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Temporal associations between low body condition, lameness and milk yield in a UK dairy herd

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Abstract

Previous work has hypothesised that cows in low body condition become lame. We tested this in a prospective longitudinal study. Body condition score (BCS), causes of lameness and milk yield were collected from 600-cow herd over 44-months. Mixed effect binomial models and a continuous outcome model were used to investigate the associations between lameness, BCS and milk yield. In total, 14320 risk periods were obtained from 1137 cows. There were 1510 lameness treatments: the most common causes of lameness were sole ulcer (SU) (39%), sole haemorrhage (SH) (13%), digital dermatitis (DD) (10%) and white line disease (WLD) (8%). These varied by year and year quarter. Body condition was scored at 60-day intervals. BCS ranged from 1–5 with a mean of 2.5, scores were higher in very early lactation but varied widely throughout lactation; approximately 45% of scores were <2.5. The key finding was that BCS<2.5 was associated with an increased risk of treatment for lameness in the following 0-2 months and >2–4 months for all causes of lameness and also specifically for SU/WLD lameness. BCS<2.5 was associated with an increased risk of treatment for SH in the following 0-2 months but not >2–4 months. There was no such association with DD. All lameness, SU/WLD, SH and DD were significantly more likely to occur in cows that had been lame previously, but the effect of BCS was present even when all repeat cases of lameness were excluded from the analysis. Milk yield was significantly higher and fell in the month before treatment in cows lame with SU/WLD but it was not significantly higher for cows that were treated for DD compared with non-lame cows. These findings support the hypothesis that low BCS contributes to the development of horn related claw lameness but not infectious claw diseases in dairy cows. One link between low BCS and lameness is a thin digital cushion which has been proposed as a trigger for claw horn disease. Cows with BCS 2 produced more milk than cows with BCS 2.5, however, this

was only approximately 100 Kg difference in yield over a 305-day lactation. Given the increased risk of lameness in cows with BCS 2, the direct costs of lameness and the small variability in milk yield by BCS, preventing cows from falling to BCS <2.5 would improve cow welfare and be economically beneficial.

Key words Dairy cow, Lameness, Body Condition Score, Milk yield, Mixed effect binomial model, MCMC parameterisation

Introduction

Lame cows are in pain and their welfare is compromised (Whay et al., 1997). The mean prevalence of lameness in dairy cows has been estimated to be 21 % (Clarkson et al., 1996) and 36 % (Leach et al., 2010) in the UK and the incidence rate has been reported to be as high as 70 cases / 100 cows / year (Hedges et al., 2001). Similar levels of lameness in dairy cows are reported in many other countries.

Non infectious and infectious causes of lameness have been associated with a reduction in milk yield both before and after treatment (Warnick et al., 2001; Green et al., 2002; Amory et al., 2008; Bicalho et al., 2008), with large decreases in yield associated with the non infectious claw lesions sole ulcer and white line disease (Amory et al., 2008; Green et al., 2010). One explanation for reduction in milk yield before treatment is that lame cows are not treated immediately (Leach et al., 2012). There is mixed evidence for this: Reader et al. (2011) reported that a reduction in milk yield occurred before cows became lame but Archer et al. (2010) reported reductions in milk yield only after cows were detectably lame. Reader et al. (2011) proposed that either the mobility scoring technique was insufficiently sensitive to detect mildly lame cows (and indeed, some non-lame cows do have foot lesions, (Manske et al., 2002; Tadich et al., 2010) or that milk yield and lameness are on a common causal pathway where an underlying insult leads to both disorders.

One associated risk for claw horn lameness is a thin digital cushion (Raber et al., 2004). In a cross sectional study, Bicalho et al. (2009) reported that lame cows had a thinner digital cushion than non-lame cows and that these cows were thin. These authors hypothesised that if the cushion became thin before a cow was lame, then lameness might occur because a thin digital cushion fails to protect the sensitive tissue of the hoof from concussive forces that lead to bruising at the site of sole ulcers, the white line or sole haemorrhage. Unfortunately the cross sectional design of the study meant that cause and effect could not be elucidated, however, the authors (Bicalho et al., 2009) did report that a thin digital condition was correlated with low body condition score (BCS).

Body condition score impacts on the health and productivity of dairy cattle considerably, with both high and low BCS affecting milk yield and health. For example, low BCS has been associated with low milk yield (Roche et al., 2007a) and conception (Pryce et al., 2001; Roche et al., 2007b) whilst a high BCS has been associated with ketosis, disease and lower milk yield (Gillund, et al., 2001; O'Boyle et al., 2006). The aim of this study was to investigate the impact of BCS on the subsequent development of lameness in dairy cows and the inter relationship between milk yield, BCS and lameness.

