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Associations between sole ulcer, white line disease and digital dermatitis and the milk yield of 1824 dairy cows on 30 dairy cow farms in England and Wales from February 2003–November 2004

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Abstract

The milk yields of 1824 cows were used to investigate the effect of lesion-specific causes of lameness, based on farmer treatment and diagnosis of lame cows, on milk yield. A three-level hierarchical model of repeated test day yields within cows within herds was used to investigate the impact of lesion-specific causes of lameness (sole ulcer, white line disease, digital dermatitis and other causes) on milk yield before and after treatment compared with unaffected cows. Cattle which developed sole ulcer (SU) and white line disease (WLD) were higher yielding cattle before they were diagnosed. Their milk production fell to below that of the mean of unaffected cows before diagnosis and remained low after diagnosis. In cattle which developed digital dermatitis (DD) there was no significant difference in milk yield before treatment and a slightly raised milk yield immediately after treatment. The estimated milk loss attributable to SU and WLD was approximately 570 and 370 kg, respectively. These results highlight that specific types of lameness vary by herds and within herds they are associated with higher yielding cattle. Consequently lesion-specific lameness reduction programmes targeting the cow and farm specific causes of lameness might be more effective than generic recommendations. They also highlight the importance of milk loss when estimating the economic impact of SU and WLD on the farms profitability.

Keywords: Dairy cows; Lameness; Milk yield; Claw lesions; Multilevel models

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1. Introduction

A number of studies have now reported that higher yielding cows are more likely to become lame (Dohoo and Martin, 1984; Rowlands and Lucey, 1986; Barkema et al., 1994; Green et al., 2002). These lame cows might produce absolutely less milk than unaffected cows (Tranter and Morris, 1991; Warnick et al., 2001; Hernandez et al., 2002) or less milk than their potential as demonstrated by Green et al. (2002) and as predicted by Rowlands and Lucey (1986).

This is of huge economic importance with the current high prevalence of lameness in dairy cows, estimated to be 15% in the USA (Wells et al., 1993) and 22% in England (Whay et al., 2002). Milk loss per cow because of lameness has been estimated to be 440 and 270 kg for early lactation and mid to late-lactation, respectively, in France (Coulon et al., 1996); 1.5–2.8 kg/day two weeks from diagnosis in Finland (Rajala-Schultz et al., 1999); 1.5 kg/day two weeks from diagnosis in the USA (Warnick et al., 2001) and up to 2 kg/day for up to 5 months before and after diagnosis in the UK (Green et al., 2002).

Few studies have differentiated the lesion-specific cause of lameness when estimating milk loss. Hernandez et al. (2002) studied 531 cows in one herd in the USA, where cows that were lame with interdigital phlegmon produced significantly less milk over a lactation than unaffected cows (7767 kg vs. 8622 kg, respectively), with no significant milk loss attributable to other foot lesions. In a study of two farms, Warnick et al. (2001) reported that on one farm cows that were lame with a sole ulcer (SU) had the greatest loss of milk, followed by sole and white line abscesses and then interdigital phlegmon with no significant effect of foot warts (digital dermatitis). However, there were no lesion-specific associations with reduced milk yield on the second farm.

One reason for the paucity of lesion-specific studies of lameness and milk yield is that whilst lameness is common, e.g., up to 70 cases/100 cows/year (Green et al., 2002), lesion-specific causes of lameness are far less common: a rate of 10 cases/100 cows/year for any one cause of lameness would be high and highly variable between farms (Green et al., 2002). Consequently, a large sample of cows with several herds is required to elucidate the associations between lesion-specific lameness and milk yield. Ideally this would be a random sample of herds, however, the compliance required from farmers to access milk records and to record treatments for lesion-specific causes of lameness make this impossible currently, certainly in GB.

As part of an EU funded project (EU funded framework 5 project OLRT-2001-00969) all 4771 cows on 30 convenience selected farms were monitored for 18 months. Farmers were trained to identify foot lesions by one veterinarian (RB) and recorded all cases of treatment for lameness from February 2003–November 2004. Individual cow productivity data were recorded on a milk quality programme (National Milk Records (NMR)). The aim of this study was to investigate the associations between milk yield and lesion-specific causes of lameness.

