The Old School, Brewhouse Hill, Wheathampstead,
Hertfordshire AL4 8AN, UK

Animal Welfare 2010, 19: 419-427
ISSN 0962-7286

Associations between lesion-specific lameness and the milk yield of 1,635 dairy cows from seven herds in the Xth region of Chile and implications for management of lame dairy cows worldwide

LE Green*[†], J Borkert[‡], G Monti[§] and N Tadich[#]

[†] Department of Biological Sciences, University of Warwick, Coventry CV4, 7AL, UK
[‡] Master of Science Programme, Faculty of Veterinary Sciences, University Austral de Chile, Chile
[§] Department of Veterinary Preventive Medicine, Faculty of Veterinary Sciences, Universidad Austral de Chile, Chile
[#] Department of Veterinary Clinical Sciences, Faculty of Veterinary Sciences, Universidad Austral de Chile, Chile
* Contact for correspondence and requests for reprints: laura.green@warwick.ac.uk

Abstract

Lameness is one of the greatest infringements of welfare in dairy cows. The objective of this study was to investigate associations between milk yield and foot lesions causing lameness in Chilean dairy cattle with the hypothesis that if we can demonstrate that lameness reduces yield, and so income, from lame dairy cows then we have both economic and welfare arguments for reducing lameness in dairy cattle. For one year, all lame cows from seven farms with Holstein Friesian cattle were treated by their herdsmen. Herdsmen were trained by the researcher and a colour atlas was utilised to assist in diagnosis of lesions. All abnormalities on the foot and the suspected cause of lameness were recorded, and cattle were treated. A two-level hierarchical model with repeated monthly test-day yields within cows was used to investigate the impact of double sole (DS), sole ulcer (SU), white line disease (WLD), digital dermatitis (DD) and all 'other' causes of lameness on milk yield before and after treatment. There were 1,635 cows with complete data. Cattle with a DS were higher yielding than cattle that were never lame with a reduction in yield from four months before treatment. Cattle lame with DD were higher yielding than non-lame cattle before and after treatment. For all causes of lameness, yield increased the month after treatment. We conclude that lesions causing lameness reduced the milk yield of dairy cows in these seven herds in Chile. We discuss the current evidence base for prevention of lameness in dairy cows and hypothesise that rapid treatment is a feasible current approach to improve cow welfare immediately and probably reduce milk lost; more evidence for effective prevention is required.

Keywords: *animal welfare, claw lesion, dairy cow, lameness, milk loss, mixed effects model*

Introduction

The prevalence of lameness in dairy cows in developed countries is high, for example in the USA and UK the prevalence of lameness is estimated to be 15% (Wells *et al* 1993) and 22% (Whay *et al* 2003), respectively. This undoubtedly affects the welfare of dairy cattle because lameness is associated with pain (Whay *et al* 1997; Dyer *et al* 2007). There is also an economic cost to lameness. A number of studies have reported that lame dairy cows have a reduced milk yield compared with their potential (Rowlands & Lucey 1986; Green *et al* 2002) with milk loss per cow estimated to range from 270 to 440 kg per lactation (Coulon *et al* 1996), 1.5–2.8 kg per day for the two weeks after diagnosis (Rajala-Schultz *et al* 1999; Warnick *et al* 2001) and up to 2 kg per day for up to five months before and after diagnosis in the UK (Green *et al* 2002). Foot-lesion-specific reductions in yield have been reported for sole ulcer (Warnick *et al* 2001; Amory *et al* 2008), interdigital phlegmon (Warnick *et al* 2001; Hernandez *et al* 2002) and white line and sole abscesses (Warnick *et al* 2001; Amory *et al* 2008). In the largest study to-date in 1,824 cattle from 30 farms in the UK, Amory *et al* (2008), reported that reduction in yield was associated with the non-infectious claw lesions, sole ulcer and white line disease, where the reduction in yield was approximately 570 and 370 kg, respectively over a 305-day lactation. These estimates are similar to those for all causes of lameness reported by Green *et al* (2002) of 360 kg and Bicalho *et al* (2007) of 314–424 kg. Several authors have reported that it is high yielding cows that are more likely to become lame with sole lesions and white line disease (Green *et al* 2002; Amory *et al* 2008; Bicalho *et al* 2008).

In the current study, we present the associations between lesion-specific causes of lameness and milk yield in cattle from seven Chilean dairy herds with 337–506 cattle per

https://doi.org/10.1017/S0962728600001901 Published online by Cambridge University Press

Table 1 Herd size, parity, average yield, lesion rate and general management for 1,635 cattle from seven herds in the Xth region Chile.

Factor	Farm number						
	1	2	3	4	5	6	7
Herd size	381	396	337	506	440	484	423
Mean parity	2.12	3.48	2.44	2.61	2.83	3.07	4.24
Annual milk yield (kg) per cow	9,682	10,271	7,911	9,816	11,203	9,885	8,024
SU per 100 cows per year	7.19	3.19	9.67	3.37	5.12	2.14	1.01
WLD per 100 cows per year	3.09	0.02	7.14	1.37	3.84	0.38	2.45
DD per 100 cows per year	0.18	0.58	2.32	3.39	0.51	2.29	0
Housing	Never	Winter	Never	Winter	All year	Winter	Winter
Bed material	n/a	Soil	n/a	Mattress	Rubber	Soil	Straw
Diet							
Grass when grazing	Yes	Yes	Yes	Yes	No	Yes	Yes
Concentrate	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Silage	No	Yes	Yes	Yes	Yes	Yes	Yes

SU: Sole ulcer; WLD: White line disease; DD: digital dermatitis.

herd that were inspected and treated by the farmer. We then discuss the implications of these results together with those above on management of lame dairy cows given the current evidence for preventing and treating lameness.

