-specific estimates of disease costs, and hence, health management plans in clinical practice.
Key words: locomotion score, somatic cell count, dairy cow

INTRODUCTION

Direct financial incentives are common to encourage the hygienic production of milk. For example, the European Commission Milk Hygiene Directive (92/46) imposes an upper geometric mean SCC limit on bulk milk destined for human consumption of 400,000 cells/mL over 3 mo, and milk purchasers often pay a premium for higher quality milk with low SCC (Bradley, 2002). As a result, controlling mastitis incidence and prevalence is a high priority, as has been the case for the last 4 decades throughout the developed dairy industry worldwide.

In contrast, lameness prevention has received less attention and historically, no direct financial incentives have existed from milk buyers for lameness control in the UK, where herd level lameness prevalence was 36.8%, but ranged from 0 to 79.2% (Barker et al., 2010). Farm assurance schemes are increasing consumer awareness of dairy cow welfare (FAWC, 2009) and the UK dairy industry has specified a standard scoring system for assessing the locomotion of cows to improve the sensitivity of lameness diagnosis (DairyCo, 2007). This has mostly been used as a regulatory tool to satisfy consumer demands for welfare assurance. The need for lameness monitoring, early treatment of affected cows, and herd-level control may be more readily accepted if reliable data on the financial implications of locomotion score (**LS**) on both milk yield and SCC were available. 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 decrease of 357 kg (95% CI: 163 to 552) per 305-d lactation (Green et al., 2002). The current authors investigated the association between serial LS and test day milk yield (**TDY**). Significant reductions in TDY were demonstrated commencing 4 mo after severe lameness was observed (Archer et al., 2010). Given the importance of payments for low SCC to economic milk production on some farms (Halasa et al., 2007), a comparable study on the association between SCC and LS assessments throughout lactation would be useful to estimate the true costs of lameness, and to investigate the nature of the relationship between lameness and mastitis.

The relationship between mastitis and lameness is unclear. Based on 10 dairy herds in the southwest of England, clinical lameness before first service was associated with a 1.4-fold increase in the odds of clinical mastitis (Peeler et al., 1994), although cows with sole ulcers at claw trimming on 102 Swedish dairy herds did not have higher odds of mastitis or high SCC than did unaffected cows (Hultgren et al., 2004). The aim was

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to quantify the association between LS and SCC in 7 UK dairy herds.

MATERIALS AND METHODS

Study Design

A database containing monthly LS, TDY, and SCC records collected over 12 mo between August 2008 and July 2009 was available from a previous study (Archer et al., 2010). The data were from 1,397 cows in a convenience sample of 7 dairy herds (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. Herd health history including the prevalence or incidence of lameness was not part of the selection criteria.

The first author assessed LS for all lactating cows within 10 d of a test day at monthly intervals for 12 mo between August 2008 and July 2009. All cows were observed walking on flat, nonslip concrete in a well-lit location that was consistent on each farm. A 4-point LS scale was used (DairyCo, 2007): 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). Freeze brand numbers were recorded to match each cow to her score. Herd details, lameness control policies, and detailed data collection methods have been described previously (Archer et al., 2010). All cows were milked twice daily through herring-bone style parlors.

Data Analysis

A minimum of 1 and up to 12 recordings of SCC and LS were available for each cow lactation. Score 0 was rarely assigned (1.7% of recordings) and these were grouped with score 1 for the analysis. Categorical variables were constructed for month of lactation (1 to 12+), parity (1 to 8+), season (quarter 1: August 08 to October 08; quarter 2: November 08 to January 09; quarter 3: February 09 to April 09; quarter 4: May 09 to July 09), and LS related to current (within 10 d), previous (from 1 to 11 mo before), and future (from 1 to 11 mo after) monthly recordings. The TDY for current, previous (from 1 to 3 mo before), and future months (from 1 to 3 mo after) was included as a continuous variable to account for a possible confounding effect of milk yield on SCC (Green et al., 2006). Two binary variables were investigated to identify cows that were ever lame (LS 2) and ever severely lame (LS 3) during the study period (Green et al., 2002) and these took the value 1 if a cow ever had LS 2 or LS 3, respectively; otherwise, it was 0. Descriptive statistical analyses were conducted in Minitab (Minitab Inc., State College, PA) and subsequent multilevel modeling in MLwiN 2.11 (Rasbash et al., 2008).