2. Materials and methods

The original data included 4771 cows from 30 farms in England and Wales. The data set included 55,294 test day yields (TDYs) from February 2003 to November 2004. In the final analysis only complete lactations within the study period were used in the analysis because of the potential that a cow might have become lame before or after the data collection period within the same lactation (i.e., non-random missing data). As a result, 17,140 TDY from 1824 cow complete

lactations were used. Lesions causing clinical lameness were recorded by farmers who had received instruction from a veterinarian (RB) at a training meeting and who used a reference sheet with illustrations and descriptions of the lesions to assist with lesion recognition. On the recording form farmers named the lesion and marked the location on a diagram of a foot. If the form was incomplete or farmers named the lesion but marked the sheet incorrectly the bovine was excluded from the analysis. No training was provided to identify lame cows. The most prevalent lesions were sole ulcer, an erosion of the horn with granulomatous proud flesh in the sole of the foot, white line disease (WLD), a separation of the wall horn with or without infection and digital dermatitis (DD), inflammation of the interdigital skin. All other lesions were categorised as 'other', the most common 'other' lesion was interdigital phlegmon. Treatments were at the discretion of the farmer.

2.1. Data analysis

Test day yield was the outcome variable; it followed a normal distribution. The data were hierarchically structured with TDY within cow within farm. The TDY were repeated measures through time by month in milk. Data from the first 300 days of lactation and the first occurrence of each lesion were included in the analysis, so a cow could have had more than one lesion, e.g., DD in month 2 of lactation and a SU lesion in month 5. The data were analysed using S-Plus for Windows (version 6.2) using the correlated data instruction library (Chao, 2003) using a maximum likelihood procedure. The lactation curve was modelled using days in milk (DIM) and the exponential $\mathrm{DIM}^{-0.05}$ (Wilmink, 1987). Covariates that were included in the analysis were parity (categorically coded from 1 to 5+) and year quarter (January–March, April–June, July–September and October–December). Each lesion-specific diagnosis was linked to the month in milk for each cow: the month lame (month 0) was the month of diagnosis of the lesion and this was then lagged backwards to define 1, 2, 3, 4 and 5 months before the diagnosis of the lesion and 1, 2, 3, 4 and 5 months after diagnosis of the lesion; this coding differentiated lame cows from cattle that were never lame.

The model took the form:

$$\mathrm{TDY}_{ijk} = \beta 0 + \sum \beta \mathrm{n} X_{ijk} + \gamma \mathrm{n} X_{jk} + \mathrm{v0}_k + \mathrm{u0}_{jk} + e_i$$

with i = TDY, j = cows and k = farms. Where β0 is the intercept, βn = coefficients for X_{ijk}, X_{ijk} = variables varying between TDY, X_{jk} = variables varying between cows, γn = coefficients for X_{jk}, $\mathrm{v0}_k$ = residual error between farms, $\mathrm{u0}_j$ = residual error between cows and e_i = residual error between TDY.

The model fitted values were plotted. A figure of the centred estimated milk loss by time of treatment, adjusted for unaffected cattle in the same month, was used to illustrate estimates of milk loss before and after this time for four groups; cattle with SU, WLD, DD and other causes of lameness. The data were centred on the mean yield of a never lame bovine adjusted for the covariates above and it was assumed that the TDY estimate was the daily yield for that month (i.e., monthly yield was estimated as TDY × 30 days). An example for illustrating cumulative milk loss for each lesion was calculated from significant differences for each monthly yield compared with the initial TDY 5 months before diagnosis at 5 months in milk. The estimated yield of cattle 5 and 3 months before diagnosis, at the time of diagnosis and 5 months after diagnosis for each of the four lesions compared with unaffected cattle was plotted using MLwiN (Rasbash et al., 1999) from fitted data from the model.

3. Results

Herd size, parity, average yield and lesion rates for the 30 farms are presented in Table 1.

Out of the 1824 cows in the study, 636 (34.9%) were lame with at least one lesion. There were 230 diagnoses of SU (84% lateral and 16% medial), 169 of WLD (93% lateral and 7% medial), 137 of DD and 238 'other' lesions. Approximately 25% cows had at least one repeat of the same lesion in the lactation.