Materials and methods

Holstein Friesian cattle in seven dairy herds located near Valdivia, Chile (latitude 39° 48' longitude 73° 14') were enrolled into a prospective study of lameness and milk yield from April 2005–May 2006. The herds were autumn-to-spring calving, commercial, dairy herds with 337–506 cows per herd with annual yields of 8,000–11,000 kg per cow; other general management factors are listed in Table 1. All data were collected by one observer (JB) who visited the farms twice each month. JB trained the farmers to use a photographic atlas (www.bienestaranimal.cl) translated from that developed in the EU Lamecow project (http://template.bio.warwick.ac.uk/E+E/lamecow/public_html/colour_atlas.pdf) to diagnose lesions. Farmers were asked to record all lesions observed with an indication of primary cause of lameness. When cows were lame they were examined and treated and the cause of lameness recorded by the farmer. At the next visit to the farm, JB checked the lesions and the suspected cause of lameness however no specific estimate of reliability was made on these farmers. At the end of the data collection period, the date that cows were lame and the cause of lameness were linked to the monthly test-day yield data together with cow parity. All cattle (n = 1,635) that were recorded for at least six months in milk with parity, calving date and test-day yield data were included in the analysis. Data after 305 days in milk were excluded.

Data analysis

Monthly test-day yield (TDY) was the continuous outcome variable. The data were hierarchically structured with TDY within cow, similar to the approach by Green *et al* (2002) and Amory *et al* (2008). The TDY were repeated measures through time and were sorted by month in milk. Only the first occurrence of the lesion considered to be causing lameness was used in the analysis but a cow could have any number of different lesions over time, eg a cow could have SU in month 1 and WLD (but not SU) in month 5. The data were analysed in MLwiN 2.0 (Rasbash *et al* 1999). The lactation curve was modelled using days in milk (DIM) and the exponential $DIM^{-0.05}$ (Wilmink 1987). Covariates that were included in the analysis were parity (categorically coded from 1 to 7+) and farm. Complex variation, that is changes in variability in the slope of the milk yield by month in milk, was introduced into the model to estimate the impact of these changes (Bicalho *et al* 2008) on milk loss due to lameness.

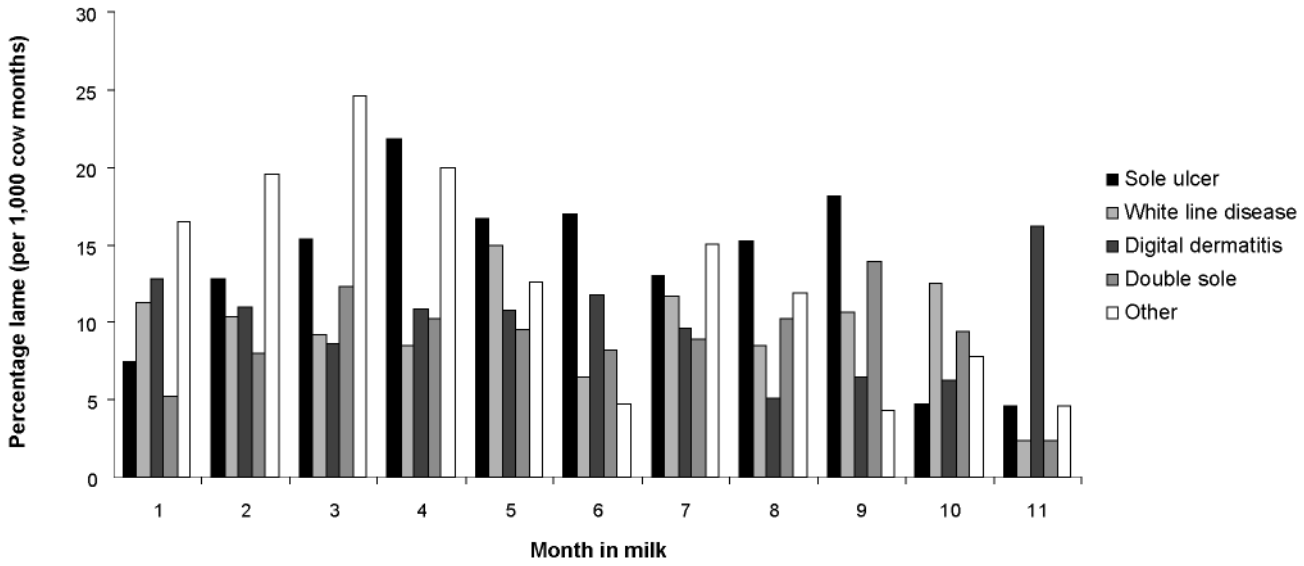
The model took the form: $TDY_{ij} = \beta 0 + \sum \beta n X_{ij} + \gamma n X_j + u0_j + e_i$

With i = TDY and j = cows. Where $\beta 0$ is the intercept, βn = coefficients for X_{ij}, X_{ij} = variables varying between TDY, X_j = variables varying between cows, γn = coefficients for X_j, $u0_j$ = residual error between cows e_i residual error between TDY.

