

Evaluation of Body Condition Score and Early Life Events as Risk Factors for Lameness in Dairy Cattle

Laura Vee Randall BVSc MRCVS

Thesis submitted to The University of Nottingham for the degree
of Doctor of Philosophy

June 2017

Abstract

Lameness is one of the most significant challenges faced by the dairy industry worldwide. The negative impacts on the health, welfare and productivity of dairy cows means that controlling lameness is important for future sustainability. The aims of this thesis were to evaluate the risk factors for lameness in dairy cows, in particular body condition score (BCS) and early life events. Claw horn disruption lesions (CHDLs) are one of the major causes of lameness in dairy cattle, however their aetiopathogenesis is still poorly understood. The primary hypotheses explored within this thesis were related to risk factors for CHDLs in particular, with the aim of progressing the current knowledge and understanding of these lesions.

Data obtained over an 8 year period (2003 to 2011) from the Crichton Royal research herd held at the Scotland's Rural College (SRUC) Dairy Research and Innovation Centre in Dumfries, Scotland were used to investigate risk factors for lameness in dairy cows. The associations between fine thresholds of body condition score (independent of body weight) and lameness were explored using mixed effects multinomial and binomial logistic regression models. Cows with BCS < 2 in the previous 3-weeks were at greatest risk of mild or severe lameness. Low BCS was found to be associated with an increased risk of repeated lifetime lameness events in all animals, as well as first lifetime lameness events in cows in their second lactation or greater, indicating that low BCS may be an important risk factor for lameness in herds managed under similar conditions to the study herd. Furthermore, a novel approach to estimating population attributable fractions (PAF) for lameness risk factors which used simulation to investigate complex changes in herd BCS and previous lameness events over time in two dairy herds, showed that a loss in BCS of 0.5 across the whole herd may contribute towards between 4 and 11% of the total number of lameness events in these herds. Despite differences in methods of lameness identification (weekly locomotion scores versus treatment of clinical cases) and the environment between the herds investigated, the estimated PAF were similar, giving some indication that findings may be to some extent generalisable. In comparison to BCS, previous lameness events appeared to have a potentially larger impact on the total number of lameness events in the herd. The median estimated PAF for all previous lameness was between 79 and 83% in the two herds i.e. approximately 80% of lameness events in these two herds may be avoidable by preventing all previous lameness events. Between 9 and 21% of total

lameness events may be attributable to previous lameness events occurring > 16 weeks before a risk period. These findings indicate that repeated lameness events (i.e. accumulation of lameness) may contribute towards a large proportion of herd level lameness. Preventing or increasing the time to first lameness events in a dairy herd may be important to reduce the number of repeat cases and lessen overall burden of lameness. Interactions with environmental and/or animal-based risk factors may also be important for preventing repeat lameness events. Further research is required to identify the reasons for and methods of preventing repeated lameness events, which could have considerable impact on reducing lameness at the herd level. The development of a method for estimating PAF for lameness risk factors using simulation enabled longitudinal data to be analysed, taking into account repeated measures and changes in risk factors over time. This is an important step forward in evaluating risk factors for lameness in dairy cattle, by identifying those that will have greatest impact on controlling disease in the population. Further work is needed to estimate the PAF for other risk factors including environmental factors.

The large effect of previous lameness events on future lameness highlights the potential importance of lameness in heifers, as the future of the dairy herd. Mixed effects logistic regression models and linear regression were used to investigate the impact of hoof lesions in heifers around the time of first calving on milk yield, future lameness and culling risk. Severe claw horn disruption lesions (CHDL) post-calving were associated with an increased risk of future lameness and reduced milk yield (average daily and adjusted for the duration of time that cows spent within the herd), indicating that managing heifers to reduce severe CHDL could have positive benefits on health, welfare and production for the dairy herd. Mild CHDL pre-calving and 2 to 4 months post-calving were found to be associated with a decreased risk of future lameness and decreased culling risk. This novel finding suggests that some degree of mild insult may confer some resilience, if occurring at a time when heifers are able to cope, potentially through adaptive changes in the hoof. This suggests that management of heifers could have major impacts on lifetime claw health.

The impact of previous lameness on herd level lameness was also investigated through exploring how different culling rates effect lameness prevalence, with the use of simulation. As herds with a lower culling rate will have an older age profile this increases the duration of time over which repeat lameness events might occur. Herds adopting a culling rate of 40% versus 20% were found to have a relative reduction in

lameness prevalence of 29.2% (38.2% versus 27.1%, respectively). In herds with lower culling rates particular attention to lameness prevention and control may be necessary to maintain the health, welfare and productivity of the herd; this would be especially true for herds with a high background risk of lameness in month 1 of each lactation.

Multilevel statistical modelling and simulation-based methods, combined with a rich longitudinal dataset, have aided in the identification of important key findings that contribute towards advancing our knowledge and understanding surrounding the aetiopathogenesis of lameness, and in particular CHDL. Identifying risk factors that, if controlled, will have the greatest effect on reducing lameness at the herd level is important and this work highlights where future research can build on this so that progress can be made in controlling lameness. This is relevant and important for the dairy industry worldwide.

Acknowledgements

Having the opportunity to complete this PhD has been a genuine privilege; from the time to work on a project such as this to working with all of the people involved. It has been because of the enthusiasm, support and guidance that I have received from supervisors and colleagues at the School of Veterinary Medicine and Science, University of Nottingham that I have enjoyed this time as much as I have.

I would like to thank my primary supervisors, Professor Jon Huxley and Professor Martin Green for the invaluable support and guidance that you have provided along the way. Thank you also to Dr Mizeck Chagunda, Professor Laura Green and Colin Mason for all of your input throughout. I would also like to recognise the support of all my colleagues who I have had the pleasure of working alongside at the University of Nottingham. I also gratefully acknowledge the funders of this project; Biotechnology and Biosciences Research Council and Boehringer Ingelheim.

Thank you also to my friends and family who have provided unparalleled support over the past 4 years in so many ways, in particular; Tom, Kim, Hannah, Sofie, Anna, Will, Dad, Peachey and Laila. A very special thank you to my mum; I definitely would not have been able to complete this PhD without your support. And last but not least, a big thank you to my beautiful daughter, Elodie.

Abbreviations

BCS	Body condition score
BW	Body weight
C	Control genetic group
CHDL	Claw horn disruption lesions
DD	Digital dermatitis
DCT	Digital cushion thickness
DMI	Dry matter intake
HF	High forage
LS	Locomotion score
LF	Low forage
MCMC	Markov chain Monte Carlo
S	Select genetic group
SE	Standard error
SH	Sole haemorrhage
SL	Sole lesion (sole haemorrhage through to ulceration)
SST	Sole soft tissue thickness
SU	Sole ulcer
TDY	Total daily yield
WLL	White line lesion (white line haemorrhage through to abscessation)
WLD	White line disease

Publications

CHAPTER 3; the work in this chapter has been published in the Journal of Dairy Science.

Randall, L.V., Green, M.J., Chagunda, M.G.G., Mason, C., Green, L.E., Archer S.C., Huxley, J.N (2015) Low body condition predisposed cattle to lameness: an 8 year study of one dairy herd. Journal of Dairy Science. 98(6):3766-77 doi: 10.3168/jds.2014-8863

CHAPTER 4; the work in this chapter has been published in Preventive Veterinary Medicine.

Randall, L.V., Green, M.J., Chagunda, M.G.G., Mason, C., Green, L.E., Huxley, J.N (2016) Lameness in dairy heifers; impacts of hoof lesions present around first calving on future lameness, milk yield and culling risk. Preventive Veterinary Medicine. 1;133:52-63 doi: 10.1016/j.prevetmed.2016.09.006

CHAPTER 5; the work in this chapter has been published in the Journal of Dairy Science.

Randall, L.V., Green, M.J., Chagunda, M.G.G., Mason, C., Green, L.E., Archer S.C., Huxley, J.N (2017) The Contribution of Previous Lameness Events and Body Condition Score to the Occurrence of Lameness in Dairy Herds: A Study of Two Herds. Journal of Dairy Science. 101:1–14. doi: 10.3168/jds.2017-13439

CHAPTER 7 and CHAPTER 1; Parts of the work in these two chapters has been submitted to The Veterinary Journal as an Invited Review.

Randall, L.V, Green, M.J., Huxley, J.N (2017) Invited Review: Use of Statistical Modelling to Investigate the Pathogenesis of Claw Horn Disruption Lesions in Dairy Cattle. The Veterinary Journal. Submitted.

Contents

CHAPTER 1: Introduction and Literature Review	19
1.1 Lameness and the UK dairy industry: An overview of the current situation.....	19
1.2 Significance of lameness in dairy cattle.....	21
1.2.1 Welfare.....	21
1.2.2 Production & economics.....	22
1.3 Prevalence and incidence of lameness in dairy cattle	23
1.3.1 Incidence of lameness	23
1.3.2 Prevalence of lameness	25
1.4 Lameness data; diagnosis & classification of lameness and hoof lesions.....	26
1.4.1 Visual assessment of locomotion.....	28
1.4.2 Other methods of lameness identification.....	29
1.4.3 Infectious causes of lameness	30
1.4.4 Claw horn disruption lesions.....	31
1.5 Aetiology and pathogenesis of claw horn disruption lesions.....	32
1.5.1 Laminitis and the suspensory apparatus.....	33
1.5.2 White line lesions.....	37
1.5.3 Sole lesions	37
1.5.4 Body condition score and digital cushion thickness	39
1.5.5 Pathological changes associated with claw horn disruption lesions	40
1.5.6 Other risk factors.....	41
1.5.7 Conclusions.....	45
1.6 Introduction to modelling and statistical techniques.....	46
1.6.1 Multilevel modelling.....	47
1.6.2 Bayesian framework	47
1.6.3 Stochastic models.....	48
1.7 Conclusions and aims	49
CHAPTER 2: General Introduction to the Study Herd, Data and Data Structuring	51
2.1 Introduction.....	51
2.2 Study herd	51
2.2.1 Overview.....	51
2.2.2 Lameness control	52
2.2.3 Feeding system.....	52
2.2.4 Genetic lines.....	53
2.2.5 Housing and environment	53