As the SCC distribution was right skewed, a $\log_{10}$ transformation was applied to normalize the data (Ali and Shook, 1980). Log_{10} SCC was the outcome variable used for multilevel linear regression; a 2-level model accounted for clustering of recordings (level 1) within cows (level 2), and parameters were estimated using the iterative generalized least squares procedure (Goldstein, 2003). Farm of origin was forced into the models as categorical fixed effects. The model took the form

$$y_{ij} = \alpha + \mathbf{X_{ij}}\boldsymbol{\beta_1} + \mathbf{X_j}\boldsymbol{\beta_2} + v_j + e_{ij}$$
$$v_j \sim N\left(0, \sigma_v^2\right)$$
$$e_{ij} \sim N\left(0, \sigma_e^2\right),$$

where y_{ij} = $\log_{10}$ SCC at test day i for cow j, α = intercept value, $\mathbf{X_{ij}}$ = a matrix of exposure variables for each test day within cow ij, $\boldsymbol{\beta_1}$ = a vector of coefficients for $\mathbf{X_{ij}}$, $\mathbf{X_j}$ = a matrix of exposure variables for cow j, $\boldsymbol{\beta_2}$ = a vector of coefficients for $\mathbf{X_j}$, v_j = random effect to reflect residual variation between cows (assumed to be a normal distribution with mean = 0 and variance σ^2_{v}), and e_{ij} = residual level 1 error (assumed to be a normal distribution with mean = 0 and variance σ^2_{e}).

Model fixed effects were investigated by backward elimination of terms from a saturated model. The procedure was then repeated for biologically plausible interactions between the remaining terms (Dohoo et al., 2009). Fixed effects and interactions were included in the model if $P \leq 0.05$. Each change to the model was evaluated by assessing the difference in log-likelihood values. Goodness of fit for the final model was assessed using conventional plots of standardized residuals and examining the influence and leverage of data points (Rasbash et al., 2008).

RESULTS

This data set contained 11,141 records of LS and SCC. Descriptive lameness results for the herds involved were reported previously (Archer et al., 2010). Table 1 shows

potential confounding factors affecting the relationship between $\log_{10}$ SCC and LS were farm of origin, parity, season, month of lactation, and milk yield at the current test day (geometric mean SCC decreased by 6,000 cells/mL per kilogram increase in TDY; $P \leq 0.05$). The LS had an influence on $\log_{10}$ SCC within 10 d, which varied by farm; this was incorporated as interaction terms (Table 1). Compared with the geometric mean SCC for cows with LS 1 on the same farm, cows with LS 2 on farm 3 produced milk with 28,000 (95% CI; 5,000 to 39,000) fewer cells/mL, and cows with LS 2 on farm 6 produced milk with 30,000 (95% CI; 7,000 to 42,000) fewer cells/mL ($P \leq 0.05$). For cows in season 1, parity 1, lactation mo 1, and with TDY of 20 kg on farms 3 and 6, respectively, this was equivalent to 13 and 11% reductions in SCC for cows with LS 2 compared with LS 1. Overall, a trend existed for cows with LS 2 on 6 of the farms, and LS 3 on 5 of the farms to produce milk with a lower $\log_{10}$ SCC within 10 d ($P > 0.05$; Table 1).

Across all 7 herds, those cows that would have LS 3 six months later produced milk with 16,000 (95% CI; 1,000 to 24,000) fewer cells/mL compared with the geometric mean for cows that would have LS 1 in 6 mo time ($P \leq 0.05$). For cows in season 1, parity 1, lactation mo 1, and with TDY of 20 kg, this was equivalent to a 10% decrease in SCC for cows that would have LS 3 in 6 mo time compared with those that would have LS 1 in 6 mo time. A trend was evident across all herds for cows that would have LS 3 five to eight months later to produce milk with a lower $\log_{10}$ SCC than cows with LS 1 at these times ($P > 0.05$; Table 1).

Diagnostics from the final model indicated a good fit to the data (Figure 1).