The model-fitted values were plotted with the change in yield of lame cattle centred on the time of treatment adjusted for stage of lactation and parity to illustrate the estimated milk production before and after treatment.

https://doi.org/10.1017/S0962728600001901 Published online by Cambridge University Press

Figure 1



Percentages of treatments by lesion type per 1,000 cow months by month in milk.

Results

There were 13,231 test-day yields with useable data for the 1,635 cows in the study. There were 582 (34.2%) cows lame with at least one lesion and 828 treatments in total by day 305 of lactation. There were 132 cases of DS, 208 of SU, 143 of WLD, 142 of DD, and 203 'other' lesions (Table 1). The causes of lameness per 1,000 cow months are presented in Figure 1. For most causes of lameness there was a peak in time of first treatment between months two and four of lactation.

In the two-level hierarchical model farm, stage of lactation and parity influenced TDY (Table 2). There was significant complex variation between the month in milk and lesions; however the estimates of milk yield changed by less than 10 g per mean estimate and complex variation led to an inclusion of an extra 50 terms in the model so complex variation is not presented in the final model.

The 95% confidence intervals were wide for all estimates of milk loss by month in milk (Table 3, Figure 2), resulting in some intervals including zero. Lame cows with DS produced significantly more milk than non-lame cows before they were lame and had a reduction in yield from four months before they were treated to one month after treatment. Cows with SU did not produce significantly more milk before they were treated but milk yield dropped each month from calving and these lame cows produced significantly less milk from three months before they were lame until approximately one month after treatment than non-lame cows. Lame cattle with white line disease did not have a significant change in milk yield until four months after treatment when they produced more milk than non-lame cows. Cows with digital dermatitis had a significantly higher milk yield than non-lame cattle for the whole lactation with a dip in yield one month before treatment. For all causes of lameness, yield increased in the month after treatment and residual plots (not included) indicated a good model fit to the data.

Table 2 Two-level hierarchical model of the impact of stage of lactation, farm and parity on milk yield on TDY for 1,635 cattle from seven farms.

Factor	Mean (± SEM) change in daily yield (kg)
Intercept	114.010 (± 2.710)
Days in milk	–0.077 (± 0.001)
Exp (days in milk$^{-0.05}$)	–33.707 (± 1.149)
Farm, baseline farm 1	
2	–5.512 (± 0.542)
3	–6.113 (± 0.513)
4	–2.401 (± 0.522)
5	–8.037 (± 0.751)
6	–3.811 (± 0.504)
7	–9.296 (± 0.615)
Parity, baseline parity 1	
2	5.309 (± 0.378)
3	6.464 (± 0.379)
4	7.148 (± 0.472)
5	6.465 (± 0.661)
6	6.752 (± 0.805)
7	4.713 (± 0.723)

https://doi.org/10.1017/S0962728600001901 Published online by Cambridge University Press

Table 3 Mean daily reduction in milk yield (kg) in cows with double sole, sole ulcer, white line disease, and digital dermatitis in the months before and after diagnosis after adjusting for fixed effects from Table 2.

Lesion by time	Mean (± SEM) difference in milk yield	Lower 95% confidence interval	Upper 95% confidence interval	Lower and upper 95% range for significant change in 30-day yield
Sole ulcer (baseline no sole ulcer)				
5+ before	0.60 (± 0.59)	–0.59	1.79	0–0
4	–0.50 (± 0.65)	–1.81	0.81	0–0
3	–1.27 (± 0.59)	–2.44	–0.10	–2.88––73.08
2	–1.01 (± 0.56)	–2.12	0.11	0–0
1	–0.98 (± 0.54)	–2.05	0.10	0–0
Month of treatment	–2.05 (± 0.53)	–3.11	–0.99	–29.82––93.42
1	–1.56 (± 0.55)	–2.65	–0.46	–13.86––79.5
2	–0.73 (± 0.58)	–1.88	0.42	0–0
3	–0.20 (± 0.61)	–1.41	1.02	0–0
4	0.24 (± 0.64)	–1.05	1.52	0–0
5+ after	1.89 (± 0.57)	0.75	3.04	0–0
Double sole, baseline no double sole				
5+	0.65 (± 0.69)	–0.74	2.03	0–0
4	1.65 (± 0.80)	0.05	3.25	0–0
3	0.14 (± 0.74)	–1.34	1.62	0–0
2	–0.39 (± 0.69)	–1.77	1.00	0–0
1	–0.95 (± 0.67)	–2.29	0.39	0–0
Month of treatment	–1.76 (± 0.66)	–3.08	–0.44	–13.26––92.34
1	–1.76 (± 0.70)	–3.16	–0.36	–10.89––94.89
2	–0.75 (± 0.73)	–2.21	0.70	0–0
3	–0.35 (± 0.69)	–1.73	1.04	0–0
4	0.16 (± 0.83)	–1.50	1.83	0–0
5+	–0.76 (± 0.72)	–2.21	0.68	0–0
White line disease, baseline, no white line disease				
5+ before	–0.56 (± 0.71)	–1.98	0.86	0–0
4	–0.86 (± 0.78)	–2.41	0.70	0–0
3	–0.85 (± 0.75)	–2.35	0.66	0–0
2	–0.46 (± 0.70)	–1.85	0.94	0–0
1	–0.64 (± 0.67)	–1.97	0.69	0–0
Month of treatment	–0.65 (± 0.65)	–1.94	0.65	0–0
1	–0.06 (± 0.67)	–1.40	1.29	0–0
2	0.33 (± 0.70)	–1.07	1.73	0–0
3	0.83 (± 0.73)	–0.63	2.29	0–0
4	1.01 (± 0.77)	–0.53	2.54	0–0
5+ after	2.09 (± 0.69)	0.70	3.48	104.25–21.09
Digital dermatitis, baseline, no digital dermatitis				
5+ before	0.30 (± 0.76)	–1.22	1.82	0–0
4	2.82 (± 0.81)	1.20	4.44	133.17–36.09
3	2.93 (± 0.74)	1.46	4.40	131.97–43.65
2	2.58 (± 0.70)	1.18	3.98	119.46–35.46
1	2.20 (± 0.67)	0.86	3.54	106.08–25.8
Month of treatment	1.06 (± 0.64)	–0.23	2.34	0–0
1	1.39 (± 0.65)	0.09	2.69	80.58–2.7
2	1.42 (± 0.68)	0.06	2.79	83.61–1.77
3	1.31 (± 0.72)	–0.13	2.75	0–0
4	0.86 (± 0.76)	–0.67	2.39	0–0
5+	2.03 (± 0.65)	0.72	3.33	99.93–21.69