Materials and methods

The 44-month study was carried out between 2008 and 2011 on one dairy farm in Somerset, England with ~600 Holstein cows. The herd was selected on size and willingness of the senior herdsman to be trained and to collect detailed and accurate farm records. Milking cows were grouped into early, mid and late lactation groups and fed accordingly. Rations were analysed regularly and adjusted by a nutritionalist with the aim of maximising milk yield whilst minimising feed costs. Dietary ingredients were kept the same where possible to limit the effects of sudden dietary changes. Biotin was added to the ration at 20mg/cow/day to improve hoof horn quality (Hedges et al., 2001). The cows were milked twice each day in a 60 point rotary parlour. Cattle were housed 24 hours / day all year around, except for those in approximately the last 2 months of lactation during the summer grazing period which were at pasture. The cows were housed in modern free stall accommodation with water mattresses in cubicles and solid concrete passageways with automatic slurry scrapers working at a frequency of 1 scrape / hour, stocked to a maximum of 95% capacity. The median age at calving was 25 months across all years of the study. Culling rates were 29% (2008), 31% (2009) 32% (2010) and 29% (2011). Mean 305-day yield was approximately 10000 Kg per cow.

A professional foot trimmer attended the herd each month to trim cows' feet to prevent lameness, typically cows at the end of lactation and those with clearly misshapen feet were trimmed. Each cow had a minimum routine foot trim at least once per year. The senior herdsmen selected lame cows (identifiably impaired mobility) during daily observations of the herd. These cows were then treated by the herdsman, generally within 2 - 3 days, under veterinary direction using agreed standard treatment protocols specific for the diagnosis and severity of lesions. Lesions were recorded using a standard definition based on that defined by the EU Lamecow project (http://warwick.ac.uk/cattlelameness/colour_atlas.pdf).

The head herdsman scored body condition on a scale of 0 - 5 in 0.5 increments based on examination of the transverse processes of the lumbar vertebrae, the ribs, ischial tuberosity, ligaments of the pelvis and surrounding fat. He was trained by veterinarians (author MJG and colleague James Breen (JB)) and scoring technique was checked during weekly routine herd visits to prevent drift in scoring. The herdsman recorded BCS for each cow at approximately 60-day intervals, throughout the entire study period. All health, production, BCS, and treatments for lameness were recorded in Interherd (National Milk Records) and updated each day.

Statistical analysis

Data were obtained for 44 months, from January 2008 - September 2011. All unusual or incorrect field entries were removed from the dataset; this was <1% of the data. Incident treatment for clinical lameness was the outcome variable and cows were categorised into not treated (0) or treated for lameness (1) in consecutive 30-day periods. Lesions causing lameness were then grouped into all causes, sole haemorrhage (SH), sole ulcer / white line disease (SU/WLD) and digital dermatitis (DD). The temporal distributions of lameness and BCS were investigated graphically by year quarter.

Mixed effect binomial logistic regression models (Goldstein, 1995) were used to analyse the lameness data. There were four models with the outcomes all causes of lameness, SH, SU/WLD and DD in a 30-day period with repeated observations included in the models as a random effect and time since last case of lameness as a fixed effect. The baseline was always non-lame (i.e. not treated) cows, so when specific causes of lameness were investigated cattle lame with any other cause of lameness were excluded.

The explanatory variables tested were parity (categorical 1 – 6+), days in milk (at the end of a 30-day period), year quarter, month in herd, time since previous episode of lameness (data were available from 2002) (categorised from time t in 30-day intervals to >150 days), milk yield (kg per day) measured at the most recent monthly milk recording, BCS (mean BCS where there were 2 recordings) in 60 day intervals (categorical on a scale of 1-5 with increments of 0.5 and also as a binary indicator; BCS ≤ 2 and BCS >2). BCS and milk yield were also lagged to investigate effects before and after a lameness event.

The models took the form

$$\text{Lame}_{ij} \sim \text{Bernoulli (probability} = \pi_{ij})$$

$$\mathrm{Logit}\,(\pi_{ij}) = \alpha + \beta_1 X_{ij} + \beta_2 X_j + u_j$$

$$u_j \sim N(0,\sigma^2_u)$$

where the subscripts i, and j denote the ith observation of the jth cow respectively, α the regression intercept, X_{ij} the vector of covariates associated with each observation, β_1 the coefficients for covariates $X_{ij,}$ X_j the vector of covariates associated with each cow, β_2 the coefficients for covariates $X_{j,}$, u_j a random effect to reflect residual variation between cows which was assumed to follow an unordered correlation structure and a normal distribution with mean zero and variance σ^2_u. Initial covariate assessment was carried out using MLwiN with penalised quasi-likelihood for parameter estimation (Rasbash et al., 2005). Missing observations were grouped and fitted in the model as a category within discrete variables to minimise loss of data (coefficients are not presented or interpreted).

Final parameter estimates were made using Markov chain Monte Carlo (MCMC) in WinBUGS (Spiegelhalter et al., 2004), to avoid the potential biased estimates that can arise from quasi-likelihood methods with binary data (Browne and Draper, 2006). Vague, flat normal distributions were specified for the fixed effects (Normal distribution, mean=0, variance=10^6) and a vague gamma distribution for random effect precision (~Gamma distribution (mean=0.001, variance=10^3)). Covariates were left in the model when the 95% credibility intervals for the odds ratios did not include 1.00. The MCMC analyses used a burn-in of 1000 iterations during which time model convergence had occurred. Parameter estimates were based on a further 9,000 iterations. Investigation of model fit was conducted by comparing posterior simulations of cumulated model probabilities with the observed data to identify areas of major discrepancy (Gelman et al., 1996). Posterior predictions of the relative risks of lameness for cows with different body condition score were also estimated and plotted. Comprehensive details of MCMC modeling (Gilks et al., 1996; Spiegelhalter et al., 2004) and the methods adopted for this research (Browne and Draper, 2006; have been described in detail previously (Green et al., 2004). The data were also analysed as descrete time survival models with the first case of lameness in a parity only included, data for a cow were censored after this first lameness event, and the covariate for previous lameness was left to account for lameness from previous parities.