The rate of SU and WLD peaked at 5 and 4 months in milk, respectively, whilst the rate of DD and 'other' lesions decreased from calving (Fig. 1). The majority of the 'other' lesions that occurred in the first 2 months in milk were interdigital phlegmon. SU, WLD and 'other' lesions increased with increasing parity (data not shown); the incidence of DD was not associated with parity.

In the three-level hierarchical model, the stage of lactation, parity and calendar year – quarter were associated with TDY (Table 2). Cows that were diagnosed with SU produced approximately 1.5 kg more milk per day 5 months before diagnosis than unaffected cows, but by 2 months before diagnosis they were producing significantly less than their yield at 5 months before diagnosis (Table 3 and Figs. 2 and 3). The total estimated milk loss per lactation for a cow diagnosed with SU was 574 kg (95% CI 307–841 kg). Similarly, cows that were diagnosed with WLD produced approximately 0.8 kg more milk per day than unaffected cows 5 months before diagnosis but these cattle did not produce significantly less milk than this figure until the month that they were treated (Table 3 and Fig. 2). The total estimated milk loss per lactation for a cow diagnosed with a WLD was 369 kg, 95% CI 137–600 kg (Table 3). Cows with DD produced more milk months after treatment when compared with before treatment but not when compared with non-lame cows. Cows with 'other' lesions causing lameness also did not produce more or less milk than non-lame cows but the pattern of milk yield change was less clear for cows with 'other' lesions. Due to the lack of clarity and the nature of the 'other' lesions variable it would be inappropriate to derive specific milk losses. However, it would appear that cows with 'other' lesions also tended to produce more milk than unaffected cows before diagnosis.

When the fitted values of milk yield were plotted for each lesion an interesting pattern of milk loss is visualised (Fig. 3). This figure allows for the fact that cows may be lame at any month in milk. It can be seen that cattle with SU produced more milk than unaffected cattle 5 months before treatment. This fell 3 months before treatment, but these cattle still produced more milk than the mean lactation output for unaffected cattle. By the time of treatment, cattle with SU were producing a similar amount to unaffected cattle and 5 months after treatment they were

Table 1
Herd size, parity, average yield and lesion rate for the 1824 cattle on 30 dairy cow farms in England and Wales from February 2003–November 2004

Farm factor	Min	Max	Mean	S.E.
Herd size	41	395	113	12.60
Mean parity	2.0	5.0	3.0	0.10
Average milk yield (kg)	4983	8885	7073	185.30
Sole ulcer/100 cows/year	1.4	17.5	6.8	0.88
White line disease/100 cows/year	0.5	16.8	5.4	0.78
Digital dermatitis/100 cows/year	0.5	38.9	6.3	1.71
'other' causes of lameness/100 cows/year	0.7	18.4	5.6	0.86

Min = minimum value, max = maximum value and S.E. = standard error.

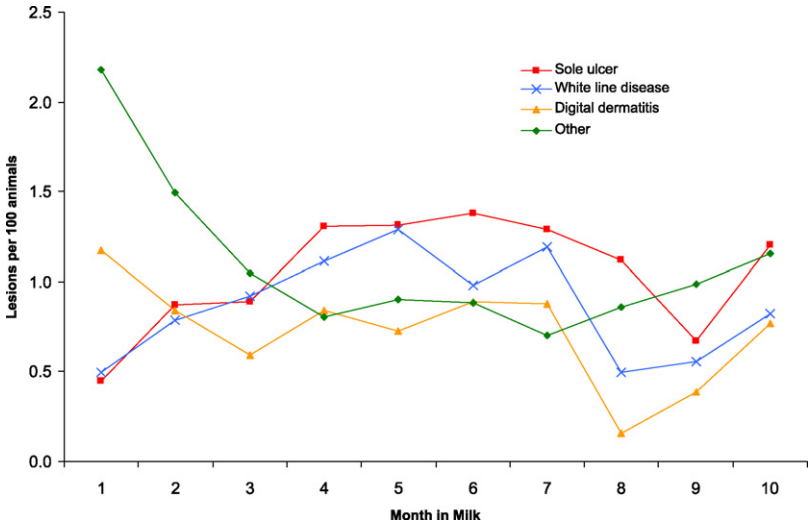

Fig. 1. Incidence of lesions causing lameness by month in milk for 1824 cattle from 30 farms in England from February 2003–November 2004.

producing significantly less milk (Fig. 3a). A similar pattern occurred in cattle with WLD (Fig. 3b). However, for DD (Fig. 3c) cattle produced less milk than unaffected cattle 5 months before diagnosis and slightly more milk than the mean production of unaffected cattle and for 'other' lesions (Fig. 3d) cattle produced a similar amount to unaffected cattle.