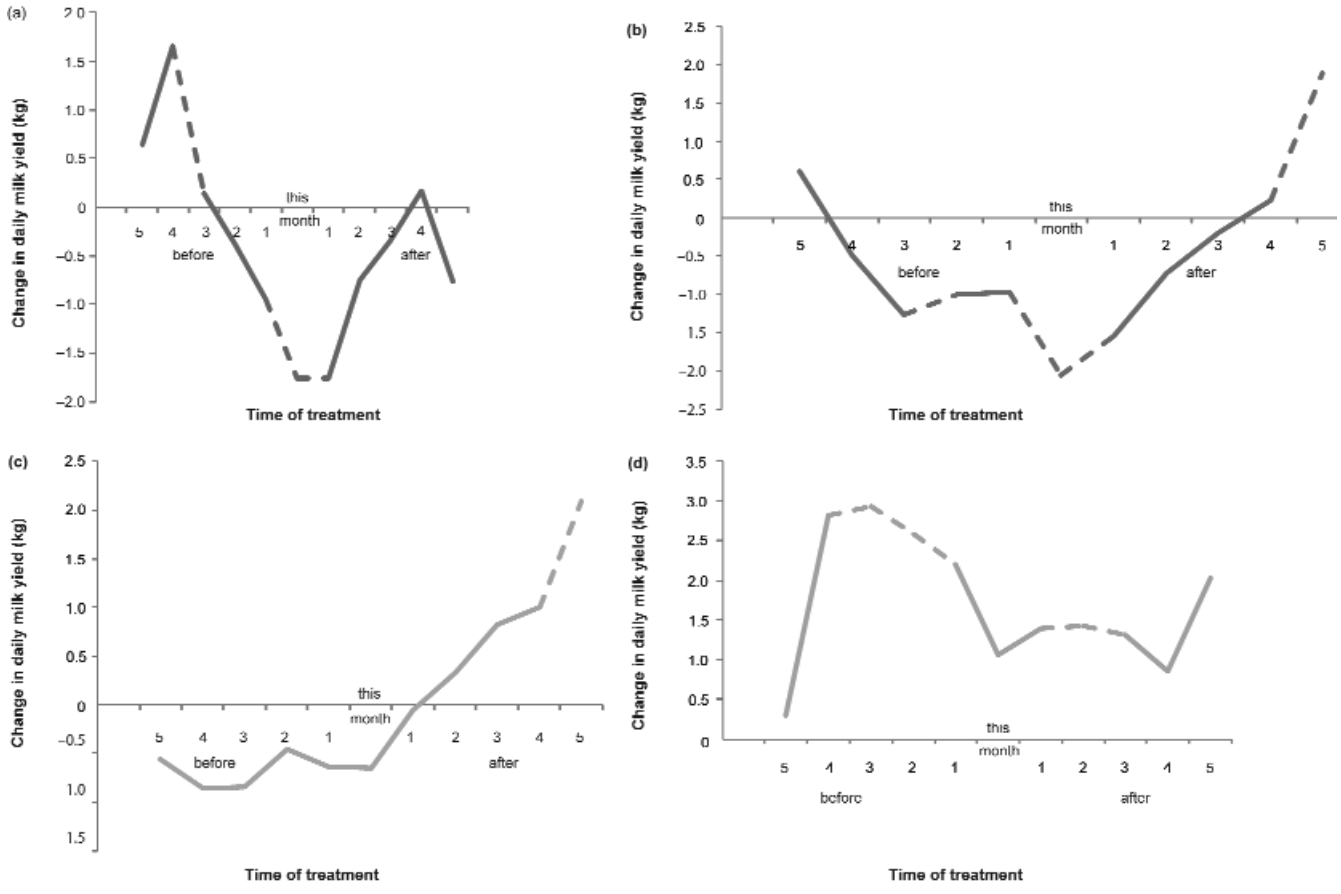
Assuming 30 days per month.
LCI, UCI = lower and upper 95% confidence intervals, respectively.

https://doi.org/10.1017/S0962728600001901 Published online by Cambridge University Press

Table 3 (cont) Mean daily reduction in milk yield (kg) in cows with double sole, sole ulcer, white line disease, and digital dermatitis in the months before and after diagnosis after adjusting for fixed effects from Table 2.