The associations between BCS and milk yield were also modelled in a continuous outcome mixed effect model with milk yield. The model took the form:

$Y_{ij} = \alpha + \beta_1 X_{ij} + \beta_2 X_j + v_j + e_{ij}$

$v_j \sim N(0, \Omega^2{}_v), e_{ij} \sim N(0, \Omega^2{}_e)$

where Y is the daily milk yield measured once each month by the milk recording organisation, the subscripts i, and j denote the i^{th} observation of the j^{th} cow respectively, α the regression intercept, X_{ij} the vector of covariates associated with each observation, β_1 the coefficients for covariates $X_{ij,}$ X_j the vector of covariates associated with each cow, β_2 the coefficients for covariates $X_{j,,}$ v_j a random effect to reflect residual variation between cows (mean = 0 and variance $\Omega^2{}_v$), e_{ij} a random error term to reflect residual variation between observations (mean = 0 and variance $\Omega^2{}_e$).

The following variables were tested in the model, parity, days in milk, exp (days in milk *-0.05) (Wilmink, 1985), lameness, BCS lagged and interactions between BCS and days in milk and the function of days in milk. Investigation of model fit was conducted using conventional residual analysis.

Results

A total of 14320 risk periods were obtained from 1137 cows with a mean of 10 (range 1- 36) observations per cow over the 44 months of the study. There were 1510 lameness treatments that occurred throughout the 44-month period with variability in number treated per year quarter, with more cases of SU and fewer of SH in the final years of the study (Figure 1a). There was a slight seasonal pattern for lameness with DD, there were more cases in winter than summer but there was no seasonal pattern with any of the other lesions (Figure 1a). Lameness occurred throughout the 305-day lactation. The most common cause of lameness was sole ulcer (39%), followed by sole haemorrhage (13%), digital dermatitis (10%) and white line disease (8%). Individuals had up to 8 treatments for lameness.

There were 15150 body condition scores (0 – 2 and >2 – 4 months before an observation) over the 44-month period; there was no trend in BCS over time (Figure 1b). Body condition score was normally distributed with a mean of 2.5; very few scores were 1 (43) or 5 (63). Throughout lactation BCS was highly variable between cows although there was a tendency for BCS to decrease in early lactation (by approximately 0.5 points) and increase towards the end of lactation (Figure 2).

In the binomial models, cows that had been lame previously were at highly significant risk of becoming lame with all four outcomes. Body condition score < 2.5 (compared with BCS > 2) in the 0 - 2 and >2 – 4 months before a 30 d risk period were both associated with an increased risk of lameness for all causes and for SU / WLD (Table 2). BCS <2.5 in the 0 – 2 months before a 30-day risk period was significant for cases of SH, but not in the risk period >2 – 4 months before a case of SH. There was no association between BCS and subsequent risk of DD. Cows lame from all causes or SU/WLD had a higher yield than non-lame cows the month before lameness and a lower yield the month they were lame. This was not the case for DD and SH (Table 2). All causes of lameness were more common in July – September compared with January – March but there were no significant patterns of lesion specific causes of lameness by year quarter. The longer cattle remained in the herd (month in herd) the less likely they were to be lame from all causes or SU/ WLD (Table 2). In the discrete time survival models approximately 4200 records of data were censored. The model coefficients were very similar to those in the full models, differing by an OR <0.04. All covariates that were significant in the full models were significant in the discrete time models (data not shown).

The effect of BCS on milk yield was complex and interacted with stage of lactation (Figure 3) and when cows became lame. Cows that were lame produced 0.9 (s.e. = 0.16) kg less milk per day than non lame cows. The longer the time from a previous case of lameness the greater the milk yield at a recording (Table 3); indicating that cows that became lame were more likely to be higher yielding cows than those that were never lame, but that yield was lower near to a lameness event. Overall, there were small differences between BCS categories in total milk yield over the 305-day lactation (approximately 100 Kg (0.9%), Figure 3). Model fit was good; the posterior estimates of the relative risk of lameness for cows with different body condition scores were similar to the observed values (Table 4).

Discussion

To the authors' knowledge this is the first longitudinal study that provides evidence that sole ulcer and white line disease, both pathologies of hoof horn, are associated with cows with prior low body condition, even when only the first case of lameness in a parity is modelled with adjustment for lameness in previous parities. Cows with BCS <2.5 (on a scale of 1 – 5) were more likely to become lame in the following 2 and >2 - 4 months than those with BCS >2 in this time period. Sole haemorrhage, often considered a more mild or earlier presentation of SU was more likely in cows with BCS <2.5 in the previous 2 months only, possibly indicating an early stage of SU or WLD. Digital dermatitis, an infectious cause of lameness, was not associated with prior low body condition.