The residual plots indicated a good model fit to the data (Fig. 4).

Table 2
Estimated effects of confounders* from the three-level hierarchical model of the impact of hoof lesions on test day milk yield for 1824 cattle from 30 farms in England and Wales from February 2003–November 2004

Exposure	Mean effect	S.E.	Lower 95% CI	Upper 95% CI
Intercept	36.19	0.72	34.79	37.59
Lactation curve				
Days in milk (DIM)	−0.07	0.00	−0.07	−0.07
Exp $DIM^{-0.05}$	−12.91	0.30	−13.49	−12.33
Parity number				
Parity 1 (reference)				
Parity 2	4.66	0.29	4.09	5.23
Parity 3	5.38	0.33	4.74	6.02
Parity 4	6.07	0.35	5.38	6.76
Parity 5–14	4.47	0.32	3.85	5.10
Month of milk recording				
January–March (reference)				
April–June	1.06	0.09	0.88	1.24
July–September	−0.90	0.10	−1.10	−0.71
October–December	−1.45	0.09	−1.62	−1.28

S.E. = standard error and 95% CI = 95% confidence interval.
* These coefficients for confounders are those calculated from the overall model including lesion diagnosis.

Table 3
Estimated effects[a], from a three-level hierarchical model, of months before and after a treatment on test day yield (kg) in 1824 cattle from 30 farms in England from February 2003–November 2004

Month	Mean effect	S.E.	Mean daily milk loss (kg)	Lower 95% CI	Upper 95% CI	Estimated monthly milk loss (kg)[b]	Lower 95% CI	Upper 95% CI
Sole ulcer								
Before diagnosis								
5+	1.46	0.50	0.00			NS		
4	0.71	0.59	−0.75	−1.90	0.39	NS		
3	0.63	0.56	−0.83	−1.92	0.26	NS		
2	−0.01	0.54	−1.47	−2.53	−0.41	−44.07	−75.79	−12.36
1	−0.31	0.54	−1.77	−2.82	−0.72	−53.19	−84.71	−21.66
After diagnosis								
0	−0.93	0.53	−2.39	−3.43	−1.35	−71.75	−102.98	−40.52
1	−1.10	0.54	−2.56	−3.62	−1.49	−76.71	−108.59	−44.83
2	−1.27	0.57	−2.73	−3.85	−1.62	−81.99	−115.52	−48.47
3	−0.97	0.61	−2.42	−3.61	−1.24	−72.73	−108.41	−37.06
4	−1.12	0.65	−2.58	−3.85	−1.31	−77.29	−115.37	−39.22
5+	−1.75	0.57	−3.20	−4.32	−2.08	−96.13	−129.74	−62.51
Total						−573.86	−841.10	−306.62
White line disease								
Before diagnosis								
5	0.83	0.55	0.00	−1.08	1.08	NS		
4	0.75	0.66	−0.09	−1.37	1.20	NS		
3	0.88	0.61	0.05	−1.15	1.24	NS		
2	0.91	0.61	0.08	−1.11	1.27	NS		
1	−0.20	0.61	−1.03	−2.23	0.17	NS		
After diagnosis								
0	−0.64	0.61	−1.47	−2.67	−0.28	−44.21	−79.99	−8.42
1	−0.70	0.62	−1.53	−2.75	−0.31	−45.90	−82.41	−9.39
2	−0.90	0.65	−1.73	−3.00	−0.46	−51.86	−90.04	−13.68
3	−1.21	0.68	−2.04	−3.37	−0.70	−61.11	−101.15	−21.07
4	−1.69	0.72	−2.52	−3.94	−1.10	−75.62	−118.22	−33.03
5	−2.18	0.65	−3.01	−4.29	−1.73	−90.24	−128.59	−51.90
Total						−368.95	−600.41	−137.49

95% CI = 95% confidence intervals and NS = not significantly different from initial milk yield.
[a] These coefficients are adjusted for the confounders presented in Table 2.
[b] Assuming 30 days per month.

