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Published in:
Journal of Dairy Science

DOI:
10.3168/jds.2017-13439

First published: 23/11/2017

Document Version
Publisher's PDF, also known as Version of record

Interpretive Summary

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Randall

Low body condition score (BCS; a measure of fatness) and occurrence of previous lameness are risk factors for lameness in dairy cows. Estimating the contribution that risk factors make towards the total number of disease events in a population can identify control measures that could lead to the largest improvements on-farm. Using longitudinal data, repeated lameness bouts were found to contribute to a very large proportion of total lameness, highlighting the importance of this risk factor. In these herds, a lower proportion of total lameness may be avoidable by moving BCS into optimum ranges, compared to reducing repeated lameness bouts.

The Contribution of Previous Lameness Events and Body Condition Score to the Occurrence of Lameness in Dairy Herds: A Study of Two Herds

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Key words

Lameness, dairy cattle, population attributable fraction, body condition score, previous lameness events

ABSTRACT

It has been demonstrated that low body condition and previous occurrence of lameness increase the risk of future lameness in dairy cows. To date the population attributable fraction (PAF), which provides an estimate of the contribution that a risk factor makes towards the total number of disease events in a population, has not been explored for lameness using longitudinal data. Estimation of PAF helps to identify control measures that could lead to the largest improvements on-farm. The aim of this study was to use longitudinal data to evaluate the proportion of lameness that could be avoided in two separate herds (two populations), through i) reduced recurrence of previous lameness events ii) and moving body conditions score (BCS) into optimal ranges.

Data were obtained from two UK dairy herds; Herd A, a 200-cow herd with 8 years of data from a total of 724 cows where lameness events were based on weekly locomotion scores (LS; 1 to 5 scale) and Herd B, a 600-cow herd with data recorded over 44 months from a total of 1,040 cows where treatment of clinical cases was used to identify lameness events. The PAF for categories of BCS were estimated using a closed equation appropriate for multiple exposure categories. Simulation models were used to explore theoretical scenarios to reflect changes in BCS and recurrence of previous lameness events in each herd.

For Herd A, 21.5% of the total risk periods (cow-weeks) contained a lameness event (LS 3, 4 or 5), 96% of which were repeat events and 19% were recorded with BCS < 2 (3-weeks previously; 0 to 5 scale). When lameness events were based on two consecutive weeks of LS 4 or 5, 4% of risk periods were recorded as lame, of which 89.5% were repeat events. For Herd B, 16.3% of the total risk periods (consecutive 30-days) contained a lameness event (72.6% were repeat events) and 20% were recorded with BCS ≤ 2 (0 to 120 days previously). The median PAF for all previous lameness was between 79 and 83% in the two herds. Between 9 and 21% of lameness events could be attributed to previous lameness occurring >

16 weeks before a risk period. The median PAF estimated for changes in BCS were in the region of 4 to 11%, depending on severity of lameness.

Repeated bouts of lameness made a very large contribution to the total number of lameness events. This could either be because certain cows are initially susceptible and remain susceptible, due to the increased risk associated with previous lameness events, or due to interactions with environmental factors. This area requires further research.

INTRODUCTION

Numerous risk factors for lameness in dairy cattle have been reported in the literature, including risk factors related to the external environment such as flooring surfaces and time spent standing (Galindo and Broom, 2000, Bergsten et al., 2015) as well as animal-based factors which might impact on structure and function of the claw such as milk yield, body condition score and previous lameness events (Green et al., 2014, Randall et al., 2015). Low body condition score (BCS) and previous lameness are both risk factors for lameness that occur repeatedly over time and have been highlighted as important for lameness control (Hirst et al., 2002, Bicalho et al., 2009, Green et al., 2014, Randall et al., 2015, Randall et al., 2016). Randall et al. (2015) showed that relatively low body condition precedes and is associated with an increased risk of a first lameness event in a cow's life. Consequently, management strategies to maintain appropriate body condition scores may provide an opportunity for the dairy industry to reduce lameness in herds. Hirst et al. (2002) demonstrated that dairy heifers with lameness causing claw horn lesions were at greater risk of lameness in subsequent lactations. A recent study suggested that this relationship might be explained by development of new bone, 'exostosis', on the distal phalanx (Newsome et al., 2016). If this is an irreparable anatomical change to the foot it would contribute towards an increased risk of a cow becoming lame again. Odds ratios reported for these two risk factors

indicate that they are highly associated with lameness; for example the OR associated with moving from non-lame to lame state for cows with BCS 1.00 – 1.75 at calving versus 2.50 – 2.75 was 7.73 (2.37 – 17.71) and the OR associated with clinical lameness for cows having been identified lame 31 – 60 days previously versus no previous lameness was 13.80 (10.58 – 17.78) (Green et al., 2014, Lim et al., 2014).

The population attributable fraction (PAF) provides an estimate of the contribution that a risk factor makes to the total disease burden in a population. Knowledge of the PAF of risk factors can facilitate decision-making for farmers and policy makers to maximise disease reduction with existing resources when the knowhow exists, or it can influence funders of research (Steenland and Armstrong, 2006) when knowledge to reduce impact of risk factors is not known.

There are a range of formulas used to calculate PAF and these have different limitations, such as biases arising when adjusted estimates of relative risk are used or when the exposure is across different levels (Rockhill et al., 1998, Benichou, 2001, Steenland and Armstrong, 2006). Where risk factors vary over time, the method used to estimate PAF must account for repeated risk events. In addition, a risk factor can be complex, for example, cows in a herd have a range of body conditions rather than a uniform BCS of e.g. 3 so assessing a change in BCS to reduce the PAF needs to use a continuous scale for BCS. Simulation can be used to estimate PAF to allow for sources of uncertainty, such as uncontrolled confounding, to be incorporated into estimates (Steenland and Armstrong, 2006) as well as allowing for more complex scenarios to be investigated (Hudson et al., 2014).

The aim of this study was to investigate the contribution of previous lameness and BCS to the occurrence of total lameness events in two UK dairy herds. A novel simulation-based approach to estimating PAF was used.