Lesion by time	Mean (± SEM) difference in milk yield	Lower 95% confidence interval	Upper 95% confidence interval	Lower and upper 95% range for significant change in 30-day yield
Other cause, baseline no other cause				
5+ before	–2.38 (± 0.68)	–3.73	–1.02	–30.51 (–111.99)
4	–0.88 (± 0.73)	–2.33	0.58	0 (0)
3	–0.91 (± 0.66)	–2.23	0.41	0 (0)
2	–0.66 (± 0.61)	–1.88	0.57	0 (0)
1	–0.85 (± 0.57)	–1.99	0.30	0 (0)
Month of treatment	–1.15 (± 0.56)	–2.26	–0.03	–1.05 (–67.77)
1	–0.47 (± 0.57)	–1.60	0.66	0 (0)
2	0.22 (± 0.58)	–0.94	1.38	0 (0)
3	0.85 (± 0.60)	–0.35	2.05	0 (0)
4	1.11 (± 0.63)	–0.16	2.38	0 (0)
5+ after	1.42 (± 0.55)	0.32	2.53	0 (0)

Figure 2



Change in daily milk yield (kg) for cows by (a) double sole, (b) sole ulcer, (c) white line disease and (d) digital dermatitis compared with unaffected cows after adjustment for covariates in Table 2 for 1,635 cattle from seven farms in southern Chile. Time of treatment is zero on the x axis, dashed lines indicate significant difference from zero.

https://doi.org/10.1017/S0962728600001901 Published online by Cambridge University Press

Discussion

These are the first estimates of the impact of lameness on milk yield from commercial dairy herds in Chile and they are similar to those reported in commercial dairy cattle in other countries. As with other similar studies, the data are limited by the fact that herdsmen recorded the causes of lameness. We limited the variability between herdsmen by using one researcher (JB) to check the diagnosed cause of lameness. However, we know that some lesions are common and were not linked to increasing severity of lameness in a larger study of dairy cows in Chile (Tadich *et al* 2010) and that there is a risk of misdiagnosis in the current study. If this was random error then the strength of association between a lesion and milk yield would be lower, leading to reduction in significance. If it was bias then the association could be strengthened or weakened.

The impact of each lesion on yield was not completely consistent with that reported by Amory *et al* (2008) or Warnick *et al* (2001). For example, in the Chilean herds there was a significant association between DD and high yields. Also, cows with SU did not have significantly higher yields than non-lame cows in the months before they were lame as reported by Amory *et al* (2008), but this is possibly because these cows were already affected with SU at calving, given the rapid fall in yield from calving (Figure 2) and the peak treatment for sole ulcer at three months in lactation (Figure 1). Cows with DS did have a significantly higher yield before they were treated (Figure 2); many of these cows had an underlying SU (Tadich, personal communication 2008), unfortunately we do not have this underlying cause recorded. A double sole forms when there is disruption of the germinal epidermal cell layer and horn separates from the sole. A new layer of horn then forms underneath. This suggests that the underlying cause of lameness in cows with DS was unknown and that they were lame for some time. The lack of association between high yield before diagnosis and WLD (reported in Amory *et al* 2008) might have occurred because WLD was sometimes misdiagnosed as the cause of lameness (Tadich *et al* 2010): we cannot be sure that farmers recorded the lesions correctly, even after training.

As with other studies, there was wide variation in the estimates of reduction in yield in lame cows. Each cow contributed at least six months milk yield to the analysis, some cows will have contributed the last part of one lactation and the first part of a second lactation. This was accounted for in the analysis by including parity and days in milk separately.

One other explanation for the variation in yield is that there was variation in the time from when a cow became lame to her treatment and that this has a varying effect on change in milk yield. One would anticipate that a short time from lameness to treatment would lead to less reduction in yield. We know that lameness causes pain in dairy cows (Whay *et al* 1997) and that pain raises metabolic rate and so lame cows will lose condition and produce less milk. In this study, as in others to-date on treatment for lameness and milk loss, we do not know when the cows became lame, only when they were treated. If duration of lameness is causing reduced milk production then the situation in dairy cows parallels that in sheep where we do know that duration of lameness affects body condition and so lamb production and growth. In a clinical trial on one farm of 700 ewes, those lame with footrot (including interdigital dermatitis) for less than six days (ie from first observed lame to recovered after treatment) had a better body condition and finished their lambs faster than sheep lame for longer than six days (Wassink *et al* 2010). Lamb growth rate is a marker for milk production in sheep so it is biologically plausible that lame cows treated promptly will have less time in pain and less time for yield to fall before treatment and, with less pathology in their feet, will recover more rapidly. Whilst the ideal is that all lame cattle are treated immediately they are seen lame, the reality is far from this; in a study of 49 dairy farms in England, 10% of cows were lame on each of four occasions over one year (Barker 2007). This is discussed further below. A study investigating locomotion score, treatment and milk yield would address this because we could establish the point at which a cow started to walk abnormally.

Context of management of lameness

The results from these herds in Chile add to the evidence that in intensive systems, higher yielding cows are more likely to become lame, and are therefore in need of greater care within a herd. We have a worldwide situation where cows have a reduced milk yield before and after treatment (Warnick *et al* 2001; Hernandez *et al* 2002; Amory *et al* 2008) and this has implications for the welfare of dairy cows and the economics of farming. The results from the current study add to the mounting evidence for the economic impact of lameness on milk production (Green *et al* 2002; Amory *et al* 2008; Bicalho *et al* 2008). Other production effects have been linked to lameness, such as reduced expression of oestrus because of increased lying times (Walker *et al* 2008).