4. Discussion

We can consider milk production before diagnosis as a predictor for lameness and reduction in milk production before diagnosis as a pre-diagnosis indication of lesion development or presence and milk production after diagnosis as a consequence of lameness and/or treatment.

In this analysis, the high yielding cattle were more likely to develop non-infectious causes of lameness, SU, WLD (and possibly other types of lameness) but not apparently more likely to develop DD. High yielding cattle might be in the same physical environment as the average and low yielding cattle but might cope less well with this same environment. This could be because they have to behave differently, e.g., spend more time feeding, being milked and/or because they

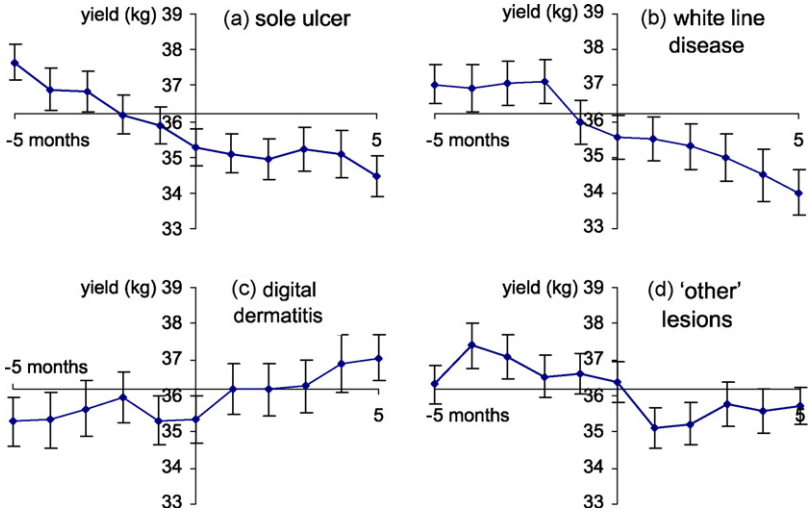

Fig. 2. Impact of adjusted lesion-specific lameness on milk yield from 1824 cattle from 30 farms in England from February 2003–November 2004. The mean unaffected milk yield runs along the *X*-axis and the origin is the month of lesion-specific treatment. The *Y*-axis is estimated daily yield (kg). S.E.M. bars shown.

are genetically more susceptible within this same environment. High yielding dairy cows are at a greater risk of other metabolic disorders (Gröhn et al., 1999) and this lack of physical robustness might also be a risk for horn-associated lameness, since metabolic disruption reduces hoof horn quality and pre-disposes to lameness (Mulling et al., 1999). A lameness reduction programme will be of more benefit if it is targeted to the high frequency lesions associated with lameness on a farm, since the recommended alterations in management are different between cause(s) of lameness. For SUs and WLD, the target areas will be those that might affect the management, feeding and genetics of the high yielding cattle within a herd since these cattle appear to be at greater risk of being treated for SU or WLD later in lactation when compared with less high yielding cattle within the herd (although of course, the management of all cattle might have to be altered to address the targeted changes).

The findings from this paper suggest that DD and 'other' lesions are less likely to be associated with metabolic dysfunction. These lesions were not associated with high initial yield or subsequent milk loss (in agreement with Warnick et al., 2001). If anything, treatment may improve milk yield in these cattle. The results also suggest that cattle are at greatest risk of DD early in lactation and that this does not change by parity (data not shown). These results indicate that exposure to the infectious organism might change as cattle join the milking herd, or it is possible that dry cows are not treated until they join the milking herd. Consequently, whole herd changes in management targeted at lowering the incidence of these lesions should assist in reduction of these infectious diseases.