MATERIALS AND METHODS

Study Herds

Data were obtained from two UK dairy herds, where detailed and accurate herd records were available. Study herds and datasets have been described in detail by Randall et al. (2015) and Green et al. (2014). They are summarised here briefly;

Herd A. A total of 724 Holstein Friesian dairy cows managed on the Langhill herd held at the Scotland's Rural College's Crichton Royal research farm, Dumfries, Scotland with data recorded over an 8 year period from 2003 to 2011 (Randall et al., 2015). Cows were managed on a long-term 2 x 2 factorial genetic and feeding system study; select and control genetic lines (Pryce et al., 1999), were divided equally into low-forage (LF) and high-forage (HF) groups and managed as one herd of approximately 200 cows, as described in detail by Chagunda et al. (2009). LF cows were continuously housed whilst HF cows were grazed during the summer grazing period (typically March to November). Cows were milked three times daily and the herd was all-year round calving. Target yields were 13,000 and 7,500 kg per cow per year for LF and HF cows, respectively. Housing was the same for LF and HF cows; cubicles with mattresses and automatically scraped grooved concrete passageways. Regular footbathing was carried out and a professional foot trimmer attended the whole herd twice a year. Locomotion scores (LS) were recorded weekly by trained assessors on a 1 to 5 scale (Manson and Leaver, 1988). Lame cows (LS 4 or 5 on a single occasion or 2 successive scores of LS 3) were treated by a veterinarian on a weekly basis before 2006 and every 2 weeks after this time. Severely lame cows were treated within 24 hours by trained farm staff. BCS was measured weekly using a 0 to 5 scale with increments of 0.25 (Mulvany, 1977). All health, production and management data were recorded in a database.

Herd B. A total of 1,040 Holstein dairy cows on one dairy farm in Somerset, England with data recorded over 44 months between 2008 and 2011 (Green et al., 2014). Cows were

136 milked twice daily in a 60 point rotary parlour and continuously housed all year around, apart
137 from summer when grazed during the last 2 months of lactation. Rations were formulated
138 with the aim of maximising yield whilst minimising feed costs and fed to milking cow groups
139 (early, mid and late lactation) accordingly. Biotin was added at 20mg/cow/day. Housing was
140 modern free stall accommodation with water mattresses in cubicles and solid concrete
141 passageways with automatic scrapers. Mean yearly yield was approximately 10,000 kg per
142 cow per annum. A professional foot trimmer attended the herd each month; typically cows at
143 the end of lactation and with mis-shapen feet were trimmed, with a minimum routine foot
144 trim once per year. Daily observations of the herd by senior herdsmen identified lame cows
145 which were treated under veterinary direction using standard protocols, generally within 2 to
146 3 days. Body condition score was recorded at 60 day intervals throughout the study period,
147 by the head herdsman with appropriate training to prevent drift in scoring, on a scale of 0 to 5
148 in 0.5 increments (based on examination of the transverse processes of the lumbar vertebrae,
149 the ribs, ischial tuberosity, ligaments of the pelvis and surrounding fat (Green et al., 2014)).
150 Health, production, BCS and lameness treatments were recorded in Interherd (National Milk
151 Records).
152
153 **Statistical Analysis**
154 To account for the longitudinal nature of the data, risk factors where events varied at
155 repeated measurements were lagged (e.g. BCS, previous lameness and milk yield) and frailty
156 models were constructed to take into account repeated measures of the outcome (lameness
157 events). The main difference between Herd A and Herd B was in defining lameness events;
158 Herd A was based on weekly locomotion scoring whilst Herd B was based on treatment for
159 lameness from the farmer's records. For Herd A two separate definitions for a lameness event
160 were investigated; these were a) one-week with LS 3, 4 or 5 (less severe lameness) and b)

two consecutive weeks with LS 4 or 5 (more severe lameness). There were three stages to estimating the PAFs; (i) constructing models to estimate adjusted relative risks (RR) for BCS and previous lameness, (ii) estimating PAF for BCS categories to compare estimates using closed equation and simulation approaches and (iii) using simulation to estimate PAF for changes in BCS and occurrence of previous lameness within the two herds to quantify the contribution of these risk factors to total lameness in each herd.

The annual incidence rate of lameness was calculated as [number of new lameness events divided by number of cow-weeks at risk] multiplied by 52 for Herd A and [number of new lameness events divided by number of cow-months at risk] multiplied by 12 for Herd B. For Herd A The weekly incidence rates over the study period were calculated as the number of new lameness events divided by the number of cows eligible (i.e. those cows not lame in the previous risk period) and prevalence was calculated as number of lameness events divided by number of observations.

Stage 1: General approach to modelling; estimating coefficients for previous lameness and body condition score

Data handling and model construction are described in detail by Randall et al. (2015) for Herd A and Green et al. (2014) for Herd B.

Binary outcomes investigated for Herd A were LS 3, 4 or 5 in one week (Model 1a) and LS 4 or 5 over two consecutive weeks (Model 1b). The model outcome in Herd B was also binary; yes / no for treatment of lameness (all causes included; sole haemorrhage (SH), sole ulcer (SU)/white line disease (WLD) and digital dermatitis (DD)) (Model 2). Mixed effects logistic regression models were constructed in MLWin 2.28 (Rabash et al., 2009). Where possible, missing observations were included as a categorical variable and fitted within the models to minimise loss of data. Initial parameter estimation for model parameters

was carried out by iterative generalized least square procedures (Goldstein, 2003) and using forward selection of explanatory variables; explanatory variables were left in the model if the 95% credible interval of the odds ratio did not include unity. Final parameter estimates were made using Markov chain Monte Carlo (MCMC) to reduce biased estimates (Rabash et al 2009), using procedures previously described by Green et al. (2004). A burn-in of 1,000 iterations was used, with final parameter estimates being based on a minimum further 9,000 iterations. Chain mixing and stability were assessed visually.

Models took the form;

$Lame_{ij} \sim$ Bernoulli (probability = π_{ij})

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

$$[u_j] \sim N(0, \sigma^2_v)$$

Where subscripts *i* and *j* denote the *i*th observation of the *j*th cow respectively. π_{ij} = probability of a lame outcome for the *i*th observation of the *j*th cow. α = intercept value, β_1 = vector of coefficients for X_{ij} (Herd A included logarithm of the week of the study up to the power 3), X_{ij} = vector of covariates associated with each observation, β_2 = coefficients for covariates X_j, X_j = vector of covariates associated with each cow, u_j = random effect to account for residual variation between cows (assumed to be normally distributed with mean = 0 and variance = σ^2_v) and residual error.

Explanatory variables included in the models for Herd A were; weeks in milk, week of the study, parity (categorical 1 to 4 +), age at first calving (categorical < 24, 24 to 27, 28 to 30, 31 to 33 and greater than 33 months), BCS change 0 to 4 weeks post-calving (categorised as 0 = loss, 1 = no change, 2 = gain), body weight (categorical < 550, 550 to 700 and > 700 kg), assessor of locomotion and body condition, feed – genetic group and milk yield 16 weeks previously (average daily kg per week; categorical < 12, 12 to 24, 25 to 37, 38 to 50 and > 50 kg). Variables of interest were time since previous lameness (categorised in 4 week

intervals from time t to > 16 weeks), and BCS (categorical < 2, 2, 2.25, 2.5, 2.75, 3 and > 3). Explanatory variables included in the model for Herd B were; parity (categorical 1 – 6 +), year quarter, month in herd, days in milk (at the end of a 30-day period), milk yield (kg per day) measured at the most recent monthly milk recording, yield lagged by one month. Variables of interest were time since previous lameness event (data were available from 2002, categorised in 30 day intervals from time t to >120 days) and BCS >2 lagged by 0 to 2 months and 2 to 4 months.