With this mounting economic evidence for the cost of lameness and the clear welfare infringement caused by lameness we can consider that we have a win-win situation: if farmers respond to evidence that they are losing money from lame cows they should be prepared to change their management to reduce the prevalence and incidence of lameness. However, the situation is complex. There are many causes of lameness and incomplete information on their prevention. Therefore, farmers might be prepared to invest money to improve the management of cattle to reduce lameness but they need evidence on what will be effective.

The two broad strategies to manage lameness are prevention and prompt treatment. When we consider prevention of new cases of lameness, a first consideration is the cow herself. Several authors have reported that higher yielding cows in a herd have a greater risk of becoming lame (Barkema *et al* 1994; Green *et al* 2002; Bicalho *et al* 2008), and in the current study, cows with DD and DS had significantly higher yields initially. Can we prevent high yielding cows from becoming lame? High yielding cows have many diseases (Rajala-Schultz *et al* 1999) and the common cause might be that farmers are tending to the needs of the average

https://doi.org/10.1017/S0962728600001901 Published online by Cambridge University Press

cows' husbandry in a herd rather than the highest producing cows. In a study in 1979, Hansen and colleagues demonstrated that when genetically selected high yielding cows are managed as unselected cows they are more vulnerable to many diseases. However, Heikkilä *et al* (2008) reported that there is considerable variation between farms in management of health, leading to a wide variation in risk of disease and that disease can be lowered with better management. Whilst we can aim to ensure that all herds are managed to the highest yielding cows in the herd, it must also be that there is an absolute maximum yield above which cows cannot feed and rest for sufficient hours to maintain their health and produce the volumes of milk required, however excellent a farm's husbandry. Hansen (1999) mooted this some twenty years after his first study (Hansen *et al* 1979) and suggested that there is an absolute maximum yield that we can expect from dairy cows. We propose that for good welfare of dairy cows there is an absolute maximum yield that can be produced and that farmers should aim to have cows that produce an optimal yield for the farm management that enables them to be healthy and long-lived.

When we consider the external factors linked to prevention of lameness we have to consider that on most farms there are many causes of lameness (Blowey 2008). The risk factors linked to each cause of lameness vary and the evidence for causality for these factors, as opposed to association, also varies by lesion. This is discussed below. There is evidence that it is possible to reduce SU and tarsal damage by minimising unnecessary standing (Barker 2007; Rutherford *et al* 2008; Barker *et al* 2009). Unnecessary standing occurs when there is lack of access or an inadequate number of cubicles or when they are too small (Haskell *et al* 2006; Fregonesi *et al* 2007a), when the lying areas are wet (Fregonesi *et al* 2007b), or uncomfortable (Cook *et al* 2004). Prolonged standing also occurs when cows queue for food, water or to be milked. Barker's (2007) intervention study suggests that 24 farmers in the UK were able to improve cow comfort and reduce standing time using very cheap measures of additional bedding and reduced queuing times for milking and this reduced sole ulcer rates in the following year by approximately 15%, or 1–2 lame cows per 100 on a farm.

We do not have strong evidence on managements that could be used to reduce WLD. Although walking and twisting actions (Chesterton 2004) and soft, wet horn (Borderas *et al* 2004; Barker *et al* 2009) all apparently contribute to the development of WLD, and possibly sole ulcer (van Amstel *et al* 2004), there are no clinical trials to inform whether this lesion can be prevented by changing the above. However, it seems biologically very sensible (but rare in practice) that cows should have dry feet and so hard hoof horn to reduce wear from hard surfaces and maintain integrity and so prevent both non-infectious and infectious claw diseases.

Digital dermatitis, once on a farm, can be controlled but not eradicated.

Routine foot trimming has been proposed to prevent lameness in dairy cows. In clinical trials it has been shown to be beneficial in tied cattle but not in loose-housed cattle (Manske *et al* 2002), possibly because in tied cattle the hoof horn is not worn away as it is in loose-housed cattle that walk considerable distances each day. In observational studies there was a higher prevalence of lameness in herds that used routine foot trimming than in those that did not (Barker *et al* 2007; Espejo *et al* 2007). This could have occurred because routine foot trimming was targeted at lame cattle rather than to prevent lame cattle. This approach could explain one reason why cows remain lame for long periods; they are waiting for treatment from an external foot trimmer.

Lack of robust clinical trials might suggest that even if farmers want to prevent lameness we cannot guarantee that changes in management, which could be expensive, would be effective. Whilst all cattle should have ready access to a clean, dry walking and lying area we do also need information from well-designed clinical trials to identify management factors that prevent lameness in dairy cows.

However, with no strong evidence for prevention, the *status quo* of 20–60% unsound cows in a herd (Green *et al* 2002; Barker *et al* 2009) (some for prolonged periods) is not acceptable. We propose that we need to change the attitude of many farmers and veterinarians towards the management of lame cows. The reduction in yield before treatment in the current study (for all lesions except white line disease) and other studies (Green *et al* 2002; Amory *et al* 2008), suggest that lameness is impacting on the well-being of cattle for a considerable time (up to four months) before they are treated for lameness. What is unknown, and unfortunately was not recorded in the current study, is at what point these cattle can be detected lame, but it is likely to be well before they were treated. Despite the ideal situation that all lame cows should be treated as soon as they are lame, the reality is that the time to treatment of many lame cows could be reduced considerably.