The significant drop in milk yield occurred from approximately 3 months before treatment in cases of SU and 1 month before diagnosis for WLD might indicate that pathogenesis of disease starts well before a bovine is considered lame. Previous work has demonstrated that cows that are lame with SU or WLD have a lowered pain threshold for up to 28 days after treatment, whilst those with acute digital tissue infection were not significant from unaffected cows following treatment (Whay et al., 1998). It might be that the pain threshold is lower before as well as after treatment. Milk production remained lower after treatment in cattle with SU or WLD. This might

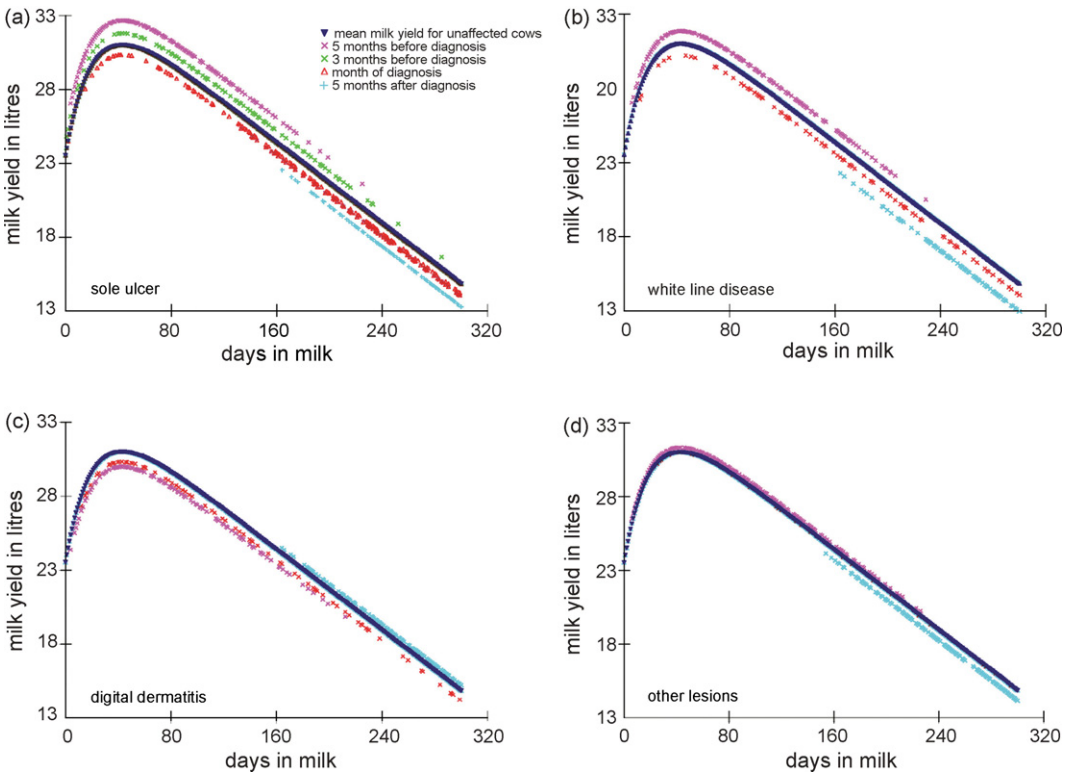

Fig. 3. Fitted values from the full model of 1824 cattle split by lesion type. For each graph *X*-axis is 'days in milk' and *Y*-axis is 'milk yield in litres'. Blue line = mean milk yield for unaffected cows. Pink = 5 months before diagnosis, green = 3 months before diagnosis, red = month of diagnosis and cyan = 5 months after diagnosis.