Posterior predictions were used to assess model fit by visual comparison to the observed data (Gelman et al., 1996). Standardized residuals at the cow level (level 2) were also assessed for normality (Rabash et al., 2009). The Hosmer-Lemeshow test (Hosmer and Lemeshow, 1989) was used as a statistical test for goodness-of-fit. Cow level residuals were found to be over-dispersed and non-normal for Models 1a and 1b, therefore random effects were removed; this improved model fit such that it was very good without random effects and were used as the final models.

Descriptive statistics and results from modelling

Herd A: Of the 724 cows ever in Herd A, 674 (93.0%) had at least one week with LS 3, 4 or 5 and 375 (51.8%) had at least one lameness event with LS 4 or 5 for two consecutive weeks. There were a total of 79,565 and 78,698 cow weeks at risk in Models 1a and 1b, respectively. The number of lameness events were 17,114 and 3,572 respectively for Models 1a and 1b. The annual incidence rate of lameness was 7.4 cases per cow-year when a lameness event was one week LS 3, 4 or 5 and 0.7 cases per cow-year when a lameness event was two consecutive weeks LS 4 or 5. The weekly incidence rates over the study period are shown in Figure 1 for Models 1a (one-week LS 3, 4 or 5) and 1b (two consecutive weeks LS 4 or 5), respectively. Figure 1 also shows the prevalence for each week of the study period for

Models 1a (one-week with LS 3, 4 or 5) and 1b (two consecutive weeks LS 4 or 5), respectively. Both weekly incidence rates and prevalence of LS 3, 4 or 5 increased during the second half of the study period for Herd A. Figure 2 shows the frequency distribution for number of consecutive weeks with LS 3, 4 or 5 and LS 4 or 5, respectively, demonstrating that the majority of lameness events had a duration of one week. The median BCS was 2.25 (range, 0.75 to 4.25) for Herd A. The proportion of the cow-week risk periods exposed to BCS categories < 2, 2, 2.25 and 3 were 0.19, 0.23 (0.24 for Model 1b), 0.26 and 0.05, respectively for Models 1a and 1b. The proportion of observations where there was a previous lameness event in the 1 to 4 weeks prior was 0.4; 5 to 8 weeks was 0.38; 9 to 12 weeks was 0.36; 13 to 16 weeks was 0.34; and > 16 weeks was 0.73 for Model 1a. For Model 1b the proportion of observations where there was a previous lameness event in the 1 to 4 weeks prior was 0.079; 5 to 8 weeks was 0.074; 9 to 12 weeks was 0.070; 13 to 16 weeks was 0.067; and > 16 weeks was 0.29. Odds ratios and 95% credible intervals from Models 1a and 1b for BCS and previous lameness are reported in Table 1. For all other covariates included in the final model, parameter values and significance were similar to those previously reported (Randall et al., 2015). Assessment of model fit was considered good. For Model 1a, BCS 3-weeks previously was positively associated with the lameness outcome LS ≥ 3. BCS = 3, 3-weeks previously had the lowest odds ratio i.e. the lowest risk of lameness and therefore was used as the baseline category for simulations described below in Stage 2. BCS < 2 had the highest odds ratio (OR (95% credible interval) = 1.29 (1.15 to 1.45)) compared with the baseline category. Previous lameness variables were also significant; lameness in the previous 1 to 4 weeks compared with no previous lameness had the highest odds ratio (OR (95% credible interval) = 3.65 (3.48 to 3.83)). For Model 1b, BCS one week previously had the largest effect size and therefore was left in the final model. As for Model 1a, BCS = 3 had the lowest odds ratio and was used as the baseline category for simulations in Stage 2. BCS <

2 had the highest odds ratio compared with the baseline category BCS = 3 (OR (95% credible interval) = 1.66 (1.27 to 2.16)). Previous lameness variables were also associated with a significant risk in lameness; lameness in the previous 1 to 4 weeks had the highest odds ratio (OR (95% credible interval) = 18.72 (16.97 to 20.66)) compared with no previous lameness.

Herd B: A total of 14,530 risk periods were obtained from 1,040 cows from Herd B and the mean number of observations was 10 (range 1–36) per cow. The annual incidence rate for the study period was 1.4 cases per cow-year. 14,461 body condition scores were included in the data set; the median BCS was 2.5 (range, 1 to 5). In total, 647 cows were treated for lameness; the proportion of observations where there was exposure to previous lameness 1 to 30 days ago, 31 to 60 days ago, 61 to 90 days ago, 91 to 120 days ago and greater than 120 days ago were 0.21, 0.10, 0.05, 0.04, 0.17 respectively. Of the 1,040 cows 62.2% were ever lame during the study. Odds ratios and 95% credible intervals from Model 2 for the explanatory variables of interest (BCS and previous lameness) are reported in Table 1. For all other covariates included in the final model, parameter values and significance have previously been reported (Green et al., 2014). For Model 2, BCS > 2 in the last 0 to 2 months or 2 to 4 months was associated with a decreased risk of lameness (all causes; SU, SH/WLD and DD) compared with BCS ≤ 2; OR (95% credible interval) = 0.63 (0.55 to 0.73) and 0.74 (0.60 to 0.90) respectively. All previous lameness categories were associated with an increased risk of lameness compared with no previous lameness; previously lame 1 to 30 days ago had the highest odds ratio (OR (credible interval) = 19.69 (15.70 to 24.69).

Stage 2: Comparing closed and simulation-based approaches to estimating PAF. Exposure to BCS categories for each of the herds' data was used to estimate PAF using closed formula and simulation.

A formula for multiple exposure categories described by Hanley (2001) was used for the closed method:

$$PAF = \frac{P_1\{RR_1 - 1\} + P_2\{RR_2 - 1\}}{1 + P_1\{RR_1 - 1\} + P_2\{RR_2 - 1\}}$$

Where,

PAF = population attributable fraction

P = prevalence of exposure

RR = relative risk (calculated from the coefficients estimated for each BCS category from Models 1a, 1b and 2).