2.2.6	Data recording.....	53
2.3	Data selection and structuring.....	54
2.3.1	Introduction.....	54
2.3.2	Materials and methods	55
2.4	Results.....	61
2.5	Discussion.....	64
CHAPTER 3: Low Body Condition Predisposes Cattle to Lameness: An 8 Year Study of One Dairy Herd		66
3.1	Introduction.....	66
3.2	Materials and methods	67
3.2.1	Study herd and data.....	67
3.2.2	Statistical Analysis.....	67
3.3	Results.....	70
3.3.1	Stage 1: Multinomial model for repeated lameness events (Model 3.1).....	70
3.3.2	Stage 2: Binomial models for first lifetime lameness events (Model 3.2a, 3.2b, 3.3a and 3.3b)	78
3.4	Discussion.....	83
3.4.1	Body condition score	84
3.4.2	Body weight	85
3.4.3	Age at first calving.....	86
3.4.4	Previous lameness events.....	87
3.4.5	Feed and genetic group	87
3.5	Conclusions.....	88
CHAPTER 4: Lameness in Dairy Heifers: Impacts of Hoof Lesions Present Around First Calving on Future Lameness, Milk Yield and Culling Risk.....		89
4.1	Introduction.....	89
4.2	Materials and methods	90
4.2.1	Study herd.....	90
4.2.2	Statistical methods	93
4.3	Results.....	97
4.3.1	Descriptive analysis	97
4.3.2	Modelling.....	100
4.4	Discussion.....	107
4.4.1	Impacts of lesions present around first calving on culling.....	108
4.4.2	Impacts of lesions present around first calving on future lameness.....	109
4.4.3	Impacts of lesions present around first calving on milk yield.....	110

4.4.4	Study limitations and generalisability.....	111
4.5	Conclusions.....	112
CHAPTER 5: The Contribution of Previous Lameness Events and Body Condition Score to the Occurrence of Lameness in Dairy Herds: A Study of Two Herds.....		
5.1	Introduction.....	113
5.2	Materials and methods	114
5.2.1	Study herds.....	114
5.2.2	Statistical analysis	115
5.3	Results.....	130
5.3.1	Comparison of closed and simulation-based approaches to estimating population attributable fractions	130
5.3.2	Estimating PAF for BCS and previous lameness.....	133
5.4	Discussion.....	134
5.4.1	Previous Lameness Events.....	134
5.4.2	Body Condition Score.....	136
5.4.3	Comparison of closed and simulation-based approach for estimating population attributable fractions	137
5.4.4	Study Limitations and Generalisability.....	138
5.5	Conclusions.....	139
CHAPTER 6: Using Simulation to Investigate the Effect of Adopting Different Culling Rates on Lameness Prevalence in Dairy Herds		
6.1	Introduction.....	140
6.2	Materials and methods	142
6.2.1	Study herd	142
6.2.2	Statistical analysis	142
6.2.3	Modelling.....	142
6.2.4	Simulation.....	144
6.3	Results.....	151
6.3.1	Descriptive statistics	151
6.3.2	Model checking.....	151
6.3.3	Simulation results.....	152
6.4	Discussion.....	155
6.4.1	Use of simulation models.....	155
6.4.2	Culling rates and reasons for culling.....	155
6.4.3	Effect of culling rates and input parameter distribution on lameness prevalence.....	156
6.5	Conclusion	158

CHAPTER 7: General Discussion and Conclusions	159
7.1 Introduction.....	159
7.2 The use of statistical modelling to evaluate risk factors for lameness	160
7.3 The role of body condition score as a risk factor for lameness.....	163
7.4 Lameness history and the role of previous lameness as a risk factor.....	165
7.5 Future research.....	167
7.6 Conclusions.....	167
References.....	169
Appendix.....	183
Supplementary to Chapter 5; Figures.....	183
Supplementary to Chapter 5; Example OpenBUGS code for Model 5.2	185
Supplementary to Chapter 6; Example OpenBUGS code for Model 6.1	187

List of Tables

Table 1.1 Summary of published incidence rates for lameness in dairy herds worldwide.	24
Table 1.2 Summary of published prevalence rates for lameness in dairy herds worldwide...	26
Table 1.3 Examples of methods for data collection and on-farm recording of lameness and lesions associated with lameness in dairy cattle.	27
Table 1.4 Summary of some of the scoring systems reported in the lameness literature and used in research.....	28
Table 1.5 Common infectious causes of lameness and terms used in the literature. Adapted from Murray (2004).	31
Table 1.6 List of claw horn disruption lesions and alternative terms used in the literature...	32
Table 2.1 Target composition for the diets offered to low forage and high forage groups at the Langhill herd at Scotland's Rural College (SRUC) Dairy Research and Innovation Centre.	52
Table 2.2 Descriptions of the locomotion scoring system used at Scotland's Rural College (SRUC) Dairy Research and Innovation Centre based on Manson & Lever (1988).....	54
Table 2.3 Initial data extracted from the database at Scotland's Rural College (SRUC) Research and Innovation Centre for the period 1 st September 2003 to 30 th September 2011.	55
Table 2.4 Summary of body weight (BW) data available from records obtained from Scotland's Rural College (SRUC) Research and Innovation Centre.....	59
Table 2.5 Summary of data available before and after selection criteria applied during the data handling process for records obtained from the Scotland's Rural College (SRUC) Research and Innovation Centre.	61
Table 2.6 Summary of the data available in the final dataset (79,565 records) for records obtained from a total of 724 cows.	61
Table 3.1 Proportions of lame (mild and severe) observations in each lagged BCS category (3, 8, 12, and 16 weeks previously) for 724 cows.	71
Table 3.2 Model 3.1: Final multinomial model of risk of repeated lameness events for 724 cows with outcomes mildly lame and severely lame ^{1,2}	73
Table 3.3 Models 3.2a and 3.2b: Final binomial models of risk of first lifetime lameness event in heifers (first lactation) with outcomes mildly lame (Model 3.2a) and severely lame (Model 3.2b)	80
Table 3.4 Models 3.3a and 3.3b: Final binomial models of risk of first lifetime lameness event in cows second lactation or greater with outcomes mildly lame (Model 3.3a) and severely lame (Model 3.3b).	81
Table 4.1 Example young-stock ration fed to dairy heifers calving for the first time during the period August 2003 to March 2006 at the Scotland's Rural College (SRUC) Dairy Research and Innovation Centre.	91
Table 4.2 Description of severity scores for hoof lesions recorded for heifers calving during the time period August 2003 to March 2006 and lesion scores recorded during the period September 2003 to March 2006 (Offer et al, 2000; Leach et al, 2005).	92
Table 4.3 Summary of the number of hoof lesions recorded for each examination point and examination point and lesion cumulative incidence in 158 heifers calving during the	

time period August 2003 to March 2006 and lesion scored during the period September 2003 to January 2006.....	98
Table 4.4 Model 4.1: Binomial model for repeated lameness events in 158 heifers calving during the time period August 2003 to March 2006 and lesion scored during the period September 2003 to January 2006.	101
Table 4.5 Linear regression model (Model 4.2) for average daily milk yield within the Langhill herd in 157 heifers calving during the time period August 2003 to March 2006, with herd lifetime data recorded from September 2003 to August 2011.....	104
Table 4.6 Binomial model for survival to culling for 157 heifers calving during the time period August 2003 to March 2006 and lesion scored during the period September 2003 to January 2006.....	106
Table 5.1 Description of explanatory variables for Herd A (724 cows) and Herd B (1,040 cows) tested in Models 5.1a, 5.1b and 5.2.	116
Table 5.2 Final logistic regression model (Model 5.1a) with the outcome lameness events based on locomotion scores ≥ 3	122
Table 5.3 : Final logistic regression model (Model 5.1b) with the outcome lameness events based on 2 consecutive weekly locomotion scores 4 or 5.	124
Table 5.4 Final logistic regression model (Model 5.2) with the outcome lameness based on lameness treatment for all causes.....	125
Table 5.5 Summary of the results of Model 5.1a, 5.1b and 5.2 for explanatory variables body condition score (BCS) and previous lameness. Model 5.1a: Final model of lameness events based on locomotion score 3, 4 or 5 in the Scotland's Rural College (SRUC) Research and Innovation Centre dairy herd 2003 to 2011. Model 5.1b: Final model of lameness events based on 2 consecutive weekly locomotion scores 4 or 5 in the Scotland's Rural College (SRUC) Research and Innovation Centre 2003 to 2011. Model 5.2: Final model of lameness treated for all causes in a dairy herd in Somerset, UK.*.....	126
Table 5.6 Description of scenarios investigated for two UK dairy herds to estimate PAF for BCS and previous lameness.....	128
Table 5.7 Proportion of observations with body condition score (BCS) recoded in each category for the scenarios investigated for Herd A for the datasets used in Model 5.1a and 5.1b. Observations relate to weekly scoring of cows i.e. cow-week risk periods.	130
Table 5.8 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 (Model 5.1a and 5.1b) and 1,040 cows from a 600-cow herd in Somerset (Model 5.2)*.....	132
Table 5.9 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).....	134
Table 6.1 Logistic regression model constructed to estimate coefficients for previous lameness, parity and month in lactation using data obtained from 724 cows.	143

Table 6.2 Distributions for input parameters that were investigated in the simulation model including source for the main variables of interest; baseline risk in month one for each parity and previous lameness risk 1 to 4 weeks previously, 5 to 8 weeks previously and 9 weeks or more previously.	146
Table 6.3 Herd scenarios explored for adopting different culling rates.	149
Table 6.4 Prevalence of lameness events (locomotion score ≥ 3) for each week of the 4th month in lactation in 724 cows.....	151
Table 6.5 Summary of predicted mean and 95% credible intervals for lameness prevalence for different herd scenarios including; a range of input parameter distributions for baseline month 1 risk and previous lameness relative risk (RR) as well as different culling rates.....	154

List of Figures

Figure 1.1 Zones of the claw as developed from recommendations established at the 6 th Symposium on Diseases of the Ruminant Digit, Liverpool, 1990 (Greenough and Vermont, 1991). Zone 1 to 3; white line in the toe, abaxial and heel-bulb regions, zone 4; sole region, zone 5; toe, zone 6; heel, zone 0; interdigital space, zone 10; heel bulbs.....	30
Figure 1.2 Hypothesised causal pathway for the development of claw horn disruption lesions which have historically been described as ‘subclinical laminitis’; the available evidence to support this hypothesis is currently relatively weak.	34
Figure 2.1 Frequency distribution for body weight (BW) from records obtained from the Scotland’s Rural College (SRUC) Research and Innovation Centre.....	57
Figure 2.2 Frequency distribution for the difference between individual body weight (BW) recordings from the 7 day average BW for that cow.	58
Figure 2.3 Box plot showing count of the number of body weight (BW) recordings over 7 days per cow against average weekly BW, with all data points > 40kg difference between test day BW and weekly average BW included. The median and interquartile ranges of average weekly BW are shown by the horizontal line and whiskers accordingly and outliers are shown by circles.	59
Figure 2.4 Box plot showing count of the number of BW recordings over 7 days per cow against average weekly BW, with all data points > 40kg difference between test day BW and weekly average BW removed. The median and interquartile ranges of average weekly BW are shown by the horizontal line and whiskers accordingly and outliers are shown by circles.	59
Figure 2.5 Frequency distribution for body condition score (BCS), milk yield (kg), and body weight (kg) data in the final dataset constructed.....	62
Figure 2.6 Frequency distribution for the number of lameness events per cow where lameness event was defined as locomotion score (LS) 3, 4 or 5.	63
Figure 2.7 Frequency distribution for the number of lameness events per cow where lameness event was defined as locomotion score (LS) 4 or 5.	63
Figure 2.8 Lameness prevalence (based on locomotion score ≥ 3) over 421 weeks of the study period, 1 st September 2003 to 30 th September 2011.	63
Figure 3.1 Scatter plot and regression line (red line) created using ‘abline’ function in R (R Core Team, 2016) for body weight and body condition scores for 724 cows.	72
Figure 3.2 Median predicted risk of lameness from multinomial model parameters (model 3.1) for 724 cows. (a) Shows the median predicted risk for BCS categories 3 weeks previously with the outcome mildly lame and (b) severely lame. (c) Shows the median predicted risk for BW categories 3 weeks previously with the outcome mildly lame and (d) severely lame. (e) Shows the median predicted risk for age at first calving categories with the outcome mildly lame and (f) severely lame.	76
Figure 3.3 Predicted risk for repeated lameness events (LS = 3) using observed and predicted data including 95% credible intervals for level 3 (cow) (dashed blue line).	77
Figure 3.4 Predicted risk for repeated lameness events (LS ≥ 4) using observed and predicted data including 95% credible intervals for level 3 (cow) (dashed blue line).	77