These results provide evidence that low BCS (<2.5) is a risk for the principal non-infectious claw diseases SU / WLD and the milder SH. One explanation for why low BCS is related to these causes of lameness is that low BCS is associated with a reduction in the depth of the digital cushion and this in turn is associated with claw horn lameness (Bicalho et al. 2009). As cows mobilise fat from all adipose tissues, including the digital cushion, the volume of fat in the digital cushion is reduced, either leading to increased bruising because the digital pad does not prevent concussive forces or leading to increased movement of the third phalanx within the hoof horn capsule (Tarlton et al., 2005) that result in the third phalanx causing pressure necrosis and ulceration over the sole or white line and disrupting hoof horn production in these areas (Lischer et al. 2002). The association between prior low BCS and lameness might also help explain results by Reader et al. (2011) who reported that milk yield decreased before locomotion was visibly impaired; if a reduction in milk yield is associated with reduced BCS and subsequent claw horn lameness then reduced yield might occur before cows are lame.

As importantly, to date, the emphasis for risks for horn diseases has been focused on external factors such as standing time and cubicle comfort (Barker et al., 2008; Norring et al., 2012) and this is the first longitudinal study to highlight that body condition <2.5, and therefore inadequate nutritional management (most likely in the highest yielding cows in a herd), is also a risk for claw horn disease.

In the current analysis, cattle that were treated for all causes and SU/WLD produced more milk than non-lame cows in the month before treatment. Milk yield fell to a small significant reduction in yield in the month of treatment. These results agree with the results from studies of the impact of a lameness event on milk yield where high yielding cows had a reduction in yield for up to five months before being treated (Green et al., 2002; Amory et al., 2008; Green et al., 2010). Several monthly lags in milk yield were tested in the models in the current paper, to investigate when milk yield started to fall, however, milk yields per month within cow were highly correlated explanatory variables and each month added negated the effect of previous months, so only the month before treatment and current month yields were left in the model. The lack of association between prior BCS, prior milk yield and DD and significant association between prior BCS, prior milk yield and claw horn diseases in this prospective study do provide weight to the evidence that the link between claw horn disease and low BCS is causal.

There was remarkably little difference in milk yield over lactation by body condition score (Figure 3). It has been reported elsewhere that cattle that are either very thin or overly conditioned yield less milk (Gillund, et al., 2001; Roche et al., 2007a). The analysis from the current study in a herd with a high average yield of ~10000 Kg per 305-day lactation suggests that the maximum milk yield was produced by cows when in BCS 2, but that this was only equivalent to 100 Kg extra milk per 305-day lactation compared with cattle in BCS 2.5. Given that the highest yielding cattle in the herd were more likely to be BCS 2 and so more likely to become lame with claw horn lesions (Table 2), and so have reduced yield, the net benefit of 100 Kg milk yield from cows in BCS 2 would not cover the cost of treatment and high risk of repeated treatments and possibly early culling. We therefore conclude that BCS 2.5 – 3.0 is optimal to maximise milk yield and minimise lameness.

There were several other differences in risk between SU/WLD and DD; SU/WLD were equally frequent in all parities of cattle whilst digital dermatitis was more frequent in parity 1 cows compared with parities 3 – 6+. This was also reported by Barker et al. (2008) in a study of treatments for DD but is in contrast to Nielsen et al. (2012) who report from 11 weeks of weekly observations of feet that older cattle had more DD events. These results are not entirely contradictory, they possibly highlight the infectious nature of DD and its complex immunity – maybe parity 1 cattle become lame and require treatment whilst older cattle are more frequently, but more mildly, diseased and have fewer treatments for DD.

Cattle were treated on up to 8 occasions in the current analysis. Whilst for some lesions the repeated event might have been different feet on the same cow it is clear that some of the repeated events were the same digit or claw. When all causes of lameness were considered together, a case was more likely in cattle that had been lame previously and this was the biggest risk for lameness in the current analyses with odds ratios of 2.5 – 23 (Table 2). Reader et al. (2011) and Neilsen et al. (2012) used multistate models and reported that previous lameness increased the risk of a state transition from non-lame to lame. Their results and the current analysis suggest that treatments for lameness are possibly not highly effective or not long lasting; this is unlikely to be due to incorrect diagnosis and therefore inappropriate treatment per se (the treatments on this farm were done by one experienced herdsmen) but that the treatment was not effective. The apparent lack of efficacy of treatments in the current study is reflected in many studies of lameness where repeated lameness events are common. There are virtually no high quality clinical trials investigating the most appropriate treatments for SU/WLD (Potterton et al., 2012) and although there are a plethora of reports of treatments for DD, all report partial cures or reduction in the size of lesions. In addition, there is no information on whether treatment efficacy varies by those making treatments. There is clearly more to be done to improve the efficacy of treatments for lameness in dairy cows.