The simulation approach used posterior predictions of the number of lameness events to estimate PAF (Gelman, 2000). Models 1a, 1b and 2 were imported into OpenBUGS version 3.2.3 (Lunn et al., 2009) alongside raw data from the respective herds. Coefficients were estimated from the models using Markov chain Monte Carlo (MCMC) and a burn-in of 4,000 iterations and a further 6,000 iterations for final parameter estimates based on visual inspection of chain mixing and stability. The number of lameness events were predicted from Models 1a, 1b and 2 for the herd raw data (baseline exposure) and with exposure to each of the BCS categories sequentially removed (i.e. coefficients equal to zero). The posterior prediction for PAF was calculated as the difference in number of lameness events with and without exposure to each BCS category present as a proportion of the total number of lameness events occurring in the herd. PAF are reported only for the BCS categories that had a significant association with the outcome (lameness events).

Stage 3. Estimating PAF for BCS and previous lameness. Simulation was used to explore more complex scenarios by quantifying the contribution that BCS and previous lameness made towards the total number of lameness events within each herd. Scenarios

307 explored are summarised in Table 2. Models 1a, 1b and 2 were imported into OpenBUGS
308 alongside raw data from respective herds. For the BCS scenarios, additional categories were
309 created for 0.5 added to the BCS score for each cow with BCS < 3 for each week in the herd
310 (i.e. 0.5 BCS gain across the whole herd apart from cows with BCS 3 or above) and 0.5 taken
311 away from each BCS score (i.e. 0.5 BCS loss across the whole herd) in the Herd A dataset;
312 data were imported to OpenBUGS. Exposure distributions for the BCS categories are
313 summarised in Table 3. Coefficients were estimated from the models using Markov chain
314 Monte Carlo (MCMC) and a burn-in of 4,000 iterations and a further 6,000 iterations for final
315 parameter estimates, based on visual inspection of chain mixing and stability. The number of
316 lameness events for the herd exposed to each of the scenarios (0.5 BCS gain and 0.5 BCS
317 loss) and not exposed to these distributions (i.e. the BCS distribution of the raw herd data as a
318 baseline) were predicted. Posterior predictions for PAF were calculated as the difference in
319 number of lameness events as a proportion of the total number of lameness events. For the
320 previous lameness scenario, the raw herd data was used as the baseline scenario, with
321 exposure to previous lameness removed for the altered scenario (i.e. all coefficients for
322 previous lameness categories equal to zero). The number of lameness events for the herd
323 exposed and unexposed to previous lameness events were predicted. Posterior predictions for
324 PAF were calculated as the difference in the number of lameness events as a proportion of the
325 total number of lameness events.

326 To remove the effect of lameness that occurred just prior to a risk period and explore
327 only the impact of lameness events that occurred earlier, Models 5.1a, 5.1b and 5.2 were used
328 to estimate PAF of lameness events that occurred a minimum of 5 weeks before a current
329 case and a minimum of 16 weeks before a current case. For models 1a and 1b the effect of
330 previous lameness events that occurred in the 4 to 8, 9 to 12, 13 to 16 and > 16 weeks
331 previously were investigated. As these were separate covariates in the model, to investigate

their effect, the relevant coefficients were set to equal zero, where the baseline was no previous lameness in that time period. For model 2, the effect of previous lameness events that occurred in the previous 31 to 60, 61 to 90, 90 to 120 and > 120 days were investigated. These were included as categories for the explanatory variable previous lameness where the baseline was none. Coefficients for the weeks being investigated were set to equal zero. The analyses were repeated as described above.

RESULTS

Comparison of closed and simulation approach for estimating population attributable fractions

The PAF estimated using both closed and simulation methods are presented in Table 4.

Model 1a. Using closed calculation methods, PAF for exposure to each BCS category were; 4.49% for BCS < 2 three weeks previously, 2.66% for BCS 2 and 2.38% for BCS 2.25 (total = 10.61%). The median (95% credible interval) PAF predicted using simulation were 3.10 % (1.71 – 4.54), 1.73% (0.14 – 3.33), 1.50% (-1.31 – 3.13) for BCS < 2, 2 and 2.25 respectively.

Model 1b. The PAF for exposure to BCS categories calculated using the closed method were; 8.90% for BCS < 2 one week previously, 6.00% for BCS = 2 and 4.68% for BCS = 2.25 (total = 19.57%). Using simulation, the median (95% credible interval) predicted PAF for BCS categories < 2, 2 and 2.25 were 7.64% (2.81 – 11.23%), 5.58% (1.05 – 9.18%) and 3.93% (-0.76 – 7.74%), respectively.

Model 2. The PAF for BCS categories calculated using closed method were 9.83 % for BCS < 2 in the 1 to 60 days previously and 5.92 % for BCS < 2 in the 61 to 120 days previously. Median PAF (95% credible interval) predicted using simulation was 7.49% (4.03

– 10.78) and 4.28% (0.64 – 7.72%) for BCS < 2 in the 1 to 60 days previously and 61 to 120 days previously, respectively.

Estimating PAF for BCS and previous lameness.

Results of the scenarios investigated are presented in Table 5.

Body condition score. A gain in BCS of 0.5, in cows BCS 3 or less, across the whole herd for the 8 years of data available for Herd A resulted in a reduction of 600 predicted lameness events, where the outcome was LS 3, 4 or 5 (Model 1a). The median PAF (95 % credible interval) for this change in exposure was -3.54 % (-5.86 – -1.28%) i.e. 3.54% of lameness events in the herd may be avoidable if all cows with BCS < 3 in the 3 weeks previously were exposed to a 0.5 gain in BCS. When the lameness severity threshold was 2 consecutive weeks LS 4 or 5 (Model 1b) there was a greater reduction in lameness events with a median PAF of -8.06% (-13.12 - -2.22%). A loss in 0.5 BCS across all BCS score categories for Model 1a resulted in an additional 1030 predicted lameness events and the median PAF (95% credible interval) for this exposure was 5.99% (3.36 – 8.74%) i.e. 5.99% of lameness in the herd may be avoidable by not exposing the herd to a loss in BCS of 0.5. The median PAF (95%) for this exposure using Model 1b, where lameness severity threshold was increased, was 11.2% (5.52 - 17.33%).

Previous Lameness. When the effect of exposure to all previous lameness events was removed across the whole herd the predicted number of lameness observations was reduced by 7576 observations for Herd A where the outcome was LS 3, 4 or 5 (Model 1a) and 2812 observations where outcome was LS 4 or 5 on 2 consecutive weeks (Model 1b). Of the predicted lameness events, 80.69% (79.01 – 82.26%) and 78.75% (76.40 – 80.98%) were attributable to exposure to previous lameness events over the study period for these two outcomes in Herd A (Model 1a and 1b), respectively. When the effect of exposure to

previous lameness was removed across the whole herd in Herd B (Model 2) the predicted number of lameness events was reduced by 1652 events; 82.69% (79.28 – 85.61%) of lameness treatments were attributable to previous lameness over the study period in Herd B.