Figure 3.5 Predicted risk for repeated lameness events ($LS \geq 4$) using observed and predicted data including 95% credible intervals for level 3 (cow) (dashed blue line). Predictions were made for weeks in milk = 20 and parity = 4.	78
Figure 3.6 Predicted risk for repeated lameness events ($LS = 3$) using observed and predicted data including 95% credible intervals for level 3 (cow) (dashed blue line). Predictions were made for weeks in milk = 20 and parity = 4. Stage 2: Binomial models for survival to first lifetime lameness event (Models 3.2a, 3.2b, 3.3a and 3.3b).....	78
Figure 3.7 Observed and predicted number of first lifetime lameness events ($LS = 3$) in heifers. Where week category 1 to 7 represent weeks of the study in 60 week blocks from 0 to 421 weeks. Posterior predictions were from Model 3.2a.	82
Figure 3.8 Observed and predicted number of first lifetime lameness events ($LS \geq 4$) in heifers. Where week category 1 to 5 represent weeks of the study in 60 week blocks from 0 to 240 and one block from 241 to 421 weeks. Posterior predictions were from Model 3.2b.	82
Figure 3.9 Observed and predicted number of first lifetime lameness events ($LS = 3$) in cows 2 nd lactation and greater. Where week category 1 to 4 represent weeks of the study; 1: 0 to 60, 2: 61 to 120, 3: 121 to 240, 4: 241 to 421. Posterior predictions were from Model 3.3a.	83
Figure 3.10 Observed and predicted number of first lifetime lameness events ($LS \geq 4$) in cows 2 nd lactation and greater. Where week category 1 to 3 represents weeks of the study; 1: 0 to 120, 2: 121 to 180, 3: 181 to 421. Posterior predictions were from Model 3.3b.	83
Figure 4.1 Cumulative frequency plot for white line lesion scores within each examination point for 158 heifers calving during the time period August 2003 to March 2006 and lesion scored during the period September 2003 to January 2006. Examination points 1 to 3 represent; 1 = 0 to 2 months pre-calving, 2 = 0 to 2 months post-calving and 3 = 2 to 4 months post-calving.....	99
Figure 4.2 Cumulative frequency plot for sole lesion scores within each examination point for 158 heifers calving during the time period August 2003 to March 2006 and lesion scored during the period September 2003 to January 2006. Examination points 1 to 3 represent; 1 = 0 to 2 months pre-calving, 2 = 0 to 2 months post-calving and 3 = 2 to 4 months post-calving.....	99
Figure 4.3 Observed and predicted repeat lameness events ($LS \geq 3$) per decile of fitted risk values. Posterior predictions were from Model 4.1, constructed using records obtained from animals for the study period 2003 to 2011.	102
Figure 4.4 Box plot showing median (black line) and interquartile ranges (whiskers) for number of days in herd for each sole lesion category 2 to 4 months post-calving (lesion scores for categories; 1 = 0 to 1, 2 = 2, 3 = 3, 4 = 4 to 8) for 157 heifers calving during the time period August 2003 to March 2006 and lesion scored during the period September 2003 to January 2006.	104
Figure 4.5 Box plot showing median (black line) and interquartile ranges (whiskers) for number of days in herd for presence or absence of digital dermatitis 2 to 4 months post-calving (lesion scores for categories; 0 = lesion absent, 1 = lesion present), for 157 heifers calving during the time period August 2003 to March 2006 and lesion scored during the period September 2003 to January 2006.....	105

Figure 4.6 Kaplan-Meier survival plot for sole lesion (SL) categories 0 to 4 months pre-calving for 157 heifers calving during the time period August 2003 to March 2006 and lesion scored during the period September 2003 to January 2006.....	107
Figure 4.7 Observed and predicted number of culling events in heifers per tertile of fitted risk values. Posterior predictions were from Model 4.3, constructed using records obtained from animals for the study period 2003 to 2011.	107
Figure 5.1 Frequency distribution for the number of lameness events per cow where lameness event is defined as locomotion score (LS) 4 or 5 on two consecutive weeks for records obtained from a total of 724 cows for the study period 2003 to 2011.	118
Figure 5.2 Observed and predicted repeat lameness events ($LS \geq 3$) per decile of fitted risk values. Posterior predictions were from Model 5.1a including random effects, constructed using records obtained from 724 cows for the study period 2003 to 2011.	118
Figure 5.3 Observed and predicted repeat lameness events ($LS \geq 3$) per decile of fitted risk values. Posterior predictions were from Model 5.1a excluding random effects, constructed using records obtained from 724 cows for the study period 2003 to 2011.	119
Figure 5.4 Observed and predicted repeat lameness events ($LS \geq 4$ on 2 consecutive weeks) per decile of fitted risk values. Posterior predictions were from Model 5.1a including random effects, constructed using records obtained from 724 cows for the study period 2003 to 2011.	119
Figure 5.5 Observed and predicted repeat lameness events ($LS \geq 4$ on 2 consecutive weeks) per decile of fitted risk values. Posterior predictions were from Model 5.1b excluding random effects, constructed using records obtained from 724 cows for the study period 2003 to 2011.	120
Figure 5.6 Observed and predicted repeat lameness events ($LS \geq 4$ on 2 consecutive weeks) per week of study category (categories 1 to 10 represent weeks 0 to 421 in blocks of 50 weeks). Posterior predictions were from Model 5.1b excluding random effects, constructed using records obtained from 724 cows for the study period 2003 to 2011.	120
Figure 6.1 Observed and predicted repeat lameness events ($LS \geq 3$) per month in lactation. Model constructed using records obtained from 724 cows.....	144
Figure 6.2 Flow diagram to demonstrate the structure of the simulation model to investigate the effect of culling rates on lameness prevalence. Blue arrow represents a draw from a binomial distribution with probability = $Lame_p$ to give a binary outcome for lameness. $Lame_p$ = lameness event probability. $Baseline_{risk}$ = lameness risk for month one for each parity. $Lame_prev1to4$ = lameness risk for previously lame in the previous 1 to 4 weeks. $Lame_prev5to8$ = lameness risk for previously lame in the previous 5 to 8 weeks.	144
Figure 6.3 Flow diagram to demonstrate the structure of the simulation model to investigate the effect of culling rates on lameness prevalence. Blue arrow represents a draw from a binomial distribution with probability = $Lame_p$ to give a binary outcome for lameness. $Lame_p$ = lameness event probability. $Prev_lact_{risk}$ = lameness risk carried over from the previous lactation. $Baseline_{risk}$ = lameness risk for month one for each parity. $Lame_prev1to4$ = lameness risk for previously lame in the previous 1 to 4	

weeks. `Lame_prev9toever` = lameness risk for previously lame in the previous 9 weeks or more. 145

Figure 6.4 Flow chart showing example herds with culling rate of 25% and 40% to illustrate the culling rate scenarios described in Table 6.3. `N` = number of animals. 150

CHAPTER 1: Introduction and Literature Review

1.1 Lameness and the UK dairy industry: An overview of the current situation

Lameness is defined as the clinical manifestation of impaired mobility and is commonly stated as being one of the most significant, growing problems the dairy industry faces (Huxley, 2012, Wilson-Welder et al., 2015). As global sustainability will undoubtedly drive the future of the dairy industry with environmental, economic and social awareness increasing; the ways in which dairy cows are managed in the UK may also change. Intensification, greenhouse gas mitigation and increasing efficiency demands on the food industry are already under the spotlight. This inherently highlights a set of health and production issues that need to be understood and managed; lameness, in particular, presents as a problem in modern intensive dairy farming systems (Nelson and Cattell, 2001). Higher yielding cows are at greater risk of lameness and the economics of producing milk may drive farmer's to retain lame cows within the herd (Archer et al., 2010b, Green et al., 2002, Barkema et al., 1994). Yet the extensive impact of lameness on herd performance is likely to be more significant than any other disease of cattle currently seen. The literature describes the significant impact lameness has on production losses (Cook and Nordlund, 2009, Warnick et al., 2001, Green et al., 2002), culling (Collick et al., 1989, Booth et al., 2004) and fertility (Hernandez et al., 2005, Garbarino et al., 2004) in dairy herds.

Currently, lameness in dairy cattle is considered to be at unacceptably high levels; Cook and Nordlund (2009) highlighted that the literature suggests the prevalence in intensively managed dairy cows is approximately 20% worldwide. This, and the growing awareness of the economic and welfare significance of the disease have contributed to a number of initiatives being launched in recent years aimed at the control of lameness including; the EU Lamecow project, national and international conferences and the AHDB Dairy Healthy Feet programme. However, lameness continues to present the dairy industry with one of its most significant challenges and in order to control the disease in a sustainable way there must be the ability to; (i) identify cases early and treat promptly and effectively, (ii) implement prevention and management strategies to reduce new cases and (iii) implement prevention and management strategies to reduce recurrent cases. A critical requirement to this being achievable is a better understanding of the evidence base for lameness including the aetiology & pathogenesis as well as

treatment and prevention. The literature for lameness, in comparison to mastitis and infertility, is comparatively lacking and much knowledge is still based on anecdotal evidence and experience rather than evidence based medicine (Bicalho et al., 2009, Hirst et al., 2002a). A recent literature review highlighted this issue for both the prevention and treatment of lameness in dairy cows (Potterton et al., 2012).