Treatments might also be ineffective if they do not address the underlying insult. If cows with claw lesions are lame primarily because the digital cushion in thin then treating the SH, SU or WLD will not resolve the thin cushion and claws might still be at risk of a new / recurrent case of lameness, particularly if the cow remains in low body condition. This was a prospective 44-month study of one large UK dairy-cow herd. The study was set up with one observer trained by veterinary researchers (MJG and JB) who made all BCS measurements and lameness treatments to avoid between observer bias. The detection of lameness was also made by the herdsman, JB and MJG and so the baseline untreated cattle were of consistent locomotion scores. This might have included some mildly lame cattle which would suggest that the results are, if anything, an underestimate of the impact of BCS on lameness and milk yield. The herdsman was monitored by both veterinarians to ensure consistency in recording over time; had the herdsman been inconsistent and misclassified animals the power of the study would have been reduced and statistical associations less strong; evidence for the consistency of these recordings comes from the statistical associations identified. We cannot know whether the herdsman was or became biased in deciding which cows to treat: bias could have led to false associations or false non-associations between lameness, BCS and yield. The associations with milk yield and lameness are consistent with other studies and we have no reason to consider that the herdsman was biased in selecting lame cows for treatment. Whilst the results come from one farm, it was a large farm with cows in a range of body condition and there is no reason to think that these results are not generalisable to other similar dairy cattle herds.

Conclusions

We conclude that lameness caused by pathology of the hoof horn (sole hemorrhage, sole ulcer and white line disease) was more likely in cattle with BCS <2.5 in the previous 0 – 2 and >2 – 4 months. Cattle lame with hoof horn lesions moved from milk yields above those of non-lame cows to those of non-lame cows in the month before they became lame. Low body condition was also associated with lower milk yield in the same 30-day interval. However, over the whole lactation there was no strong association between milk yield and BCS, indicating that cows with BCS < 2.5 were not more productive but were more likely to become lame and so reduce animal welfare and increase costs from treatment and milk loss. Digital dermatitis was not associated with low prior BCS or high prior milk yield and this adds to the specificity of the association between BCS and claw horn diseases and the hypothesis that these are aetiologically linked, possibly through thinning of the digital cushion.

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Figure 1a. Number of cases of sole ulcer and white line disease (black), digital dermatitis (grey) and sole haemorrhage (white) by year quarter from January 2008 – September 2011 in one herd of ~600 cows.

Figure 1b. Mean and 95% standard deviation body condition score for 15150 observations from January 2008 – September 2011 in one herd of ~600 cows.

Figure 2. Mean and 95% standard deviations of body condition score by days in milk from a 44 month prospective study of one 600 cow dairy herd, Somerset, UK

Figure 3. Predicted milk yields by body condition score (from model parameters in Table 3) per 305-day lactation from the 44 month prospective study in a 600 dairy cow herd, Somerset, UK

Table 1. Number and percent of each claw lesion identified during treatment for clinical lameness from a mean of 600 cows recorded for 44 months on one UK farm with 600 cows

Cause of claw lameness	number	percent
Sole ulcer	584	38.68
Bruised sole	196	12.98
White line disease	125	8.28
Digital dermatitis	151	10.00
Under run sole	112	7.42
Overgrown claw	47	3.11
Abscess	80	5.40
Interdigital phlegmon	30	1.99
Interdigital growth	68	4.50
Toe ulcer	27	1.79
Unknown	90	5.96
Total	1510	100.00

Table 2. Final models of risks for all causes of lameness and lameness caused by sole haemorrhage, sole ulcer / white line disease and digital dermatitis in a 600 cow herd in Somerset, UK

Variables	All causes of lameness			Sole haemorrhage			Sole ulcer / White line		
	Odds Ratio	L95% CI	U95% CI	Odds Ratio	L95% CI	U95% CI	Odds Ratio	L95% CI	U95% C
BCS > 2 last 0 - 2 m	0.63	0.54	0.73	0.41	0.28	0.59	0.72	0.57	0.9
BCS > 2 last 2 – 4 m	0.73	0.60	0.89	0.70	0.41	1.18	0.60	0.44	0.8
January - March	Baseline			Baseline			Baseline		
April - June	1.13	0.97	1.33	1.05	0.68	1.64	0.92	0.72	1.1
July - September	1.30	1.11	1.52	1.35	0.86	2.10	0.97	0.76	1.2
October - December	1.04	0.87	1.23	1.34	0.83	2.18	1.00	0.77	1.2
Month in herd	0.99	0.98	1.00	1.00	0.97	1.02	0.99	0.97	1.0
No previous lameness	Baseline			Baseline			Baseline		
lame: 1-30d ago	19.75	15.60	24.75	5.48	3.03	10.02	10.73	7.26	16.1
31-60d ago	13.80	10.58	17.78	7.08	3.67	13.50	12.53	8.17	18.8
61-90d ago	14.63	10.60	19.75	10.48	4.80	22.20	19.81	12.32	32.1
91-120d ago	14.10	10.08	19.51	16.09	7.34	34.12	23.69	14.64	39.1
>120d ago	16.12	12.35	20.68	15.26	8.47	28.93	19.95	13.44	30.3
Yield month before	1.011	1.004	1.018	1.006	0.986	1.026	1.020	1.010	1.03
Current yield	0.977	0.967	0.985	0.983	0.961	1.005	0.988	0.975	1.00
Days in milk	0.999	0.998	1.000	0.996	0.994	0.999	1.001	1.000	1.00
Parity 1	Baseline			Baseline			Baseline		
Parity 2	0.46	0.37	0.57	0.39	0.23	0.67	0.58	0.42	0.8
Parity 3	0.49	0.39	0.62	0.35	0.19	0.61	0.64	0.45	0.9
Parity 4	0.46	0.36	0.59	0.29	0.15	0.54	0.72	0.49	1.0
Parity 5	0.45	0.34	0.61	0.25	0.12	0.53	0.92	0.61	1.4
Parity 6+	0.40	0.30	0.55	0.36	0.17	0.71	0.76	0.49	1.2
Random term (variance and SD)	0.41 (0.05)			0.37 (0.21)			0.86 (0.11)		