When PAF was estimated for lameness events that occurred at least 5 weeks previously, the median (95% credible interval) PAF were 58.97% (56.11 - 61.67%), 41.67% (36.90 to 46.19%) and 46.31% (42.08 to 50.14%), respectively for Models 1a, 1b and 2.

When PAF was estimated for lameness events that occurred at least 16 weeks previously, the median (95% credible interval) PAF were 9.34% (5.14 – 13.58%), 11.36% (5.49 to 17.09%) and 21.07% (16.30 to 25.50%), respectively for Models 1a, 1b and 2.

DISCUSSION

Previous Lameness Events

This is the first study to quantify the PAF of previous lameness events in cattle on herd level lameness. Estimates of PAF for the two herds suggested that between 79% and 83% of lameness was attributable to exposure to previous lameness events (regardless of when they occurred), indicating that this is an important risk factor. When the effect of lameness events that occur > 4 weeks and > 16 weeks previously were investigated, the contribution from previous lameness reduced markedly, although it was still considerable. This finding suggests that lameness might last for some duration (as shown in Figure 2) or that cows can take a considerable amount of time to recover, but that some do fully recover. It appears from these results that a large proportion of the total lameness events in these herds are accounted for by an accumulation of repeat cases. Across the two herds between 52 and 93% of cows were ever lame during their respective study periods, indicating that significant resources are going into treating a large number of lameness cases.

The challenge therefore is to understand why repeat cases are occurring and how to prevent them. The number of repeat lameness events could be influenced by the duration of time individual animals spend within the herd and therefore if cows are not culled for being lame they may experience a higher number of repeat lameness events. It is also possible that there are some other environmental or animal-based factors that could explain a high number of repeat lameness events in certain cows. For example, there may be an interaction between previous lameness and the environment that influences whether cows will go on to have repeated lameness events. It may also be important to prevent the occurrence of the first lifetime lameness event, although based on this analysis it is not possible to know whether it was the first lifetime lameness event or some other environmental or animal-based interaction which is important in consigning a cow to repeat lameness events. In addition, findings from this study highlight that early and effective treatment of lameness reducing the likelihood of recurrence or cases becoming chronic (Thomas et al., 2015) may also be crucial to lameness control at a herd level.

It is widely reported that lameness events increase the risk of future lameness events occurring (Hirst et al., 2002, Green et al., 2014, Randall et al., 2015). Hirst et al. (2002) investigated the relationship between lameness in heifers and the association with future risk, reporting a positive association between claw horn lesions and future risk. These findings were similar to those reported by Randall et al. (2016); more severe claw horn disruption lesions occurring around the time of first calving were associated with a long-term increased risk of lameness. One hypothesis for this association is that underlying pathology carries over from one case to the next making future cases more likely. The increase in lameness prevalence or risk with increasing parity that is widely reported would support this hypothesis (Barker et al., 2009, Randall et al., 2015, Solano et al., 2015). In addition, Newsome et al. (2016) demonstrated that bone development on the caudal aspect of the distal phalanx at

slaughter were positively associated with claw horn lesions during life, providing evidence for underlying pathology being associated with previous lameness. An additional element to the hypothesis explaining the association between previous and future lameness and increased lameness risk with increasing parity is that hypersensitivity and reduction in pressure pain thresholds may develop as a result of long term pain associated with lameness. Although poorly understood, it is widely reported in the medical literature that disease can lead to long term changes in the nociceptive nervous system leading to allodynia (pain associated with non-noxious stimuli) and hyperalgesia (noxious stimuli causing pain of longer duration and higher intensity than normal) (Nielsen and Henriksson, 2007, Latremoliere and Woolf, 2009, Woolf, 2011). Laven et al. (2008) demonstrated that there is a long duration of allodynia associated with lameness even after treatment, highlighting the importance of lameness prevention. When the high prevalence of lesions in heifers reported by Maxwell et al. (2015) and Capion et al. (2009) is considered, this becomes even more significant.

The findings from this study highlight the importance of previous lameness events as a risk factor for lameness and therefore the urgent need for further research to identify how to prevent the occurrence of repeat lameness events.

Body Condition Score

The results of this study demonstrated the impact of changing BCS across the whole herd; 4% of all lameness events (one-week with LS 3, 4 or 5) could potentially be avoidable with exposure to a 0.5 increase in BCS in all cows with BCS < 3, whilst 8% of all lameness events may be preventable by avoiding exposure to a loss of 0.5 BCS. These figures increased to 6% and 11%, respectively, when the lameness severity threshold was increased. Previous studies have demonstrated that BCS is a risk factor for lameness in all ages of dairy

cattle (Hoedemaker et al., 2009, Green et al., 2014, Lim et al., 2014). Randall et al. (2015) found that cows with BCS < 2 in the previous 3 weeks were at greatest risk of lameness in a longitudinal study using the same dataset from Herd A as in the current study. Similarly, Green et al. (2014) has shown that cows with BCS ≤ 2 were more likely to be treated for lameness in the following 2 and 2 to 4 months compared with cows BCS > 2, using the same dataset from Herd B as in the current study. However, this is the first study to evaluate the importance of BCS changes at a herd level in terms of its impact on the total amount of lameness in a dairy herd using simulation that accounts for variability. This is an important step forward from identifying BCS as a risk factor for lameness towards quantifying the effect that this risk factor has on the proportion of lameness events in herds that could be prevented if BCS was altered. Alawneh et al. (2014) calculated PAF for liveweight using closed equations and demonstrated that the population level impact of a decrease in liveweight over the first 50 days in milk was relatively small; a 3% (95% confidence interval = 1 – 6%) reduction in the incidence risk of lameness was reported if excessive liveweight loss was prevented. The impacts of BCS reported for each of the scenarios investigated in this study are similarly relatively small compared with the impact of previous lameness events. Although, in herds with fewer repeated lameness events, BCS relatively could be more important.

Comparison of closed and simulation-based approaches for estimating population attributable fractions

Formulas for calculating population attributable risk or fractions have been derived for different epidemiologic designs, including situations where there are more than one exposure level or where confounding factors exist (Benichou, 2001). However there are limitations in the use of these formulas when applied to more complex scenarios that are

often present in real-life situations which mean they are not directly useable in application. Simulation can be useful in addressing these issues by modelling dynamic interactions between individual animals or groups of animals whilst taking into account factors that may vary within and across levels of influence. Galea et al. (2010) used obesity as an example to demonstrate how traditional analytical approaches, which focus on the isolation of single disease states and causes, has been challenged by the recognition of dynamic and complex interactions of factors influencing disease outcomes. Complex systems dynamic models can offer an alternative approach. Simulation models parameterized using observations from epidemiological data can be used to investigate inputs and outputs of a complex system and therefore become useful as a tool to test different scenarios. The use of simulation for estimating PAF where data has repeated measures is a novel approach. Therefore estimates using a closed equation method were compared to those using simulation. In this study the formula applicable for multiple exposure levels was used to calculate the PAF for BCS categories using data from two herds. Results using this closed method were compared to the results generated from posterior predictions. Simulation methods estimated PAF values that were within the 95% credible interval for PAF estimated using closed methods. These results illustrate that simulation based approaches produce similar, although slightly more conservative, estimates of PAF. As simulation methods account for the variability and can propagate this through the model to be included in the posterior predictions, the simulation-based results may be the more realistic figure for PAF.