A major concern is the still poorly understood aetiology and pathophysiology of the non-infectious causes of lameness (Bicalho et al., 2007a, Bicalho et al., 2009). Whilst infectious causes of lameness such as digital dermatitis are a major problem for the dairy industry, claw horn disruption lesions (CHDLs), principally sole haemorrhage, sole ulcer and white line disease, represent one of the most common causes of lameness with the highest associated economic losses (Lischer et al., 2002b, Logue et al., 2004, Thoefner et al., 2004). Traditionally, CHDLs have been described in the literature with the term ‘subclinical laminitis’, implying a nutritional basis to the aetiology. Although acute laminitis with resultant inflammation of the laminae as well as clinical signs of lameness have been reproduced experimentally through oligofructose overloading, the link between subacute ruminal acidosis (SARA) and CHDLs as a subclinical form of laminitis has not yet been demonstrated (Donovan et al., 2004, Danscher et al., 2010). In fact, more recent research raises challenges to this theory and subsequently nutritional risk factors for claw horn lesions are now being viewed in a different light (Bicalho et al., 2009, Schopke et al., 2013). A number of studies have demonstrated a link between body condition score (BCS) and lameness that provides an alternative hypotheses for the aetiopathogenesis of CHDLs (Tisdall et al., 2013, Green et al., 2014). The multifactorial nature of lameness means that no single risk factor can be considered in isolation. Increasing use of complex statistical methods in the veterinary field has enabled researchers to investigate risk factors for lameness taking into account the multifactorial nature of the disease. These statistical techniques can help to provide insight and a better understanding of the aetiopathogenesis of lameness; which is critical for making progress in lameness control through identification and management of risk factors.

1.2 Significance of lameness in dairy cattle

1.2.1 Welfare

Welfare is a growing concern in the dairy industry with consumer awareness and farm economics driving a focus on improvement of dairy cow welfare. In the UK the Animal Health and Welfare Strategy for Great Britain, launched in 2004, set out objectives aimed to improve health and welfare standards and farm assurance schemes provide a formal framework to ensure the availability, validity and delivery of assurance information for food produced from farms (FAWC, 2005). The World Organization for Animal Health definitions for good animal welfare have been used to describe a lame cows as suffering from an unpleasant state (in pain), having poor biological function (affected milk production and fertility) and behavioural expressions being impacted (reduced mobility) (Keyserlingk et al., 2009). Whay and Shearer (2017) describes lameness as compromising all of the five freedoms (freedom from hunger and thirst, freedom from discomfort, freedom from pain, injury and disease, freedom to express normal behaviour and freedom from fear and distress).

Within the dairy industry lameness is considered as one of the most important welfare concerns, given the high prevalence as well as the evidence of pain; reported as lowered nociceptive thresholds in lame compared to non-lame cows (Whay et al., 1997, Barker et al., 2010, Machado et al., 2010, Ito et al., 2010). The increased risk of culling is also a serious consideration for poor welfare associated with clinical lameness (Juarez et al., 2003).

As an obvious sign of poor welfare that is evident to the casual observer, lameness has become increasingly important in terms of dairy cattle welfare, having been identified by the Farm Animal Welfare Council (FAWC) as a major welfare determinant (Wells et al., 1993). In recent years the UK dairy industry has developed standardized mobility scoring systems and the AHDB Dairy ‘Healthy Feet Programme’; to increase the level of awareness of lameness, increase the level of engagement with control on farm, and reduce its welfare impacts through improved diagnosis and management (AHDB Dairy, 2017a). The pressure on dairy farmers to maintain or improve welfare standards within their herds is likely to increase, and therefore the need to understand the methods by which lameness can be reduced is a high priority. An improved understanding of the causative factors of CHDLs in

particular will allow recommendations for reducing risk factors to be provided, with the aim of reducing lameness levels within herds.

1.2.2 Production & economics

Increased pressures on production efficiency and global sustainability within the dairy industry raises the need to address the negative impact lameness has on production parameters including culling rates, fertility and milk yield. This need is driven both at a farm level from a requirement for a cost-benefit of production and from an industry level to improve efficiency of food production and reduce impacts on climate change.

The antagonistic genetic correlation between 305-day milk yield and health traits including lameness implies that selection for milk yield is likely to result in more lameness (Pryce et al., 1997, Pritchard et al., 2013). Archer et al (2010) reported a milk yield reduction in severely lame cows from 4 months after the occurrence, however cows that were ever identified lame during the study were the highest yielding animals. This has implications in terms of lame cows potentially being retained within the herd due to their perceived high milk yield. However, the economic impact of clinical lameness has been clearly demonstrated in terms of milk yield reduction and the lost potential of high yielding cows (Green et al., 2002). This impact was seen from 4 months before until 5 months after diagnosis of a clinical case and results in up to 360 kg (range 160-550 kg) lost milk yield over a lactation. The significance of this is important for assessing the economic impact and importance of early identification of clinical cases.

The impacts of lameness on culling and fertility have been well documented. Culling has been reported as 2 times more likely in lame cows compared to non-lame cows by Booth et al (2004) and as 8.4 times more likely in cows with lameness score > 2 by Sprecher et al. (1997). Whilst, Bicalho et al. (2007b) reported a hazard ratio of 1.45 (SE = 0.17; P = 0.03) in lame versus non-lame cows. This may be as a result of decreased milk production or reduced reproductive performance of severe clinical lameness (Bicalho et al., 2007b).

The effect of lameness on reproduction is one of the main mechanisms by which lameness reduces productivity. All stages of fertility are affected, for example Garbarino et al. (2004) showed a 3.5 times greater odds of delayed cyclicity in lame versus non-lame cows, Lucey et al. (1986) showed longer calving-to-conception intervals in Friesian, Holstein and Ayrshire crossbred cows and Walker et al. (2008)

reported a reduced expression of oestrus. It has been hypothesized in the literature that the pronounced loss in body condition seen in lame cows during early postpartum and hence negative energy balance, impacts on fertility. Therefore if body condition could be maintained to reduce lameness, an additional additive effect on improved fertility potentially may be seen.

1.3 Prevalence and incidence of lameness in dairy cattle

There is wide variation in prevalence and incidence of lameness in dairy cows reported in the literature; explained partly by the absence of a standard definition for a clinical case of lameness, the subjectivity of diagnosis and variability in observer sensitivity (Archer et al., 2010b, Whay et al., 2003). Large variability in both the incidence and types of lameness between farms clearly exists, however the skill level of those identifying lame cows also varies (Green et al., 2002). It has been demonstrated that farmers will underestimate the prevalence and incidence of lameness in their herds and incidence has been found to be poorly correlated with records of treatment (Whay et al., 2003). Whay (2002) reported farmers identify fewer than 25% of lame cows in their herd. The AHDB Dairy mobility scoring system has been adopted in the UK as an industry standard to raise awareness and address this issue (Bell and Huxley, 2009, AHDB Dairy, 2017a).

1.3.1 Incidence of lameness

Incidence relates to the number of new events in a defined population within a specific time period and therefore deals with ‘new’ cases of disease (Dohoo et al., 2003). In terms of lameness, incidence rate is usually expressed as limb cases per 100 cow-years (Archer et al., 2010a).

$$\text{limb cases per 100 cow years} = \frac{\text{Number of new cases}}{\text{Time in herd spent not lame}} \times 100$$

The literature indicates a general trend for increasing lameness incidence over time. Archer et al. (2010a) suggested that in the UK the true extent of lameness levels is unknown, although based on studies reporting herd incidence a typical value of 50 limb cases per 100 cow-years was suggested. A large variation in reported incidence rates exists which may be due to a number of different reasons including; method of detecting lameness (e.g. vet treatments or farmer reported), experience and/or training

of the person identifying cases and whether treatment was incentivized (Table 1.1). Clarkson et al. (1996) used data recorded by farmers, veterinary surgeons, foot trimmer or other workers to calculate incidence rates with the aim that this dataset was as complete as possible. Whilst average incidence rates of 54.6 cases per 100 cow-years were reported these varied widely between farms (10.7 – 107.1%). Hedges et al. (2001) provided veterinary examination and treatments free of charge in a study of 5 farms and the whole herd were examined every 2 months by the vet to ensure no cows were left undiagnosed. Average incidence rate was 68.9% and again a wide variation was reported. Both these studies reported higher incidence rates compared with other UK studies where data was collected less intensively; Esslemont and Kossaibati (1996) reported an average incidence of 24.0 cases per 100 cows from retrospective health records. Where herds are under-reporting lameness or detection is poor, incidence rates may be underestimated.

Table 1.1 Summary of published incidence rates for lameness in dairy herds worldwide.

UK Data		
Reference	Incidence (cases per 100-cow years)	Number of farms/ practices
Prentice and Neal (1972)	30	7 farms
Eddy and Scott (1980)	7.3 (0 – 32)	273 farms
Russell et al. (1982)	5.5	48 practices
Whitaker et al. (1983)	25 (2-55)	185 farms
Collick et al. (1989)	17 (8-28)	117 farms
Esslemont and Kossaibati (1996)	24	90 farms
Clarkson et al. (1996)	54.6 (10.7 – 170.1)	37 farms
Whitaker et al. (2000)	23.7	340 farms
Hedges et al. (2001)	68.9 (31.6 – 111.5)	5 farms
Whitaker et al. (2004)	20.7 to 23.3	244 – 434 farms
Non-UK Data		
Reference	Incidence (cases per 100 cow-years)	Country
Gitau et al. (1996)	18	Kenya
Manske et al. (2002)	5.1	Sweden
Dembele et al. (2006)	22	Czech Republic
Barkema et al. (1994)	9-50	Netherlands
(Sanders et al., 2009)	49.1	USA
Tadich et al. (2010)	33.2 (large herds) 28.7 (small herds)	Chile
Alawneh et al. (2011)	15	New Zealand

1.3.2 Prevalence of lameness

Prevalence refers to the number of cases at a specific time point and therefore gives a ‘snap shot’ assessment. In terms of lameness this will be the proportion of lame cows in the at risk population at a specific time point (Dohoo et al., 2003). Mobility scoring can be used to provide an estimate of lameness prevalence in the herd.

$$Prevalence = \frac{\text{Number of lame cows}}{\text{Total number of cows}}$$

Lameness prevalence rates in larger UK studies have been reported at between 20.6% (2 – 53.9%) and 36.8% (0 - 78.9%) (Clarkson et al., 1996, Barker et al., 2010). Similar rates have been reported worldwide (Table 1.2) (Dembele et al., 2006, Solano et al., 2015, Cook, 2003). Whilst a trend towards increasing prevalence over time in the UK has been highlighted (Archer et al., 2010a), Cook et al. (2016) reported the converse pattern in herds in Wisconsin. This may have been due to herd selection being based on those with good production, reproduction and health records. Variation in reported prevalence rates may also be due to differences in methods of lameness identification and their sensitivity.

Table 1.2 Summary of published prevalence rates for lameness in dairy herds worldwide.