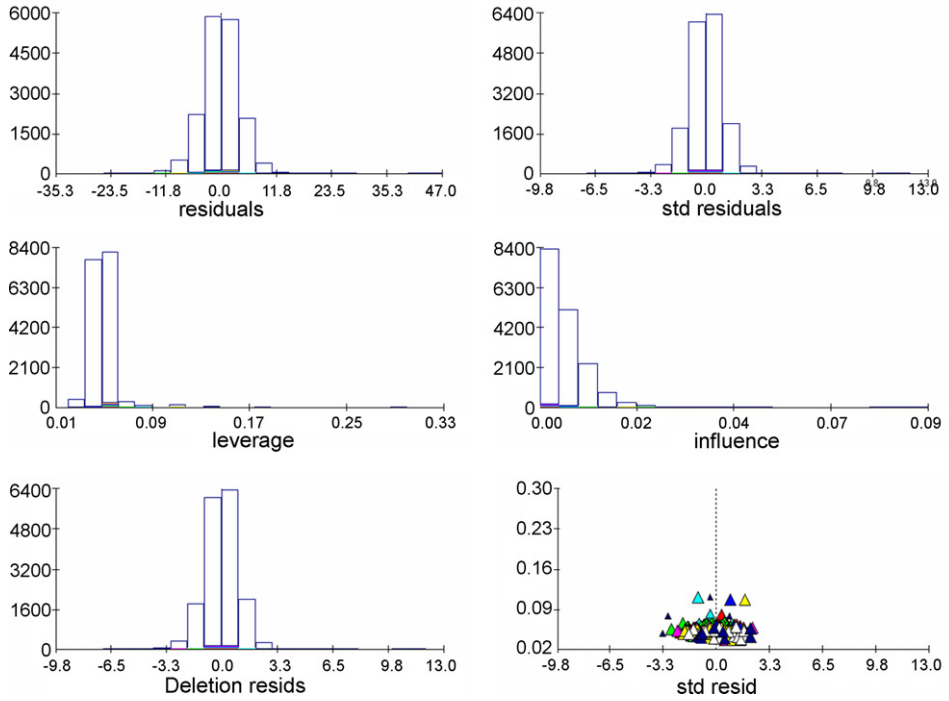

Fig. 4. Residual plots for the fitted model of lesion-specific lameness on milk yield, indicating a good fit to normality.

be associated with behavioural changes such as reduced feeding and drinking, due to increased pain or due to physiological changes, such as increased cortisol concentration and raised metabolic rate (El-Ghoul and Hofmann, 2002). If milk yield is linked to pain then this might help explain Fig. 3. One would anticipate that lack of treatment would have led to a continued fall in yield in line with the time before diagnosis and that treatment at least stabilised the reduced yields of these cows. It would be pertinent to note that in including only cows with complete lactation data in the analysis, there is likely to be an underestimate of milk losses due to the exclusion of cows culled for lameness mid-lactation.

The change in milk production both before and after diagnosis indicates that lesion-specific lameness had varying effects on milk production. Cattle with SU had approximately twice the milk loss of those with WLD. The estimated milk loss for any cause of lameness by Green et al. (2002), Warnick et al. (1995) and Coulon et al. (1996) was approximately the average milk loss attributable to the combined causes of SU and WLD in this study, despite all farms in all studies being non-random. We cannot speculate as to whether these results can be extrapolated to all farms but the evidence from several papers of studies of non-random farms suggest that milk yield and lameness are linked; it may be that the quantitative estimates are not externally valid. As with other studies, the confidence intervals were wide in our study, partly because cases of specific lesions were relatively rare and because lesions occurred throughout lactation and so not all cows contributed to all months, as discussed in Green et al. (2002). However, variability remained even with lesion-specific causes of lameness removing the low effects from DD and other causes of lameness. This might be because there were differences in management of lame cows, e.g., lead time to treatment from observing lameness and lesion recording, e.g., misclassification of lesions (although we aimed to minimise this with training and a requirement

for a name and location for the lesion), between farmers and probably also because many other management and disease factors impact on TDY.

In this study clinical lameness was observed and managed by the farmer. It is likely, given previous research that indicated that farmers did not detect all cases of lameness (Whay et al., 2002), that some cows were not treated and so no diagnosis was made. There were certainly some lame cows on the farm at routine visits assessing locomotion (Barker et al., 2007). The result of this would be to reduce the difference and increase variability in estimated differences in milk production between the reported lame and unaffected cattle.

5. Conclusions

High yielding dairy cows were more likely to become clinically lame with SU or WLD than unaffected cows, whilst cattle that were treated with DD or 'other' causes of lameness were not higher yielding than unaffected cows. Farmer diagnosis of SU and WLD were associated with significant milk loss. For example, cows diagnosed with either SU and WLD at 5 months in milk were associated with a mean decreased yield per lactation of 574 kg (95% CI 307–841 kg) and 369 kg (95% CI 137–600 kg), respectively. Digital dermatitis or the presence of any 'other' lesion was not associated with economically significant reduction in milk production. This research emphasises the importance of recording the lesion-specific causes of lameness to determine both the possible economic consequences for a herd and to inform on management decisions to reduce lameness.

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