Several authors have reported that farmers underestimate the prevalence of lameness in their dairy cattle (Whay *et al* 2003; Espejo *et al* 2006). This might be because they do not recognise cattle as lame or that, as with sheep farmers (Kaler & Green 2008), they recognise even mildly lame cows but make a separate decision on whether a cow is 'sufficiently lame' to treat. The use of locomotion or mobility scoring (eg Sprecher *et al* 1997) is being used to assess lameness in dairy herds. This is a useful tool if it assists farmers' sensitivity in detection of unsound cows. Researchers use locomotion scores to define lameness, but unsound (mildly lame) cattle are often defined as clinically not lame (eg Nordlund *et al* 2004). This suggests that locomotion scoring is too sensitive (ie selects non-lame cows). In fact, it is probably too specific: many mildly lame cows and some non-lame cows have hoof lesions (Manske *et al* 2002; Bicalho *et al* 2007; Tadich *et al* 2010).

Animal welfare implications

If we use mobility scoring or another technique to train farmers to recognise and treat cattle as soon as they are mildly lame then this will be a great welfare benefit and it will reduce the prevalence of lameness in a herd. It would

https://doi.org/10.1017/S0962728600001901 Published online by Cambridge University Press

probably also reduce the loss in milk yield and lead to a more rapid recovery because the duration of lameness and foot pathology will be less severe. Whilst rapid treatment will not alter the incidence (new cases) of lameness in a herd, it will highlight the number of cows requiring treatment. We hypothesise that this, in turn, might prompt farmers to alter management of their cows to reduce the incidence of lameness, trying out managements listed above and their own experiences, eg when cows in their herd become lame and with which lesion might highlight that management of newly calved cattle could be altered to prevent lameness in this particularly vulnerable group. It will also drive further research into preventing lameness in dairy cows.

Conclusion

In 1,635 cattle from seven herds in Chile studied for one year, lameness was associated with a reduced milk yield before treatment and an improvement in yield after treatment. The association between treatment for lameness and reduced yields has been reported in several other studies. Lameness in dairy cows is one of the most intractable welfare issues affecting dairy cows today. Its aetiology is complex and multifactorial and only elucidated in part. We will spend many more years resolving lameness in dairy cows including challenging issues such as aetiology and pathogenesis and prevention. However, there is a considerable amount that we can do now by prompt treatment that will improve cow welfare and reduce the impact of lameness on dairy cow welfare.

Acknowledgements

We thank the farmers who participated in this study and Dr Efren Flor for helping to collect the data. The present study was financed by the Projects FONDECYT 1040176 and 7040181.

References

Amory JR, Barker ZE, Wright JL, Mason SA, Blowey RW and Green LE 2008 Associations between sole ulcer, white line disease and digital dermatitis and the milk yield of 1,824 dairy cows on 30 dairy cow farms in England and Wales from February 2003-November 2004. *Preventive Veterinary Medicine 83*: 381-391

Barkema HW, Westrik JD, van Keulen KAS, Schukken YH and Brand A 1994 The effects of lameness on reproductive performance, milk production and culling in Dutch dairy farms. *Preventive Veterinary Medicine 20*: 249-259

Barker ZE 2007 *Epidemiology of lameness in dairy cows.* PhD Thesis, University of Warwick, Coventry, UK

Barker ZE, Amory JR, Wright JL, Blowey RW and Green LE 2007 Management factors associated with impaired locomotion in dairy cows in England and Wales. *Journal of Dairy Science 90*: 3270-3327

Barker ZE, Amory JR, Wright JL, Mason SA, Blowey RW and Green LE 2009 Risk factors for increased rates of sole ulcers, white line disease, and digital dermatitis in dairy cattle from twenty-seven farms in England and Wales. *Journal of Dairy Science 92*: 1971-1978

Bicalho RC, Cheong SH and Cramer G and Guard CL 2007 Association between a visual and an automated locomotion score in lactating Holstein cows. *Journal of Dairy Science 90*: 3294-3300

Bicalho RC, Warnick LD and Guard CL 2008. Strategies to analyse milk losses caused by diseases with potential incidence throughout the lactation: a lameness example. *Journal of Dairy Science 91*: 2653-2661

Blowey RW 2008 *Cattle Lameness and Hoofcare, An Illustrated Guide, Second Edition*. Old Pond Publishing Ltd: Ipswich, UK

Borderas TF, Pawluczuk B, de Passillé AM and Rushen J 2004 Claw hardness of dairy cows: relationship to water content and claw lesions. *Journal of Dairy Science 87*: 2085-2093

Chesterton RN 2004 Linking farm physical conditions herd management and cow behaviour to the distribution of foot lesions causing lameness in pasture-fed dairy cattle in New Zealand. *Proceedings of the 13th International Symposium and 5th Conference on Lameness in Ruminants* pp 200-202. 11-15 February 2004, Maribor, Slovenia

Cook NB, Bennett TB and Nordlund KV 2004 Effect of free stall surface on daily activity patterns in dairy cows with relevance to lameness prevalence. *Journal of Dairy Science 87*: 2912-2922

Coulon JB, Lescourret F and Fonty A 1996 Effect of foot lesions on milk production by dairy cows. *Journal of Dairy Science 79*: 44-49

Dyer RM, Neerchal NK, Tasch U, Wu Y, Dyer P and Rajkondawar PG 2007 Objective determination of claw pain and its relationship to limb locomotion score in dairy cattle. *Journal of Dairy Science 90*: 4592-4602