Reference	Country	Prevalence (%)	Number of herds
Wells et al. (1993)	USA	13.7 (summer), 16.7 (spring)	17
Gitau et al. (1996)	Kenya	11.7	78
Clarkson et al. (1996)	UK	20.6 (2-53.9)	37
Manske et al. (2002)	Sweden	5.1	101
Whay et al. (2003)	UK	22.1 (0 to 50.0)	53
Cook (2003)	USA	21 (summer) 24 (winter)	30
Huxley et al. (2004)	UK (organic herds)	24.2 (6.8 to 55.6)	15
Haskell et al (2006)	UK	15 (grazing) 39 (zero-grazed)	37
Dembele et al. (2006)	Czech Republic	22	24
Cramer et al. (2008)	USA	46.4 (free-stall) 25.7 (tie-stall)	204
Archer et al. (2010b)	UK	62 (48-72)	7
Barker et al. (2010)	UK	36.8 (0 to 79.2)	227
Keyserlingk et al. (2012)	USA	28 to 55	121
Solano et al. (2015)	Canada	21	141
Cook et al. (2016)	USA	13.2 (2.8 – 36.1)	66

The general increasing trend that has been reported in incidence and prevalence rates adds to the concern regarding lameness in UK dairy herds, as despite increased awareness and knowledge the condition is still a major problem. Higher milk yields have been associated with increased risk of lameness which may have contributed to this trend as average milk yield continues to increase (Reader et al., 2011, Green et al., 2002, AHDB Dairy, 2017b).

1.4 Lameness data; diagnosis & classification of lameness and hoof lesions

Measuring lameness levels requires effective record keeping in addition to standardised methods for identification and recording lameness events. Combined with appropriate data analysis, data generated can be used to make informed decisions and motivate change at a herd level with the aim of controlling lameness on-farm.

Historically, good quality data recording for lameness has lagged behind that for fertility and mastitis (Green, 2012). Table 1.3 summarises some of the lameness records (including lesions associated with lameness) that might be accessed on farm.

Table 1.3 Examples of methods for data collection and on-farm recording of lameness and lesions associated with lameness in dairy cattle.

Data	Comments on collection methods
Mobility (locomotion) scores	This may be the whole herd or systematic sampling. Scoring particular groups may skew the data
Trimming records	Methods of identification and classification of lesions may differ as well as what is recorded
Treatment records	Should include diagnosis, although methods of identification and classification of lesions may differ
Culling records	Should include rate and reason

Classification of a herd problem by grouping of lesions according to aetiology can be useful in order to rank the predominant causes of lameness and take appropriate steps to minimize the problem (Guard, 2000, Nordlund et al., 2004a). Nordlund et al. (2004a) suggests dividing lesions into 3 primary groups as a useful framework to approach herd level lameness problems;

- i. Infectious
- ii. Non-infectious
- iii. Excessive hoof wear and/or trauma

Based on descriptions of gross pathology at least 16 different forms of hoof lesions causing lameness have been recognised, although, of these there are two categories of lesions that most commonly present as a herd-level problem; digital dermatitis and CHDL (Murray et al., 1996, Knott et al., 2007).

Identification of lameness as well as causal lesions is subjective and therefore standardisation of methods becomes important. A standardised method of mobility scoring has been adopted in the UK (AHDB Dairy, 2017a), however a similar standardisation of recording hoof lesion data has not yet been so successful (Shearer et al., 2012). A number of systems exist including those developed by Bergsten et al (1998), Burgi (2000), and Shearer et al (2002). Greenough & Weaver introduced a concept of recording lesions by means of claw zones (Appendix 1) in 1992. The American Association of Bovine Practitioners (AABP) further developed this to

incorporate a lesion code, however this is reported to have not been widely used (Nordlund et al., 2004b). A reference list for the identification and diagnosis of lesions was produced by the EU lame cow project and the National Association of Cattle Foot trimmers (NACFT) utilise a standard recording sheet. The International Committee for Animal Recording (ICAR) have more recently produced the ICAR Claw Health Atlas that provides a new standard for the identification and recording of foot and claw lesions (ICAR, 2015), which helps to move the industry towards a standardized system and nomenclature for recording lesion type and severity. By standardising the recording of lesions this will aid in the monitoring of hoof disease and their relative importance (Nordlund et al., 2004b, Guard, 2000).

1.4.1 Visual assessment of locomotion

Lameness is defined by impaired mobility; a number of scoring systems using posture and gait evaluation have been developed to allow systematic identification and measurement of lameness (Table 1.4) (Manson and Leaver, 1988, Tranter and Morris, 1991, Wells et al., 1993, Sprecher et al., 1997, AHDB Dairy, 2017a, Cook, 2003).

Table 1.4 Summary of some of the scoring systems reported in the lameness literature and used in research.

Reference	Scoring System
Manson & Lever, 1988	9-point scale (1 to 5, with 0.5 increments)
Tranter & Morris, 1991	4-point scale (0 to 4)
Wells et al, 1993	4-point scale (0 to 4)
Sprecher et al, 1997	5-point scale (1 to 5)
Whay et al, 1997	6-point scale (1 to 6)
Vokey et al, 2001	4-point scale (1 to 4)
AHDB Dairy ‘mobility scoring’ system	4-point scale (0 to 3)

Sprecher et al. (1997) introduced the component of incorporating back posture into a scoring system, which is also included in the AHDB Dairy mobility scoring system. Most scoring systems rely on identifying lameness based on changes in movement such as nodding of the head, changes in stride length, and changes in pace (Chapinal et al., 2009). Training of operators scoring locomotion is important to ensure repeatability and consistency so that reliable data can be generated and a secondary observer can help to reduce bias introduced by subjectivity (Green, 2012). In the UK the Register of Mobility Scorers has been introduced to ‘*encourage widespread use of*

standardised, independent mobility scoring conducted by trained and accredited scorers on UK dairy farms'(RoMS, 2017).

Regular assessment of locomotion on-farm enables potential lesions to be identified and appropriately treated/managed. Observations suggest that approximately 90% of lameness is associated with lesions of the foot in dairy cattle; 92% of which are in the hindlimb and 65% with the lateral claw affected (Shearer et al., 2012, Murray et al., 1996). This finding was supported by Barker et al (2007) where causes of lameness were reported in the hindlimb in 79 to 96% of cases across 50 herds.

1.4.2 Other methods of lameness identification

In the last 15 years there have been considerable advancements in the use of technologies to automate lameness detection. Achieving this automation could provide benefits in terms of reducing the time resources associated with monitoring and identifying lameness compared with visual detection, the ability to continuously monitor and potentially identify lameness earlier, as well as providing an objective method of measuring lameness. Methods of automatic lameness detection include kinetic and/or kinematic analysis with the use of force plates to analyse gait length and weight distribution (Maertens et al., 2011, Pastell et al., 2010), video analysis to analyse gait (e.g. step over lap) or posture (e.g. back position) (Viazzi et al., 2013, Pluk et al., 2010, Poursaberi et al., 2010) and accelerometers and positioning to detect changes in movement and behaviour (Thorup et al., 2015, Chapinal et al., 2011). Increasingly these technologies are becoming commercially available (e.g. StepMetrix™ (BouMatic, Madison, WI, USA) and IceTag 3D™ (IceRobotics, Edinburgh, UK), although further research and development to optimise lameness detection, for example in detecting mildly lame cows, is still required. Infrared thermography is another technique that has been increasingly explored as a method for identifying lameness in cattle. This may be useful for the detection of early lesions, before cows show signs of pain through alterations in gait (Alsaad et al., 2015). Using combinations of sensors or other technologies may provide improved performance outcomes for lameness detection, as well as combining with other information such as lameness history (Alsaad et al., 2015, Van Hertem et al., 2013). Van Nuffel et al. (2015) reviewed some of the practical considerations associated with automatic lameness detection methods including the need for early detection and the added value of this for farmers, the ability for real-time analysis and detection of lameness and how systems can be incorporated into the

existing farm infrastructure, which all need to be considered as these technologies are adopted by the dairy industry.

1.4.3 Infectious causes of lameness

Terminology used in the literature to describe infectious causes of lameness varies (Table 1.5). The claw zones affected by lesions of an infectious aetiology include zone 0 and zone 10, although heel erosions may be recorded as zone 6 (Shearer et al., 2012) (Figure 1.1).

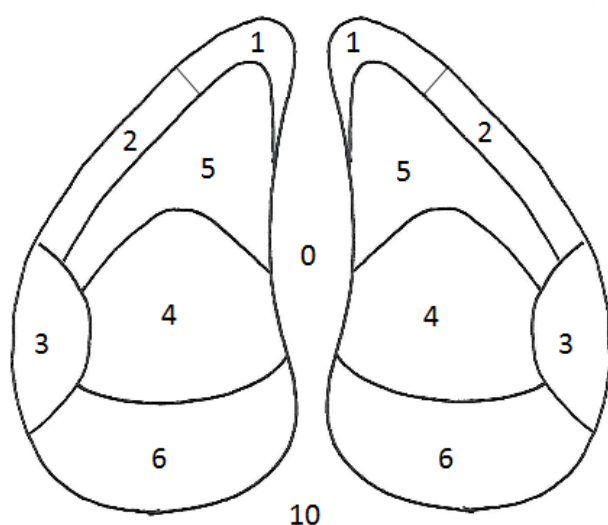


Figure 1.1 Zones of the claw as developed from recommendations established at the 6th Symposium on Diseases of the Ruminant Digit, Liverpool, 1990 (Greenough and Vermunt, 1991). Zone 1 to 3; white line in the toe, abaxial and heel-bulb regions, zone 4; sole region, zone 5; toe, zone 6; heel, zone 0; interdigital space, zone 10; heel bulbs.

Digital dermatitis is the most common infectious cause of lameness and is found worldwide (Wilson-Welder et al., 2015). Studies have demonstrated that lesions progress through different stages and classification systems have been developed to identify these stages (Berry et al., 2012, Dopfer et al., 1997). The precise aetiology of digital dermatitis still remains to be determined, however infection by numerous species of treponemes in addition to other anaerobic bacteria have been identified as being associated with disease (Wilson-Welder et al., 2015). Both herd and cow-level risk factors for digital dermatitis have been identified; a recent review of the literature highlighted physical factors such as hoof conformation and skin properties, physiological factors such as immune response and behavioural factors such as perching

in cubicles, as possibly influencing individual variations in susceptibility (Palmer and Connell, 2015).

Table 1.5 Common infectious causes of lameness and terms used in the literature. Adapted from Murray (2004).