DISCUSSION

Contrary to previous work (Peeler et al., 1994; Hultgren et al., 2004), this study identified negative associations between LS and SCC, such that cows that suffer with lameness, on some farms produce milk with lower geometric mean SCC than unaffected cows; this occurred for cows with LS 2 on 2 of the 7 study farms within 10 d of LS assessment, and for cows on all farms with LS 3 six months later. This highlights differences in disease dynamics between farms, and illustrates the complexity of developing farm-specific estimates of disease costs, and health management plans in clinical practice.

The biological explanation of our findings is of interest. In terms of farm management practices, a decrease in geometric mean SCC associated with lameness could be explained by affected cows spending more time standing than unaffected cows (Cook et al., 2004) and thus, decreased exposure to pathogens at the teat end. The differences between farms in geometric mean SCC within 10 d of LS assessment could be explained by variation in the hygiene of cow lying areas. So, if nonlame cows spend more time lying down compared with lame cows, their risk of IMI is higher on farms where environmental hygiene is poor.

To demonstrate the potential conflict between lameness and mastitis control, keeping cows standing for at least 30 min after milking was one of the most cost-effective mastitis control measures for a typical Dutch dairy farm (Huijps et al., 2010). It is possible that such extra standing time may have deleterious effects on lameness, and this type of conflict may be behind the explanation of the findings in this study.

Although a causal relationship between increased standing time, reduced IMI risk, and increased risk of lameness has yet to be established, increased standing time in the transition period was associated with the development of claw horn lesions up to 15 wk later (Proudfoot et al., 2010). It is unknown to what extent standing times were actively managed to control mastitis on these farms. That keeping cows standing up after milking is a risk factor for lameness, as well as a cost-effective control measure for mastitis needs to be considered in the context of an individual farm when bespoke herd health advice is delivered.

Cows that become lame tend to be higher yielding (Green et al., 2002; Archer et al., 2010), and the time they spend standing to eat increases with milk yield (Gomez and Cook, 2010). This may increase foot trauma in lactation and the risk of future cases of lameness, as well as decrease SCC by the causal pathway suggested. Once lameness developed, the time cows spent standing to eat decreased (González et al., 2008; Gomez and Cook, 2010). How this may influence SCC requires further investigation.

Despite negative associations between LS and TDY at the cow level, at the herd level this may be hard to observe, as a result of the tendency for lame cows to be the higher-yielding individuals (Archer et al., 2010). When combined with the results from the current study, on some farms lame cows may have an apparent economic advantage if they produce a higher yield of lower-SCC milk than do unaffected cows. Despite welfare concerns, this may contribute to both the retention of persistently lame cows in herds, and the breeding of replacement heifers that are genetically predisposed to succumb to lameness during their productive lifetime, if genetic selection criteria are weighted toward selecting for increased milk yield and decreased SCC.

Unidentified confounding factors that explain the association between LS and SCC may remain (e.g., other disease or management events that have not been re-

Table 1. Final model of $\log_{10}$ cow test-day SCC (10^3 cells/mL response); impact of the main fixed and random effects[1]