BCS = body condition score, m = months, d = days, SD = standard deviation

Table 3. Mixed effect model on the impact of body condition score on daily milk yield (Kg) in a 44 month prospective study of one 600 cow dairy herd, Somerset, UK

Variables		Milk yield at current recording			
		mean	s.e.	lower 95% CI	upper 95% CI
Intercept		37.8	0.44	36.94	38.66
BCS in last 60 d:					
BCS = 2.5		Baseline			
BCS ≤1.5		2.81	0.91	1.03	4.59
BCS = 2.0		0.83	0.49	-0.13	1.79
BCS = 3.0		-0.87	0.42	-1.69	-0.05
BCS = 3.5		1.02	0.59	-0.14	2.18
BCS ≥ 4.0		-2.2	0.64	-3.45	-0.95
DIM		-0.06	0.002	-0.06	-0.06
Exp(DIM*-0.05)		-11.6	1.79	-15.11	-8.09
Interaction BCS and DIM and DIM*-0.05 BCS ≤2.5*DIM		Baseline			
BCS = 1.5*DIM		-0.016	0.006	-0.03	0.00
BCS = 2.0*DIM		-0.001	0.002	0.00	0.00
BCS = 3.0*DIM		-0.001	0.002	0.00	0.00
BCS = 3.5*DIM		-0.016	0.003	-0.02	-0.01
BCS ≥ 4.0*DIM		-0.008	0.003	-0.01	0.00
BCS ≤2.5*(Exp(DIM *-0.05))		Baseline			
BCS = 1.5*(Exp(DIM *-0.05))		-9.86	8.17	-25.87	6.15
BCS = 2.0*(Exp(DIM *-0.05))		-1.1	3.67	-8.29	6.09
BCS = 3.0*(Exp(DIM *-0.05))		7.47	2.48	2.61	12.33
BCS = 3.5*(Exp(DIM *-0.05))		4.34	2.71	-0.97	9.65
BCS ≥ 4.0*(Exp(DIM *-0.05))		7.19	2.63	2.04	12.34
Parity 1		Baseline			
Parity 2		4.23	0.19	3.86	4.60
Parity 3		6.75	0.24	6.28	7.22
Parity 4		7.1	0.3	6.51	7.69
Parity 5		8.38	0.38	7.64	9.12
Parity 6+		8.11	0.5	7.13	9.09
Not lame in last 30d		Baseline			
Lame in last 30d		-0.88	0.16	-1.19	-0.57
No previous lameness		Baseline			
Previously lame ago	31-60d	0.51	0.25	0.02	1.00
	61-90d ago	0.26	0.34	-0.41	0.93
	91-120d ago	0.2	0.38	-0.54	0.94
	121-150d ago	0.69	0.23	0.24	1.14

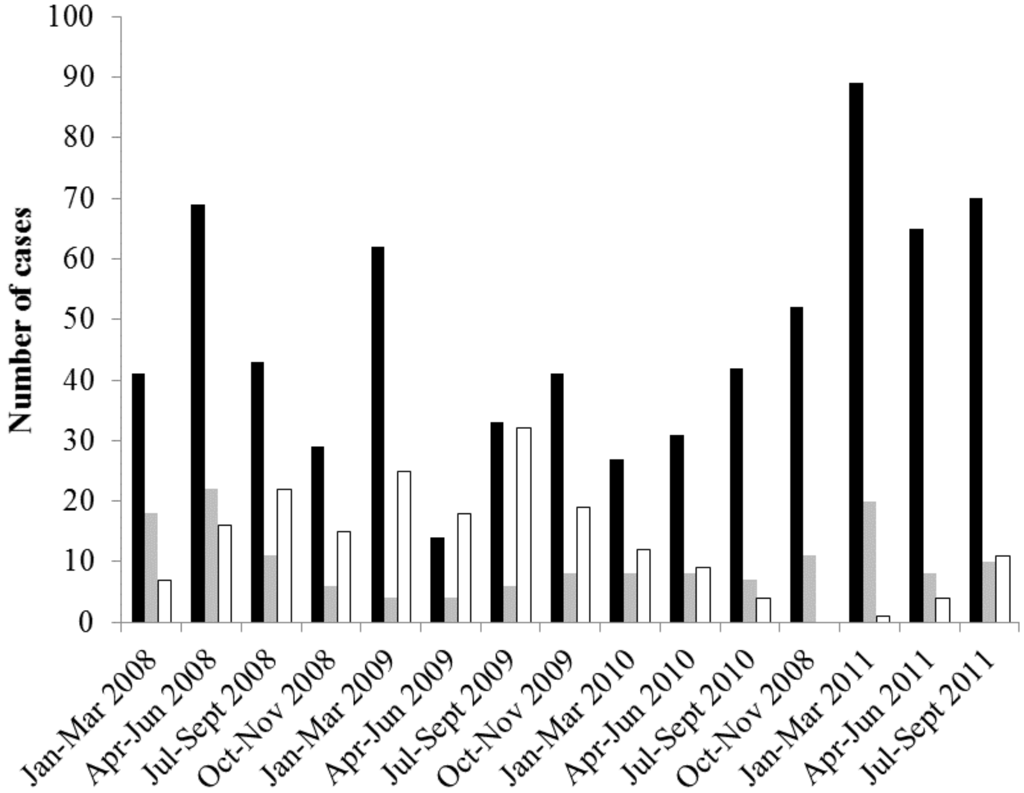
	>150d ago	0.83	0.34	0.16	1.50
Random effects		Variance	Standard error		
Cow		35.8	1.796		
Observation		41.8	0.512		