Infectious cause	Alternative terms used in the literature
Digital dermatitis	Mortellaro disease Digital papillomatosis Papillomatous digital dermatitis Strawberry footrot Raspberry heel Hairy warts Hairy footwarts
Interdigital necrobacillosis	Interdigital phlegmon Infectious pododermatitis Foul-in-foot Hoof-rot
Heel horn erosion	Slurry heel

1.4.4 Claw horn disruption lesions

CHDLs principally includes sole haemorrhage, sole ulceration and white line disease (haemorrhage, separation and abscess). Claw zones can be used to record the location of the lesion; for example, toe lesions occur at the apex of the toe corresponding to zone 5, sole ulcers occur at the heel-sole junction corresponding to zone 4 and heel ulcers occur at the heel corresponding to zone 6. Lesions within zones 1, 2 or 3 that correspond to areas of the white line are described as white line disease (WLD) (Shearer et al., 2012). Sole ulcer is defined as a full-thickness defect of the epidermis, exposing underlying corium, whilst haemorrhage is believed to represent earlier stages of damage (Shearer et al., 2012, Shearer, 2010). Although full thickness deficits in the horn exposing the corium may occur at any site on the sole, the term sole ulcer is used to describe lesions at the typical site under the flexor tuberosity, whilst toe ulcer describes lesions at the apex site. White line disease describes a range of lesions; Cramer et al. (2008) separated these into white line disease associated with separation (breaks, separations or fissures that reach the corium but do not have associated pus or affected corium) and WLD associated with abscesses (abscess in the white line region). As with

sole lesions, white line lesions may vary in severity from haemorrhage through to separation reaching the corium.

Different methods for scoring CHDLs have been developed for research purposes, mostly based on a severity scale and/or area of lesion (Offer et al., 2000, Leach et al., 2005). As with infectious causes of lameness, a variety of terms have been used in the literature to describe CHDLs (Table 1.6).

Table 1.6 List of claw horn disruption lesions and alternative terms used in the literature.

Claw horn disruption lesions	Alternative terms used in the literature
Sole haemorrhage	Sole bruising Pododermatitis aseptica diffusa Laminitis
Sole ulcer	Rusterholz disease Pododermatitis circumscripta Laminitis
White line lesion	White line disease White line haemorrhage White line separation

Sole ulcer and white line disease are associated with the highest economic losses compared to other foot lesions (Bicalho et al., 2009). Although they are the most common cause of lameness in dairy cattle their underlying pathogenesis is still not fully understood (Bicalho et al., 2009, Manske et al., 2002). Investigating the risk factors for CHDLs and impacts of these lesions is one of the primary aims of the research reported in this thesis; the current literature surrounding the aetiology and pathogenesis of CHDLs is therefore explored further within this chapter (section 1.5).

1.5 Aetiology and pathogenesis of claw horn disruption lesions

Despite the significant economic impacts and high prevalence of CHDLs, the aetiology and pathogenesis of these lesions is still poorly understood. A range of risk factors have been identified including animal-based factors (i.e. internal factors originating within the animal; predominantly factors related to the structure and function of the claw) and environmental risk factors (i.e. external factors which operate beyond the individual; predominantly factors which directly or indirectly increase the pressure on the hoof capsule). CHDLs have been found to occur more commonly (75%) in the lateral hind claws compared to other claws (Tarlton et al., 2002, Winkler and

Margerison, 2012). Risk factors for sole lesions and white line lesions appear to be similar with some lesion specific factors having been identified in the literature (Barker et al., 2009). Historically, CHDLs have been commonly referred to a ‘subclinical laminitis’ with an implied nutritional aetiology, although increasingly the research is challenging this traditional hypothesis.

1.5.1 Laminitis and the suspensory apparatus

Use of the terminology ‘subclinical laminitis’ to describe the presence and diagnosis of CHDLs has a long history stretching back to the 1970’s (Maclean, 1971, Peterse, 1979). With that description comes an implied nutritional aetiology (rumen acidosis) based on an extrapolation from the described pathogenesis for equine laminitis associated with starch overloading. In horses, carbohydrate overload models (or toxin administration) have been used to demonstrate that an inflammatory response leads to loss of lamellar integrity (van Eps and Pollitt, 2006, Pollitt and Davies, 1998, Pollitt, 1996). The proposed pathogenesis for this was that the carbohydrates enter the hindgut, changing the bacterial population and lowering the pH which leads to caecal lining degeneration and absorption of toxins (Katz and Bailey, 2012). Following lamellar separation, rotation and sinking of the pedal bone occurs in horses, which may ultimately lead to the death of the animal. Despite extensive research, this pathological process has yet to be demonstrated to occur as a consequence of feeding high concentrate diets in dairy cows. Limitations with regard to the statistical modelling carried out in much of the original laminitis research in cattle have contributed to a lack of strong, but highly prevalent evidence in the literature; consequently the volume of potentially weak findings became the received norm of understanding in the field. More recently, in the equine field research has investigated the role of insulin resistance or hyperinsulinaemia in the development of laminitis (Katz and Bailey, 2012). Abnormal keratinisation and lengthening of secondary epidermal lamellae have been demonstrated with this form of ‘endocrinopathic’ laminitis, although the pathophysiology is still not fully understood. Similarly, in cattle it has been suggested from in vitro studies that glucose may have a critical role in keratinocyte viability in the bovine claw. Further work is required to understand the importance of this in terms of the effects of negative energy balance on the claw and development of CHDLs (Lubbe et al., 2017).

By definition *laminitis* refers to inflammation of the hoof laminae (rather than the presence of CHDLs), the interdigitating interface between dermal and epidermal components of the tissue suspending the distal phalanx within the claw capsule. Early research in cattle described laminitis as a condition associated with mostly *systemic* clinical signs alongside local clinical signs, such as foot pain and strong digital pulses (Nilsson, 1963, Andersson and Liberg, 1980), and the hypothesised disease process explaining the associations which were identified have been described by a number of authors (Ossent and Lischer, 1998, Nocek, 1997). This founding hypothesis for CHDLs maintained that rumen acidosis was central to initiating this disease process, with increasing recognition over time that other potential factors such as the environment, were likely involved (Bergsten, 2003, Livesey and Fleming, 1984, Nocek, 1997). The hypothesis was latterly extrapolated to describe a subclinical scenario; subacute rumen acidosis (SARA) was thought to be the cause of *subclinical* laminitis in high producing dairy cows fed diets high in concentrate, manifesting as CHDL (Figure 1.2) (Peterse, 1979, Stone, 2004, Nordlund et al., 2004a).

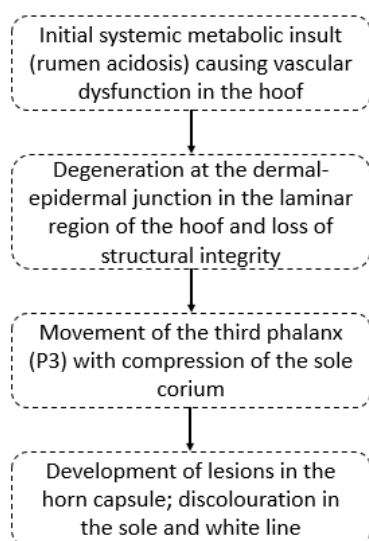


Figure 1.2 Hypothesised causal pathway for the development of claw horn disruption lesions which have historically been described as ‘subclinical laminitis’; the available evidence to support this hypothesis is currently relatively weak.

Early literature provided weak evidence from case studies or expert opinion to identify associations between dietary factors and CHDL (Maclean, 1971, Greenough, 1985). Since then, very few blinded RCTs and no systematic reviews have been conducted and reported. Most observational studies were conducted under field conditions (Bazeley and Pinsent, 1984, Manson and Leaver, 1988, Bergsten, 1994) and analysed without statistical modelling, to control for confounding factors, because

capabilities in this area were limited before software become widely available post-2000. Consequently, inferences from this early work should be interpreted with caution.

To explore the role of nutritional factors on the development of CHDLs, the effect of type and quantity of concentrates and forage have been investigated. A series of studies principally investigating the effect of forage types and dry matter content on CHDLs in heifers suggested a potential role in the aetiopathogenesis (Offer et al., 2001, Offer et al., 2003, Webster, 2001). Confounding factors such as body condition score (BCS) and days in milk were not controlled for which may have had an effect on the results reported. It is possible that effects of type and quantity of forage on CHDL identified may be due to mechanism not associated with inflammation of the laminae.

Studies exploring associations between dietary factors and the presence of CHDL have rarely investigated whether inflammation of the laminae actually occurs (a key step in the theory). Experimental oligofructose (a readily available carbohydrate) overload models have more recently been used to successfully induce acute laminitis (with histopathological changes in the lamellae) in profoundly acidotic animals with acute systemic clinical signs (Thoenes et al., 2004, Danscher et al., 2009). However, it has not yet been demonstrated that this translates into the SARA scenario commonly seen on-farm or whether inflammation of the laminae manifests as CHDL. Danscher et al. (2010) used the oligofructose overload model in an RCT to investigate whether acute laminitis was associated with any changes in biomechanical properties of the laminae in 6 heifers in the treatment group, compared with 6 heifers in a control group. No effect of oligofructose overload on physiological support of the suspensory tissue at 24 and 72 h after overload was found, although in the absence of sample size calculations it is not possible to assess whether this was due to insufficient study power. No information was provided on how this level of acute overload equates to diets commonly fed to high yielding dairy cattle on-farm. As these experimental studies have yet to provide strong evidence to support that laminitis occurs leading to development of CHDL following acute acidosis, its role in SARA manifesting as CHDL remains unproved. One RCT has investigated the association between CHDL and transition diets varying in levels of rumen-soluble carbohydrate, energy and fibre (Donovan et al., 2004). Rumen pH was monitored to determine whether there was an association between SARA (defined as rumen liquor pH ≤ 5.8) and CHDLs. Generalised linear models were used to control for confounding factors, although descriptions of model fit were not reported. While the diets effectively induced SARA, the study found no direct association between rumen

acidosis and CHDLs. Although this study was performed on only one research herd and further robust studies should be carried out to provide evidence to support the results reported, the findings further challenge the causal assumption that SARA is involved in the pathogenesis of CHDLs.

Much of the research investigating the effects of diet on CHDLs has focused on the change from pre to post-calving diets (Offer et al., 2001, Webster, 2001, Livesey et al., 1998); a time period when major management changes occur. However, major physiological and hormonal changes also occur with calving and findings from a number of studies have suggested that these may be more important for the development of CHDLs than dietary influences (Livesey et al., 1998, Webster, 2001, Knott et al., 2007). Lischer et al. (2002b) identified in a study of 19 cows that although gross or microscopic inflammatory changes of the lamellae were not associated with sole haemorrhage or ulcerations, thinning of the corium and displacement of the distal phalanx was. An alternative hypothesis was suggested that parturition and/or lactation are involved in the weakening of suspensory tissues (Tarlton et al., 2002). This hypothesis was supported by findings reported in a study investigating the effect of diet, housing and parturition on claw horn lesions in heifers (Knott et al., 2007). Multiple regressions were used to explore treatment effects on biomechanical and biochemical properties. Confounding factors were not controlled for in the analysis and model fit was not described, however findings suggested that housing and parturition may be important risk factors for the development of CHDLs. These studies suggest that a ‘parturition effect’ causing a weakening of the suspensory tissues may be responsible for instability of the distal phalanx and resultant damage to underlying corium, causing CHDL and provides a plausible alternative hypothesis that should be proven with further robust studies.