Espejo LA and Endres MI 2007 Herd-level risk factors for lameness in high-producing Holstein cows housed in free-stall barns. *Journal of Dairy Science 90*: 306-314

Espejo LA, Endres MI and Salfer JA 2006 Prevalence of lameness in high-producing Holstein cows housed in free-stall barns in Minnesota. *Journal of Dairy Science 89*: 3052-3058

Fregonesi JA, Tucker CB and Weary DM 2007a Overstocking reduces lying time in dairy cows. *Journal of Dairy Science 90*: 3349-3354

Fregonesi JA, Veira DM, von Keyserlingk MAG and Weary DM 2007b Effects of bedding quality on lying behavior of dairy cows. *Journal of Dairy Science 90*: 5468-5472

Green LE, Hedges VJ, Schukken YH, Blowey RW and Packington AJ 2002 The impact of clinical lameness on the milk yield of dairy cows. *Journal of Dairy Science 85*: 2250-2256

Hansen LB 1999 Consequences of selection for milk yield from a geneticist's viewpoint. *Journal of Dairy Science 83*: 1145-1150

Hansen LB, Young CW, Miller KP and Touchberry RW 1979 Health care requirements of dairy cattle in response to milk yield selection. *Journal of Dairy Science 62*: 1922-1931

Haskell MJ, Rennie LJ, Bowell VA, Bell MJ and Lawrence AB 2006 Housing system, milk production, and zero-grazing effects on lameness and leg injury in dairy cows. *Journal of Dairy Science 89*: 4259-4266

Heikkilä A-M, Nousiainen JI and Jauhiainen L 2008 Optimal replacement policy and economic value of dairy cows with diverse health status and production capacity. *Journal of Dairy Science 91*: 2342-2352

Hernandez J, Shearer JK and Webb DW 2002 Effect of lameness on milk yield in dairy cows. *Journal of the American Veterinary Medical Association 220*: 640-644

Kaler J and Green LE 2008 Recognition of lameness and decisions to catch for inspection among sheep farmers and specialists in Great Britain. *BMC Veterinary Research 14(4)*: 41

Manske T, Hultgren J and Bergsten C 2002 The effect of claw trimming on the hoof health of Swedish dairy cattle. *Preventive Veterinary Medicine 54*: 113-129

Nordlund KV, Cook NB and Oetzel GR 2004 Investigation strategies for laminitis problem herds. *Journal of Dairy Science 87*: E27-E35

https://doi.org/10.1017/S0962728600001901 Published online by Cambridge University Press

Rajala-Schultz PJ, Gröhn YT and McCulloch CE 1999 Effects of milk fever ketosis and lameness on milk yield in dairy cows. *Journal of Dairy Science 82*: 288-294

Rasbash J, Brown W, Healy M, Cameron B and Charlton C 1999 *MLwiN Version 11 Multilevel Models Project.* Institute of Education: University of London, UK

Rowlands GJ and Lucey S 1986 Changes in milk yield in dairy cows associated with metabolic and reproductive disease and lameness. *Preventive Veterinary Medicine 4*: 205-222

Rutherford KM, Langford DFM, Jack MC, Sherwood L, Lawrence AB and Haskell MJ 2008 Hock injury prevalence and associated risk factors on organic and nonorganic dairy farms in the United Kingdom. *Journal of Dairy Science 91*: 2265-2274

Sprecher DJ, Hostetler DE and Kaneene JB 1997 A lameness scoring system that uses posture and gait to predict cattle reproductive performance. *Theriogenology 47*: 1179-1187

Tadich N, Flor E and Green LE 2010 associations between hoof lesions and locomotion score in 1,098 unsound dairy cows. *The Veterinary Journal 184*: 60-65

van Amstel SR, Shearer JK and Palin FL 2004 Moisture content thickness and lesions of sole horn associated with thin soles in dairy cattle. *Journal of Dairy Science 87*: 757-763

Walker SL, Smith RF, Routly JE, Jones DN, Morris MJ and Dobson H 2008 Lameness activity time-budgets, and estrus expression in dairy cattle. *Journal of Dairy Science 91*: 4552-4559

Warnick LD, Janssen D, Guard CL and Gröhn YT 2001 The effects of lameness on milk production in dairy cows. *Journal of Dairy Science 84*: 1988-1997

Wassink GJ, King EM, Grogono-Thomas R, Brown JC, Moore LJ and Green LE 2010 A within-farm clinical trial to compare two treatments (parenteral antibacterials and hoof trimming) for sheep lame with footrot. *Preventative Veterinary Medicine 96*: 93-103

Wells SJ, Trent AM, Marsh WE and Robinson RA 1993 Prevalence and severity of lameness in lactating dairy cows in a sample of Minnesota and Wisconsin herds. *Journal of the American Veterinary Medical Association 202*: 78-82

Whay HR, Main DCJ, Green LE and Webster AJF 2003 Assessment of the welfare of dairy cattle using animal-based measurements: direct observations and investigation of farm records. *Veterinary Record 153*: 197-202

Whay HR, Waterman AE and Webster AJF 1997 Associations between locomotion claw lesions and nociceptive threshold in dairy heifers during the peripartum period. *Veterinary Journal 154*: 155-161

Wilmink JBM 1987 Adjustment of test-day milk fat and protein yields for age season and stage of lactation. *Livestock Production Science 16*: 135

https://doi.org/10.1017/S0962728600001901 Published online by Cambridge University Press