Study Limitations and Generalisability

The main findings of this study were demonstrated in two UK herds with different methods of lameness detection. The PAF of comparable scenarios were similar in both herds giving an indication for possible generalisability of these findings to herds with similar

management systems. Although it should be recognised that the PAF estimates reported here are only applicable to changes in the original exposure distribution in these herds i.e. in herds with a higher median BCS compared with these study herds, the PAF for changes in BCS may differ to that reported in this study. The mean prevalence of lameness over the study period in Herd A for LS 3, 4 or 5 was 21.3%, which is lower than prevalence rates reported in other UK studies (Archer et al., 2010, Barker et al., 2010).

This study only investigated the population level impacts of the risk factors body condition score and previous lameness. The impact of other risk factors, including environmental risk factors should also be quantified to understand how these effect lameness at a herd level compared to the risk factors explored in this study.

CONCLUSIONS

This study quantified the impacts of the risk factors BCS and the occurrence of previous lameness events on herd level lameness. A loss in BCS of 0.5 across the herd was estimated to contribute towards 6% of the total number of lameness events (one-week with LS 3, 4 or 5), indicating that this proportion of total lameness could potentially be avoidable in the herds investigated. When the lameness severity threshold was increased (2 consecutive weeks LS 4 or 5) this figure increased to 11%. By comparison, between 79% and 83% of lameness events were estimated to be attributable to exposure to all previous lameness events and between 9% and 21% attributable to exposure to lameness events that occurred at least 16 weeks previously. These findings suggest that repeated lameness events (i.e. an accumulation of previous lameness events) contributes towards an over-whelming proportion of the total amount of lameness in the herds investigated. Interactions with environmental or animal-based factors may be important for influencing whether animals go on to have repeated lameness events. Preventing the first case of lameness could potentially be important

532 in avoiding an escalation of repeated lameness events. A novel approach to estimating PAF
533 using simulation enabled complex scenarios to be investigated whilst accounting for
534 variability within the herds in this study using longitudinal data with repeated measures.

535

536 **ACKNOWLEDGEMENTS**

537 This work was supported by an Industrial CASE studentship. Funding from the
538 Biotechnology and Biological Sciences Research Council (BBSRC) and Boehringer
539 Ingelheim is gratefully acknowledged. We also acknowledge staff at the SRUC Dairy
540 Research and Innovation Centre for access to and collection of the data.

541

542 **Conflicts of interest**: none identified

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647 Table 1. Results of Model 1a, 1b and 2 for explanatory variables body condition score (BCS) and
648 previous lameness using data obtained from the Scotland's Rural College (SRUC) Research and
649 Innovation Centre dairy herd (Model 1a and 1b) and a 600-cow herd in Somerset, UK (Model 2)*

Variable	N[1]	Odds ratio	Lower 95% CrI[2]	Upper 95% CrI
Model 1a; outcome = one-week with LS 3, 4 or 5 Total N = 79565				
BCS 3 wk previously				
3	3612	Baseline		
<2	14762	1.29	1.15	1.45
2	18603	1.14	1.02	1.27
2.25	20711	1.11	1.00	1.23
2.5	11444	1.07	0.96	1.19
2.75	4385	1.03	0.91	1.16
>3	2046	1.05	0.91	1.22
Previous lameness (LS 3,4 or 5)				
None	38133	Baseline		
1 to 4 wk	31483	3.65	3.48	3.83
None	3672	Baseline		
5 to 8 wk	30041	2.15	2.05	2.27
None	35636	Baseline		
9 to 12 wk	28687	1.64	1.53	1.77
None	34547	Baseline		
13 to 16 wk	27373	1.52	1.44	1.59
None	12218	Baseline		
>16 wk	57690	1.21	1.12	1.31
Model 1b; outcome = 2 consecutive weeks with LS 4 or 5 Total N = 78698				
BCS 1 wk previously				
3	3718	Baseline		
<2	15122	1.66	1.27	2.16
2	18910	1.44	1.11	1.87
2.25	20990	1.29	1.00	1.66
2.5	11632	1.06	0.82	1.37
2.75	4481	1.11	0.83	1.48
>3	2119	1.16	0.84	1.61
Previous lameness (2 consecutive LS 4 or 5)				
None	67770	Baseline		
1 to 4 wk	6181	18.72	16.97	20.66
None	65262	Baseline		
5 to 8 wk	5812	1.99	1.78	2.22
None	62901	Baseline		
9 to 12 wk	5517	1.51	1.34	1.69
None	60682	Baseline		
13 to 16 wk	5245	1.48	1.32	1.67
None	46587	Baseline		
>16 wk	23064	1.62	1.46	1.79
Model 2; outcome = all causes of lameness (SH, SU/WLD and DD)[3]				
BCS				
BCS > 2 last 0 to 2 m		0.63	0.55	0.73
BCS > 2 last 2 to 4 m		0.74	0.60	0.90

Previous lameness			
None	Baseline		
1 to 30 days ago	19.69	15.70	24.69
31 to 60 days ago	13.75	10.72	17.64
61 to 90 days ago	14.51	10.76	19.58
91 to 120 days ago	13.99	10.08	19.40
>120 days ago	16.02	12.50	20.53

650 [1]N = Number of observations
651 [2]CrI = credible interval
652 * Only coefficients for explanatory variables BCS and previous lameness are reported here. Other
653 covariates tested were found to be significant as reported by Randall et al. (2015) and Green et al.
654 (2014)
655 [3]SH, SU/WLD and DD = sole haemorrhage, sole ulcer/white line disease and digital dermatitis
656

Table 2. Description of scenarios investigated for two UK dairy herds described by Green et al. (2014) and (Randall et al., 2015).

Herd	Model	Outcome (interval)	Scenario	description
A	1a	LS[1] 3,4 or 5 (weekly)	BCS gain	Whole herd gains 0.5 BCS if < 3
			BCS loss	Whole herd loses 0.5 BCS
			No previous lameness	Effect of all previous lameness events removed
	1b	2 consecutive LS 4 or 5 (weekly)	BCS gain	Whole herd gains 0.5 BCS if < 3
			BCS loss	Whole herd loses 0.5 BCS
			No previous lameness	Effect of all previous lameness events removed
B	2	Clinical lameness; all causes (60 days)	No previous lameness	Effect of all previous lameness events removed

[1]LS = locomotion score

Table 3. Proportion of observation in each body condition score (BCS) category for scenarios investigated for Herd A; 724 cows held at the Scotland's Rural College (SRUC) Research and Innovation Centre. Observations relate to weekly scoring of cows i.e. cow-week risk periods.