BCS = body condition score, DIM = days in milk, exp = exponential, d = days, CI = credibility interval,

Table 4. Model predictions of the relative risks of lameness conditional on body condition score over 44 months from a 600 cow from one farm in the UK

	Model predictions			Observed risk
	Mean	L 2.5% CI	U 97.5% CI	from raw data
RR of any cause of lameness if body condition score <2.5 in last 2 months to > 2 in last 2 months	1.61	1.43	1.82	**1.54**
RR of any cause of lameness if body condition score<2.5 in last 2-4 months to > 2 in last 2-4 months	1.42	1.17	1.70	**1.42**
RR of SU/WLD if body condition score <2.5 in last 2 months to > 2 in last 2 months	1.56	1.25	1.94	**1.44**
RR of SU/WLD if body condition score <2.5 in last 2-4 months to > 2 in last 2-4 months	1.81	1.28	2.64	**1.62**
RR of DD if body condition score<2.5 in last 2 months to > 2 in last 2 months	1.06	0.53	1.80	**0.94**
RR of DD if body condition score<2.5 in last 2-4 months to > 2 in last 2-4 months	1.54	0.63	3.33	**1.35**
RR of bruised sole if body condition score <2.5 in last 2 months to > 2 in last 2 months	2.62	1.54	4.20	**2.25**
RR of bruised sole if body condition score <2.5 in last 2-4 months to > 2 in last 2-4 months	1.67	0.75	3.38	**1.50**

RR = relative risk, L 2.5% CI = lower 2.5% credibility interval, U 97.5% CI = upper 97.5% credibility interval



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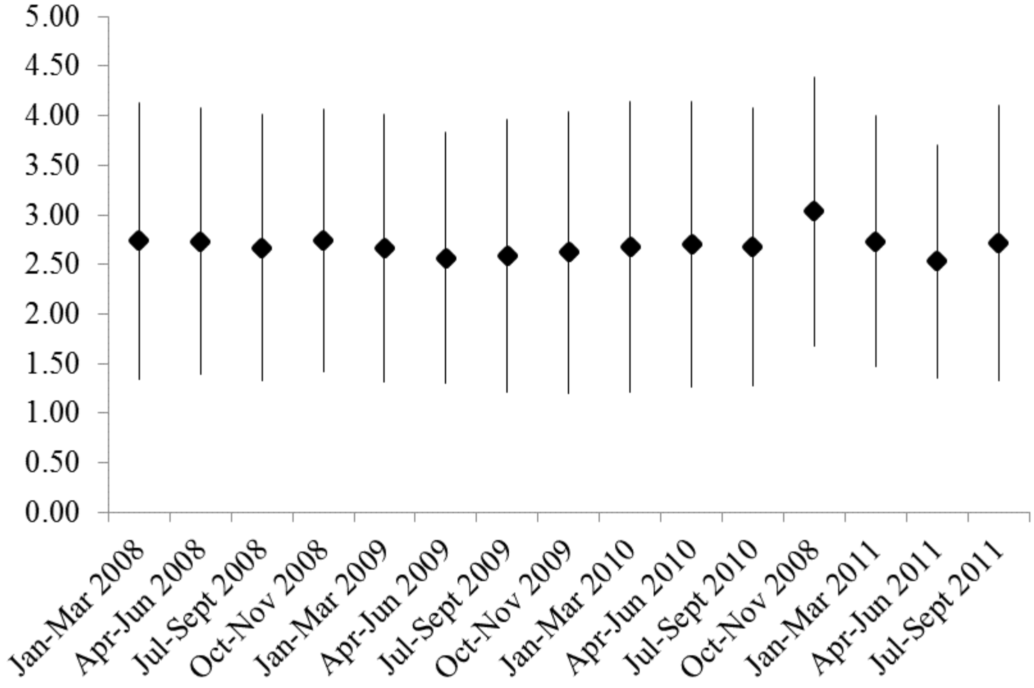