In summary, the quality of available evidence to support a causal relationship between SARA and CHDLs is relatively weak and cannot currently substantiate this aetiopathogenesis. Continued use of the terminology ‘subclinical laminitis’ to describe CHDLs adds to the bulk of literature with an implied nutritional aetiology; consequently the terminology “laminitis” should be reserved exclusively for studies demonstrating inflammation of the laminae, as suggested by previous authors (Livesey et al., 2003, Laven, 2006). It is incumbent on the research community to conduct high quality, robust intervention studies to provide stronger causal evidence in this contentious and currently disputed area.

1.5.2 White line lesions

The white line is a complex structure at the junction between the horn wall and sole of the claw; produced by epidermal cells in the wall region (Mulling, 2002). The white line region is particularly susceptible to penetration. Mulling (2002) describes the reasons underlying this as the surface area of dermis being small relative to rates of horn production in the region where it originates as well as the sensitivity of the vasculature to disturbance. The heterogenous nature of the horn in this region where sole and wall horn meet is susceptible to fissures, foreign body penetration and microorganisms. This is in addition to susceptibility to increased mechanical stresses.

The pathogenesis of white line lesions has been described as; lesions occurring without prior weakening where WLD is a primary disease and/or as lesions with prior weakening due to subclinical laminitis and disturbance of microcirculation (Mulling, 2002). Although similar risk factors have been described for white line lesions and sole lesions, it has been shown that these lesions occur at slightly different times post-partum; WLD at 2 months and sole lesions at 4 months (Offer et al., 2003). The aetiology for white line lesions has yet to be fully understood, however a number of risk factors specific to these lesions have been identified. Solid grooved concrete floors and access to pasture were associated with white line disease in a study of 27 UK dairy herds where multilevel modelling was used to control for clustering effects and confounding (Barker et al., 2009).

1.5.3 Sole lesions

Sole lesions (sole ulcer and sole haemorrhage) are one of the commonest causes of lameness in housed dairy cows (Lischer et al., 2002a). The lesion is significant in terms of its high prevalence, high recurrence rates and the insidious nature of onset with associated difficulties in identification and treatment (Lischer et al., 2001, Lischer et al., 2002a). The majority of sole ulcers are observed within the first 10 weeks after calving with a reported prevalence of 4.2% and 27.8% for parities 1 and >1 respectively (Tarlton et al., 2002, Bicalho et al., 2009). Manske et al. (2002) reported a prevalence of 30% for sole haemorrhages and 8.6% for sole ulcers in 101 Swedish dairy farms, whilst Barker et al. (2009) reported from a UK study of 27 dairy herds the rates per 100-cow months of treatment for sole ulcer was 0.38 and 0.56 in herds with 80 to 159 cows and ≥ 160 cows, respectively.

Sole ulceration, as described first by Rusterholz (1920), is still considered to occur subsequent to sole haemorrhage; with chronic injury of the corium at the palmaro/plantarodistal edge of the distal phalanx resulting in inflammation and interruption of claw horn production to the extent that pathological exposure of corium occurs. Nuss (2014) described sole ulcers as occurring subsequent to compression of sole corium, causing inflammation, alterations in keratogenesis and development of sole ulceration. Bicalho and Oikonomou (2013) highlighted in a recent review that it is now widely accepted that sole ulcers occur as a consequence of contusions within the claw capsule; providing an explanation for why sole ulcers more commonly occur on the lateral claw of hindlimbs where higher biomechanical forces have been measured compared with the medial claw (Van der Tol et al., 2002). The digital cushion sits between P3 and the sole horn and is thought to provide a supportive function by dissipating concussive forces within the hoof capsule during claw strike, therefore thinning of the digital cushion provides another potential hypothesis to explain the pathogenesis of sole lesions (Lischer et al., 2002b, Raber et al., 2004). As sole haemorrhage is considered a precursor to sole ulceration they are considered to have the same aetiology.

Numerous risk factors for CHDLs have been described in the literature including both animal-based and environmental factors (Foditsch et al., 2016, Mulling and Greenough, 2006). Hormonal, metabolic, nutritional and postural changes around parturition have all been implicated in the pathogenesis of sole ulceration (Chapinal et al., 2009, Tarlton et al., 2002, Winkler and Margerison, 2012). Newsome et al. (2016) summarised the hypothesised pathways associated with the development of CHDLs. This incorporated a hypothesis that has previously been unexplored; the role of inflammation in the pathogenesis of CHDLs. Body condition score has been identified as a potential risk factor for CHDLs in a number of recent longitudinal studies, although the underlying pathogenesis is poorly understood and the complex relationships between low BCS and lameness have not yet been fully elucidated (Lim et al., 2014, Green et al., 2014). As lameness itself may cause a loss in BCS it is important to understand the temporal relationships between BCS and lameness; this is one of the primary aims of this thesis.

1.5.4 Body condition score and digital cushion thickness

A number of longitudinal studies have used multivariable modelling to demonstrate an association between low BCS and lameness whilst controlling for milk yield and other confounding factors. Hoedemaker et al. (2009) reported that cows with low BCS (< 3.0) at calving and 4-10 weeks post calving were at increased risk of lameness (OR 2.9 - 9.4), similarly Green et al. (2014) reported cows with BCS < 2.5 had an increased risk of CHDLs (sole ulcer / white line disease) 0 – 2 months and 2 – 4 months later in a longitudinal study of 1137 cows over 44 months on one UK dairy herd. Lameness in this latter study referred to treatment incidence and mixed effects multivariable models were used to account for clustering and control for confounding factors e.g. days in milk. Lim et al. (2014) used mobility scores recorded every 14 - 15 days across 4 UK herds to investigate associations between BCS and lameness, again using mixed effects multivariable models. Cows with BCS < 2.25 at calving had an increased risk of becoming lame and reduced risk of recovery from lameness. In addition a loss or gain of BCS after calving impacted on lameness occurrence and recovery.

One hypothesis to explain this association between low BCS and lameness, is that the digital cushion, a structure sitting beneath the distal phalanx that contains depots of adipose tissue and functions to dissipate concussive forces, becomes thinner with loss in body condition (Bicalho et al., 2009). Bicalho et al. (2009) highlighted that previous work by suggested the digital cushion had an important role in dampening compression of the corium under the distal phalanx (Raber et al., 2004, Raber et al., 2006). In addition, Raber et al. (2004) had shown that older cows tend to have lower BCS, reduced digital cushion thickness and increased levels of hoof damage. In a cross-sectional study, Bicalho et al. (2009) used ultrasonographic examination of the soles of 501 lactating Holstein dairy cows to investigate the association between CHDLs and the thickness of the digital cushion. A significant positive association was found between thin digital cushion and prevalence of sole ulcers and white line disease as well as body condition score and digital cushion thickness. In addition, changes in composition of the digital cushion were identified with varying thickness and a nadir in thickness was measured 120 days post-partum. It was therefore suggested that reduced supportive capacity of the digital cushion with lower BCS resulted in greater pressure being exerted by the distal phalanx on underlying soft tissue and subsequent development of claw horn lesions due to the contusion. However, as this was a cross-

sectional study causal mechanisms could not be established. More recently (Newsome et al., 2017b) investigated the relationship between sole soft tissue thickness (digital cushion and corium), back fat thickness and CHDL in a longitudinal study of 179 cows using survival models. Although thin sole soft tissue increased the likelihood of lesion occurrence and lameness, an additional effect of thin sole soft tissue and thinning of back fat thickness was observed. These findings suggested that back fat thickness and sole soft tissue thickness had effects on lesion occurrence that were independent of each other. Further work is needed to understand these different effects and understand the underlying mechanisms that influence development of CHDLs. The relationship between milk yield, BCS and lameness is complex; Reader et al. (2011) hypothesised that reduction in milk yield and risk of lameness may be related by a common causal pathway; low body condition score, however there may also be alternative common causal pathways that explain these associations. Lameness may impact on body condition score through altered feeding behaviours (Norrington et al., 2014, Thorup et al., 2016), therefore understanding the temporal associations between BCS, milk yield and lameness is important. So far studies have not investigated the impact of BCS on first lifetime lameness events and therefore whether changes in BCS have been influenced by previous lameness has yet to be clarified fully. In addition, optimum BCS thresholds for controlling lameness have not yet been identified.

A greater understanding of the relationship between BCS and lameness may help to reduce lameness in herds through improved management of BCS; the primary aim of Chapter 3 was to investigate this relationship.

1.5.5 Pathological changes associated with claw horn disruption lesions

It has been widely reported that lameness events increase the risk of future lameness events occurring (Green et al., 2014, Hirst et al., 2002b). Hirst et al. (2002b) suggested that this association may be explained by the presence of underlying pathology that is carried over from one lactation to another. The increase in lameness prevalence or risk with increasing parity would support this hypothesis (Barker et al., 2009, Solano et al., 2016). Newsome et al. (2016) recently reported findings from a study investigating the association between anatomical changes on the distal phalanx at culling with previous history of lameness and lesion occurrence. X-ray micro-computed tomography was used to image 282 hind claws from 72 Holstein-Friesian culled cows. Linear regression models controlled for confounding factors including age and previous

locomotion scores, although with this type of study design caution should be taken with regards to causal inferences. Treatment for CHDL during life was associated with 7.0mm (SE 2.2) greater increase in bone development (maximum value) compared with cows not treated and cows that had been lame at > 50% of scores within the previous 12 months before slaughter had increased bone development compared to cows not lame. These findings provide evidence to support the hypothesis that long term pathological changes in the hoof are associated with CHDLs contribute to the pathogenesis of future development of lesions, although further work is required to confirm causality.

1.5.6 Other risk factors

Besides the animal-based risk factors highlighted and discussed previously including body condition score and calving, other animal-based and environmental risk factors for CHDLs have been identified including;

1.5.6.1 Animal behaviour and management

Animal behaviour and factors that influence behaviour, including handling and cow flow, have been identified as potential risk factors for lameness. Chesterton (1989) reported in a case-control study of 62 dairy herds in New-Zealand that the degree of patience shown by the farmer in bringing the cows in for milking explained significant variation between case and control herds. Barker et al. (2010) demonstrated in a longitudinal study of 205 dairy farms that cows pushing each other or turning sharply near the parlour entrance or exit was a risk factor for increased lameness. Both these studies suggest that management factors that influence cow flow may be important in terms of increasing the risk of lameness. This may be due to increased forces on the claw with abnormal loading associated with for example pushing and sharp turning.