Item (reference category)	Mean effect	SE	Lower 95% CI	Upper 95% CI
Intercept	2.205	0.044	2.119	2.291
Farm (farm 1)				
2	0.097	0.041	0.017	0.177
3	0.229	0.042	0.147	0.311
4	0.062	0.043	−0.022	0.146
5	0.137	0.036	0.066	0.208
6	0.202	0.043	0.118	0.286
7	0.149	0.042	0.067	0.231
Season (quarter 1[2])				
2	−0.067	0.012	−0.091	−0.043
3	−0.068	0.013	−0.093	−0.043
4	−0.044	0.013	−0.069	−0.019
Parity (parity 1)				
2	0.112	0.018	0.077	0.147
3	0.225	0.021	0.184	0.266
4	0.266	0.025	0.217	0.315
5+	0.420	0.025	0.371	0.469
Lactation month (lactation mo 1)				
2	−0.066	0.017	−0.099	−0.033
3	−0.053	0.017	−0.086	−0.020
4	−0.033	0.018	−0.068	0.002
5	−0.021	0.018	−0.056	0.014
6	0.010	0.018	−0.025	0.045
7	0.014	0.020	−0.025	0.053
8	0.020	0.020	−0.019	0.059
9	0.042	0.021	0.001	0.083
10	0.072	0.022	0.029	0.115
11	0.077	0.025	0.028	0.126
12+	0.076	0.024	0.029	0.123
Test-day milk yield (/kg)	−0.017	0.001	−0.019	−0.015
LS[3] (LS 1)				
2	0.031	0.026	−0.020	0.082
3	−0.020	0.380	−0.765	0.725
LS+5 mo[4] (LS+5 mo 1)				
2	0.006	0.016	−0.025	0.037
3	−0.009	0.018	−0.044	0.026
LS+6 mo[5] (LS+6 mo 1)				
2	0.018	0.019	−0.019	0.055
3	−0.046	0.022	−0.089	−0.003*
LS+7 mo[6] (LS+7 mo 1)				
2	0.024	0.023	−0.021	0.069
3	−0.007	0.027	−0.060	0.046
LS+8 mo[7] (LS+8 mo 1)				
2	−0.041	0.029	−0.098	0.016
3	−0.053	0.032	−0.116	0.010
Interactions				
Farm 2 LS (farm 2 LS 1)				
2	−0.028	0.036	−0.099	0.043
3	0.038	0.055	−0.070	0.146
Farm 3 LS (farm 3 LS 1)				
2	−0.082	0.036	−0.153	−0.011*
3	−0.024	0.050	−0.122	0.074
Farm 4 LS (farm 4 LS 1)				
2	−0.074	0.038	−0.148	0.000
3	−0.029	0.053	−0.133	0.075
Farm 5 LS (farm 5 LS 1)				
2	−0.029	0.032	−0.092	0.034
3	−0.032	0.043	−0.116	0.052

Continued

Table 1 (Continued). Final model of $\log_{10}$ cow test-day SCC (10^3 cells/mL response); impact of the main fixed and random effects[1]

Item (reference category)	Mean effect	SE	Lower 95% CI	Upper 95% CI
Farm 6 LS (farm 6 LS 1)				
2	−0.091	0.038	−0.165	−0.017*
3	−0.049	0.051	−0.149	0.051
Farm 7 LS (farm 7 LS 1)				
2	−0.035	0.037	−0.108	0.038
3	0.053	0.052	−0.049	0.155
Random effects	Variance		SE	
Level 2 (cow)	0.086		0.004	
Level 1 (test day)	0.131		0.002	

[1]Log-likelihood from model = −5,693; null model = −6,901.

[2]Quarter 1: August 2008 to October 2008; quarter 2: November 2008 to January 2009; quarter 3: February 2009 to April 2009; quarter 4: May 2009 to July 2009.

[3]Locomotion score; assessed within 10 d of a test day for SCC and milk yield.

[4]LS; assessed in 5 mo time.

[5]LS; assessed in 6 mo time.

[6]LS; assessed in 7 mo time.

[7]LS; assessed in 8 mo time.

*$P \leq 0.05$.

corded). Intercurrent disease may interact with LS and SCC; disease event records were incomplete for these farms and were not included in the analysis. Further work is required to elucidate the reasons for this negative relationship between LS and SCC on particular farms. Possible sources of bias within the study population were the generally high prevalence of lameness at the herd level [62% (range 48 to 72)] and geographical clustering, with all farms under the care of a single veterinary practice (Archer et al., 2010). These results

Figure 1. Plots of residuals for the model in Table 1, including the influence and leverage of data values to indicate that the model is a good fit to the data.

can only be extrapolated to other herds with caution. In the absence of IMI, cows with higher milk yields have lower SCC through dilution alone (Green et al., 2006). Increased TDY was associated with decreased SCC in the current study; this is accounted for in the model alongside other confounding variables, and the negative associations between LS and SCC remain.

For serial LS assessment to be more widely adopted for monitoring herd lameness prevalence, larger studies should be conducted to examine if associations exist between LS and lifetime production traits, including SCC. It will be important to fully evaluate the farm-specific economic effect of lameness in dairy herds, including any effect on other diseases, to develop rational health management plans and improve dairy cow welfare.

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