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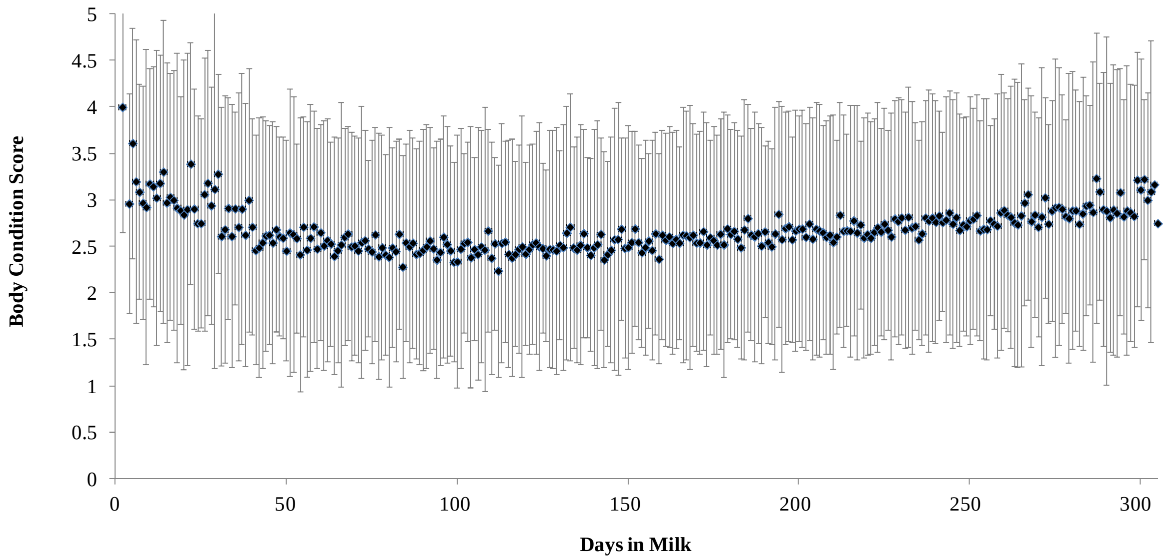
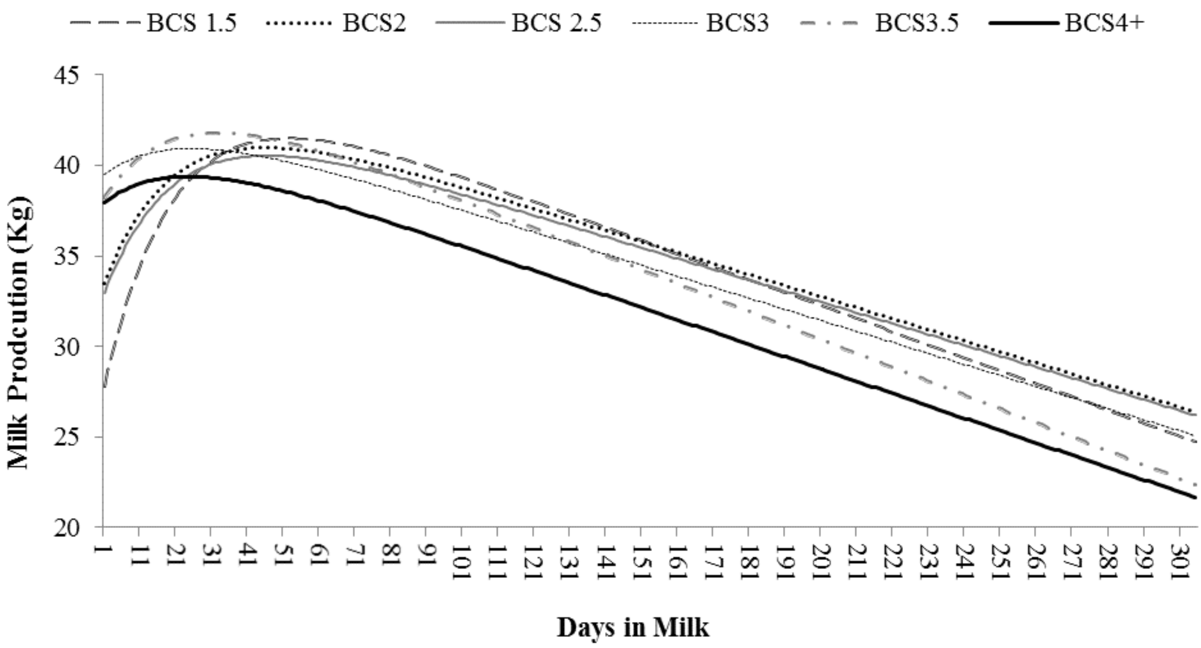


Figure 3. Predicted milk yields by body condition score (from parameters in Table 3) per 305-day lactation from 44 months prospective study from a 600 dairy cow herd, Somerset, UK



With these predictions the 305 day yields by body condition score (BCS) are BCS ≤ 1.5 = 10497 kg, BCS 2.0 = 10641, BCS 2.5 = 10537 ,BCS 3.0 = 10392, BCS 3.5 = 10234, BCS 4+ = 9669