BCS categories	Baseline		BCS gain		BCS loss	
	No. observations	Proportion	No. observations	Proportion	No. observations	Proportion
Model 1a; total observations = 79565						
<2	14762	0.19	1323	0.02	54076	0.68
2	18603	0.23	4121	0.05	11444	0.14
2.25	20711	0.26	9318	0.12	4385	0.06
2.5	11444	0.14	18603	0.23	3612	0.05
2.75	4385	0.06	20711	0.26	1321	0.02
3	3612	0.05	15056	0.19	575	0.01
>3	2046	0.03	6431	0.08	150	0.002
Model 1b; total observations = 78698						
<2	15122	0.19	1380	0.02	55022	0.69
2	18910	0.24	4224	0.05	11632	0.15
2.25	20990	0.26	9518	0.12	4481	0.06
2.5	11632	0.15	18910	0.24	3718	0.05
2.75	4481	0.06	20990	0.26	1369	0.02
3	3718	0.05	15350	0.19	587	0.01
>3	2119	0.03	6600	0.08	163	0.002

Table 4. Population attributable fraction (PAF) calculated using closed equations and a simulation-based approach using data recorded from 2 UK dairy herds; 724 cows held at the Scotland's Rural College (SRUC) Research and Innovation Centre over an 8 year period (Model 1a and 1b) and 1,040 cow herd in Somerset, UK over a 44 month period (Model 2)*.

	Closed calculation					Simulation-based approach					
BCS category	N[a]	Proportion of total N	Odds	Relative Risk	PAF (%)	Number of lameness observations			PAF (%)		
						$Median_{baseline}$	$Median_{exp}$	No. Observations attributable to exposure	Median	2.5	97.5
Model 1a											
3[b]	3612	0.05	Baseline								
< 2	14762	0.19	0.07	1.27	4.49	3400	3929	529	3.10	1.71	4.54
2	18603	0.23	0.06	1.13	2.66	3675	3969	297	1.73	0.14	3.33
2.25	20711	0.26	0.06	1.10	2.38	3869	4123	256	1.50	-1.31	3.13
	Total N for herd: 79565										
Model 1b											
3[c]	3718	0.09	Baseline								
< 2	15122	0.19	0.009	1.60	8.90	731	1003	272	7.64	2.81	11.23
2	18910	0.24	0.008	1.38	6.00	704	905	201	5.58	1.05	9.18
2.25	20990	0.26	0.007	1.23	4.68	719	861	142	3.93	-0.76	7.74
	Total N for herd: 78698										
Model 2											
1 – 60 d[d]; BCS > 2	7525	0.52	Baseline								
1 – 60 d; BCS < 2	2935	0.20	0.11	1.47	9.83	428	578	150	7.49	4.03	10.78
61 – 120 d; BCS > 2	2102	0.14	Baseline								
61 – 120 d; BCS < 2	2789	0.19	0.09	1.30	5.92	373	458	85	4.28	0.64	7.72
	Total N for herd: 14530										

* Only results where BCS categories were significant (95% credible intervals for odds ratios did not include 1.00) have been reported in this table.
[a]N: Number of observations (observations relate to; Herd A, weekly risk periods for each cow and Herd B, consecutive 60 day risk periods for each cow)
[b]BCS 3 weeks previous to lameness events, [c]BCS 1 week previous to lameness events, [d]d = days

Table 5. Population attributable fraction for body condition score (BCS) and previous lameness estimated by simulation-based approach using data recorded from 2 UK dairy herds; 724 cows held at the Scotland's Rural College (SRUC) Research and Innovation Centre over an 8 year period (Herd A) and 1,040 cow herd in Somerset over a 44 month period (Herd B).*

Scenario		Number of lameness observations[1]			PAF (%)		
		$Median_{baseline}$[2]	$Median_{exp}$[3]	N_{exp}[4]	Median	2.5%	97.5%
Herd B							
Model 1a	BCS gain	17110	16510	-600	-3.54	-5.86	-1.28
	BCS loss	17110	18140	1030	5.99	3.36	8.74
	No previous lameness	17110	3304	-13806	-80.69	-79.01	-82.26
Model 1b	BCS gain	3571	3282	-289	-8.06	-13.12	-2.22
	BCS loss	3571	3968	397	11.20	5.52	17.33
	No previous lameness	3571	759	-2812	-78.75	-76.40	-80.98
Herd B							
Model 2	No previous lameness	1998	346	-1652	-82.69	-79.28	-85.61

[1]Observations relate to; Herd A, weekly risk periods for each cow and Herd B, consecutive 60 day risk periods for each cow

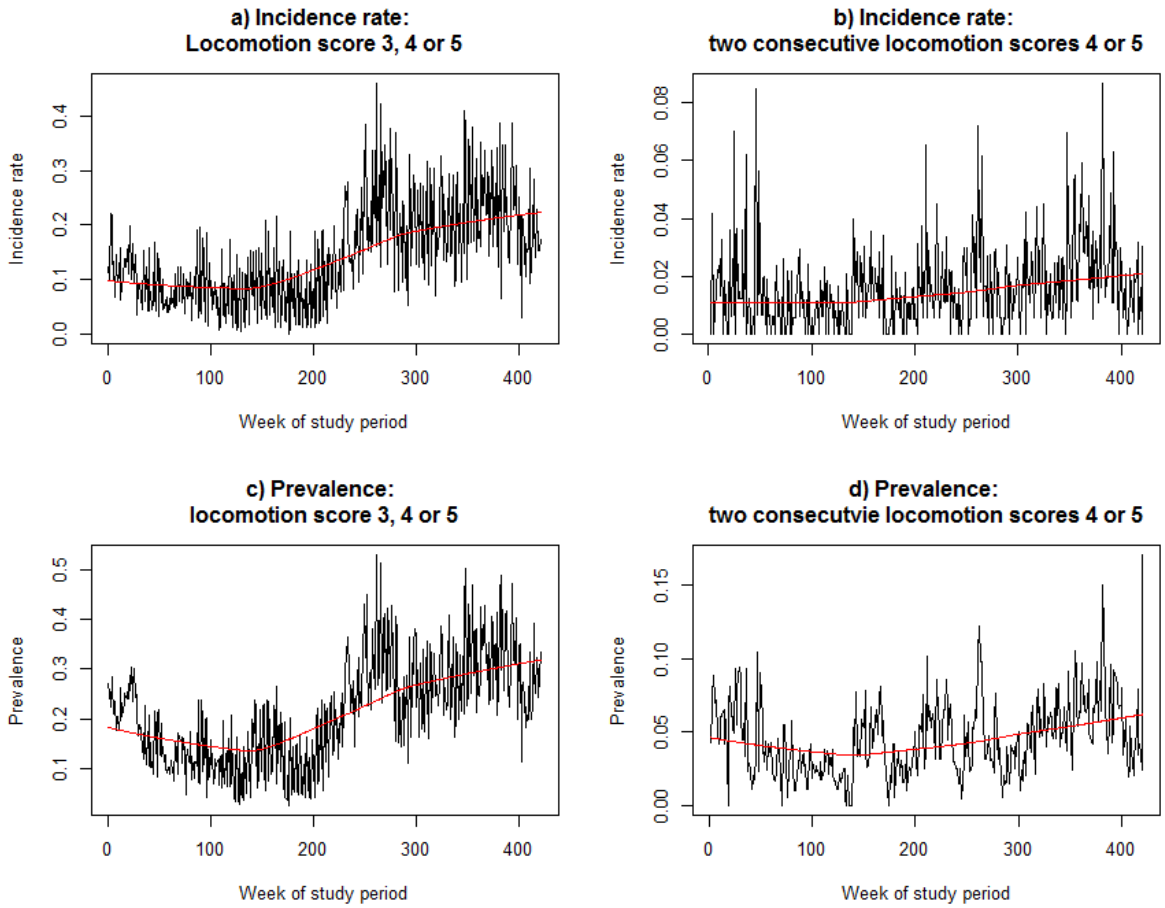
[2]$Median_{baseline}$ = median number of lameness observations for the baseline scenario

[3]$Median_{exp}$ = median number of lameness observations for the exposed scenario

[4]N_{exp} = Number of observations attributable to exposure

*Where the exposure has a protective effect, the PAF is reported as negative e.g. a gain in BCS reduces the risk of lameness (see Table 1) and therefore this exposure will result in less lameness events.

Randall, Figure 1



Randall, Figure 2

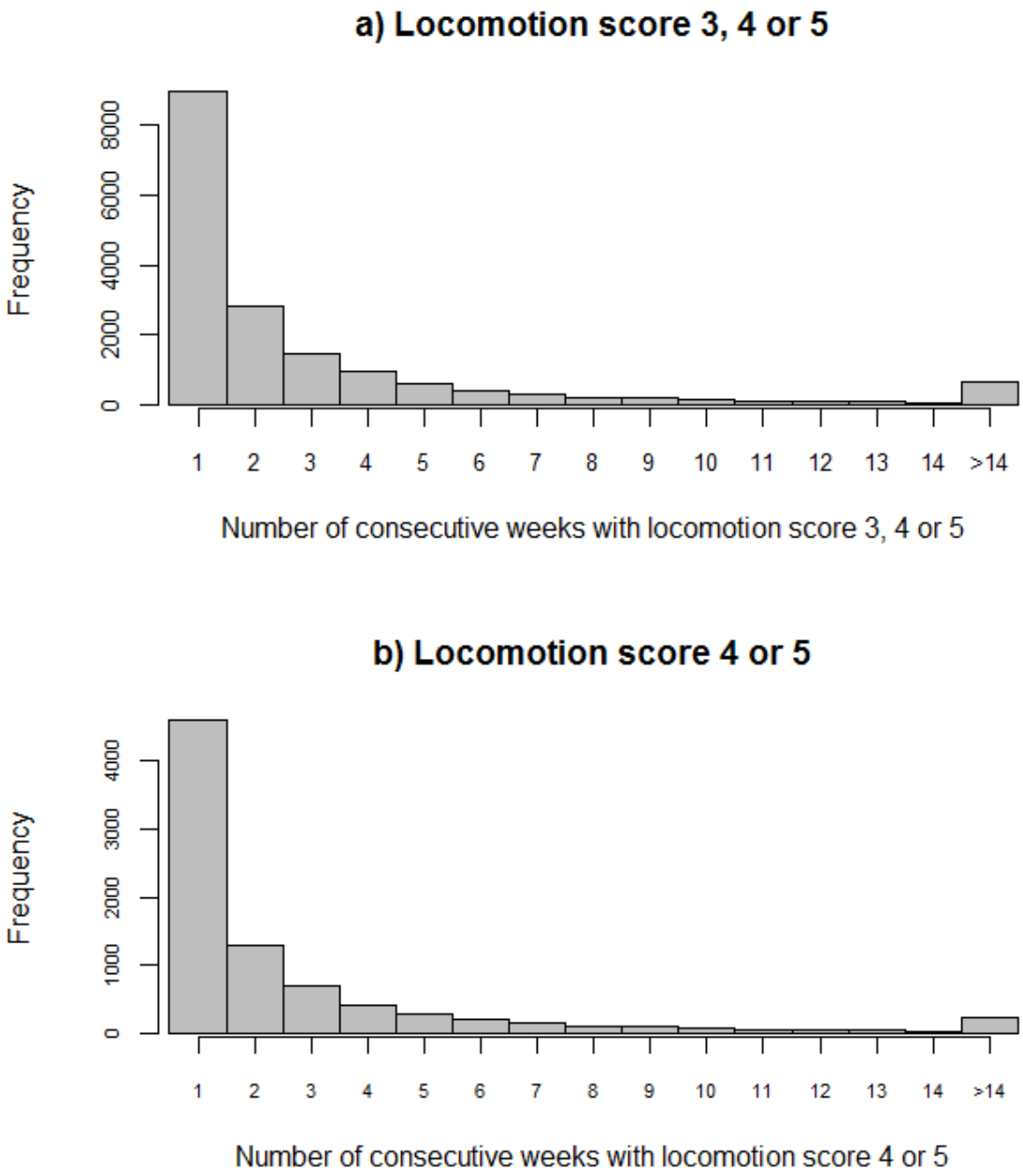


Figure 1. Weekly lameness incidence rate and prevalence over 421 weeks of the study period 2003 to 2011 for Herd A, 724 cows held at the Scotland's Rural College (SRUC) Research and Innovation Centre. The black line shows the weekly incidence rate and the red line shows locally weighted linear regression line created using the lowess function in R (R Core Team, 2016). In (a) and (c) lameness event is defined as locomotion score 3, 4 or 5. In (b) and (d) lameness event is defined as two consecutive weeks of score 4 or 5.

Figure 2. Frequency distributions showing the number of consecutive weeks that cows were locomotion scored as 3, 4 or 5 (a) and 4 or 5 (b) in Herd A, 724 cows held at the Scotland's Rural College (SRUC) Research and Innovation Centre, over the study period 2003 to 2011.