Cow behaviour is influenced by the social hierarchy within groups of cows and it has been demonstrated that this may also impact on lameness risk. For example, Barker et al. (2007) demonstrated in a study of 49 farms that pregnant heifers kept with milking cows in winter compared with being kept with dry cows was a risk factor for lameness. The lower social hierarchy of heifers may result in longer standing times and increased competition for feed and space as well as increased evasive manoeuvres that may increase pressures on the claw. Galindo and Broom (2000) reported that low-ranking cows spend less time lying and more time standing half in the cubicles compared with

higher-ranking cows (see also section 1.5.6.4) and a significantly reduced survival rate to lameness in low-ranking versus higher-ranking cows.

Further work to identify optimum cow management, including reducing social stressors, may be important in reducing lameness in dairy herds.

1.5.6.2 Claw conformation and posture

Inappropriate claw conformation may contribute to development of CHDLs due to imbalances in weight distribution, which increase biomechanical forces through the hoof during limb loading. Maintaining appropriate distribution of weight across claws to re-establish function within the claw is one of the aims of routine foot trimming, with the aim of preventing claw disease. Conversely, over-trimming can result in imbalances in claw thickness or excessive removal of claw horn and is therefore recognised as a risk factor for CHDLs (Nordlund et al., 2004a). In a study of 53 cattle over-trimming was accounted for as the major cause of toe ulcers (49%) (Kofler, 1999). Barker et al. (2007) identified routine hoof trimming of all cows as a management risk factor for lameness. One explanation suggested for this association was poor hoof trimming technique may result in increased lameness.

Capion et al. (2008) used multilevel modelling in a longitudinal prospective study of 147 heifers from 5 farms to explore the dynamics of abnormal hind leg conformation, asymmetric claws, lameness and foot lesions. Authors highlighted that cause and effect relationships were not clear and a vicious cycle may be occurring; for example cow-hock posture may be both a cause and effect of a foot lesion. Increased severity of white line lesion was associated with greater claw asymmetry whereas lameness score and sole haemorrhage appeared more severe in symmetric claws; the latter an indicator potentially of the early stages of this cycle. Further studies are required to support this hypothesis. Other postural and claw conformation factors that have been suggested to be associated with CHDLs include foot/claw angle, bone structure and hock angle (Perez-Cabal and Charfeddine, 2016). Currently, there is a lack of evidence for the role of claw conformation and posture in the pathogenesis of CHDLs, although the increase in biomechanical forces is biologically plausible and has been demonstrated to be present in areas associated with injury (Van der Tol et al., 2002).

1.5.6.3 Body weight

Mechanical stress acting through the claw during standing and walking are influenced by body weight and it therefore appears plausible that increased body weight would be associated with increased risk of CHDLs (Bergsten, 2001, Gearhart et al., 1990). However, body weight may influence lying times/feeding times, social hierarchy and milk yield which could also impact on claw horn lesions through other causal pathways. Perez-Cabal and Charfeddine (2016) demonstrated a positive relationship between BW and claw horn lesions, although modelling did not control for milk yield effect. Mixed effects modelling has been used to demonstrate a negative association between BW and lameness (Onyiro et al., 2008, Norring et al., 2014), although these studies do not provide information on CHDLs specifically. Further work to understand the complex associations between BW and CHDLs will aid in understanding where management factors may have an impact on reducing these lesions.

1.5.6.4 Standing/walking surface

Floor surface hardness and/or abrasiveness are believed to influence the development of CHDLs by increasing biomechanical forces on the claw capsule. This may occur either as a direct effect of surface hardness or an indirect effect of surface abrasiveness altering claw wear and growth rates leading to a medial-lateral foot imbalance, thinning of the sole or increased sole surface contact area (Vanegas et al., 2006, Bergsten et al., 2015, Telezhenko et al., 2009, Bergsten, 2001, Cook et al., 2004). A number of randomized trials have explored the associations between flooring surfaces of differing hardness / elasticity and abrasiveness; in particular rubber versus concrete (Bergsten et al., 2015, Sogstad et al., 2005, Vokey et al., 2001, Vanegas et al., 2006). Bergsten et al. (2015) reported findings supporting the hypothesis that slatted rubber versus slatted concrete in cubicle housing resulted in a reduced risk of CHDLs; odds ratios (confidence interval) of sole haemorrhages and white line haemorrhages were 2.19 (1.00 – 4.97) and 2.82 (1.28–6.43), respectively. In contrast, Vokey et al. (2001) used univariable methods and was unable to detect significant differences between rubber and concrete treatment groups; a limitation that was acknowledge by the authors. Vanegas et al. (2006) was also unable to detect a significant effect of flooring surface, however as sample size calculations were not reported, it is possible these findings may have been due to insufficient study power. Sogstad et al. (2005) identified that concrete alleys were associated with CHDLs in a cross-sectional observational study using

mixed effects modelling to account for the cluster effects within herd and claw trimmer. With this type of study design caution should be taken with regards to causal inferences.

Findings from these studies provide some evidence to demonstrate an association between flooring surface and CHDLs, however they did not explore the underlying mechanisms. Knott et al. (2007) explored the impact of housing on biochemical and biomechanical properties of the hoof; regression analysis showed an increase in elastin in the corium of cows calving and housed in cubicles which would confer properties of increased laxity. This increase in elastic fibres was thought to be associated with the mechanical stresses being exacerbated by cubicle housing. Descriptions for whether and how models controlled for confounding factors were not provided, impacting on the interpretation of results; further work is needed to substantiate these findings. Multivariable logistic regression was used to control for confounding factors in this study. Computer-based finite element models have also been used to investigate the effect of flooring conditions on mechanical stresses and may provide a valuable method for investigating the effect of flooring surfaces on bovine claws (Hinterhofer et al., 2005, Franck et al., 2008). Differences in growth and wear associated with flooring surface have not yet been demonstrated to impact on claw imbalance or CHDLs (Telezhenko et al., 2009, Vanegas et al., 2006). As an alternative hypothesis, Muggli et al. (2016) suggested the disparity between the length of the lateral and medial digits in the hindlimb could explain the predisposition of the lateral hind claw to develop CHDLs. This may also have clinical relevance for cattle exposed to hard flooring surfaces such as concrete (Nuss, 2014). The role of this finding in the pathogenesis of CHDLs has yet to be investigated fully.

The abrasiveness and/or unevenness of the surface which animals stand or walk on is another environmental risk factors that has been investigated. Chesterton (1989) demonstrated in a case-control study of 62 dairy herds that level of maintenance of cow tracks was one of the most influential variables investigated. The outcome was high or low herd level lameness prevalence and multivariable analysis was used to investigate risk factors, but again causality cannot be attributed using this study design. Furthermore, Burow et al. (2014) showed prepared track covers were significantly associated with a lower probability of lameness although this study did not identify individual lesions. Further research using well-designed intervention studies is needed to better understand how track surfaces influence the pathogenesis of CHDLs and to identify optimum substrates.

1.5.6.5 Standing/lying times

Maximising lying times in housed cattle has been suggested to reduce the rate of new cases of lameness because of the reduced time spent standing on concrete (in housed herds); good stall design and appropriate access to stalls are important in achieving this (Cook and Nordlund, 2009, Bergsten, 2001). Numerous studies have demonstrated an association between cubicle design and bedding surfaces with lameness in cattle (Chapinal et al., 2013, Vokey et al., 2001, Philipot et al., 1994, Livesey et al., 2003, Faull et al., 1996, Espejo and Endres, 2007, Cook, 2003, Sogstad et al., 2005). Where studies have investigated the effect of access to pasture or straw yards compared with cubicle housing, improved lying times may also have provided a beneficial effect for CHDLs (Bergsten and Frank, 1996, Hultgren and Bergsten, 2001, Fregonesi et al., 2009, Haskell et al., 2006).

The suggested mechanism by which standing/lying times increase the risk of CHDLs is due to increased biomechanical forces acting on the claw because of either i) increased standing time per se or ii) standing half in cubicles (perching) leading to increased pressure on the hind feet. As lameness itself impacts on lying behaviour (Solano et al., 2016), understanding the relationship between environmental risk factors that increase lying times and CHDLs is complex. Proudfoot et al. (2010) used multivariable analysis in a study of 26 Holstein dairy cows to demonstrate that sole lesions in mid-lactation were associated with increased standing times in the 2 weeks before and 24 hours after calving. Sole lesions were found to be associated with an increased time spent half-in cubicles and an increase in standing bout duration. Although the longitudinal nature of this study provides some evidence towards cause and effect it is possible that factors not measured, such as previous lameness events, may have confounded these results. Further work to understand the temporal associations between standing/lying times and the development of CHDLs are needed to identify how environmental and management factors may influence lameness.

1.5.7 Conclusions

Since the laminitis hypothesis was first put forward in the 1960s there has been a lack of strong evidence to substantiate the rumen-acidosis laminitis causal pathway for the development of CHDLs. Despite extensive research having been conducted investigating the association between high concentrate diets and CHDLs much of this work was carried out prior to statistical methods and software being readily accessible

to researchers. This has meant that a large body of research has been published where the multifactorial nature of lameness was not fully recognised and confounding factors were not always adequately controlled for. More recently, there have been developments in our understanding surrounding the aetiology and pathogenesis of CHDLs, in particular some of the animal-based risk factors. Body condition score is potentially an important risk factor for lameness, however the temporal associations between low BCS or loss in BCS and lameness need to be explored in more detail; in particular the associations with first lifetime lameness events. In addition, identifying optimum BCS would aid in providing recommendations to help support better management of nutrition and BCS in order to reduce lameness. The long-term pathological changes associated with lameness and the increased risk of future lameness is another area where further work is needed. Research to date has highlighted the importance of controlling lameness in heifers in particular, however understanding the impacts of lameness in heifers on future health and production would help to highlight this further. Quantifying the contribution that risk factors make towards the total number of lameness events in herds would also help to identify where control measures will be most effective in reducing lameness at the herd level.

1.6 Introduction to modelling and statistical techniques

Increasingly, more advanced statistical techniques are being used within veterinary research which enables complex biological systems to be evaluated. For multifactorial diseases such as lameness where temporal associations are also important, multivariable and multilevel modelling can be used to control for confounding and clustering of data to provide more accurate parameter estimates. The primary dataset used within this thesis was retrospective data collected over an 8 year time period with repeated measurements of the variables investigated. Depending on whether the outcome was categorical, dichotomous or continuous, either multinomial, logistic regression or linear regression was used in a multilevel framework (Chapters 3, 4 and 5), therefore a general introduction to multilevel modelling is provided within this section. Throughout this thesis a Bayesian framework is used for parameter estimation for a number of reasons including; improved parameter estimation of logistic regression models (Rabash et al., 2009) and use of posterior predictions to provide results that are easier to interpret. An introduction to Bayesian statistics is therefore provided. Finally, an introduction to

stochastic models is also given as this was the primary approach to data analysis used within Chapter 6.

1.6.1 Multilevel modelling

Data used in veterinary research very commonly has a multilevel or hierarchical structure; for example lesions can be recorded on a claw (level 1), which belong to a particular cow (level 2), which are managed on a particular farm (level 3). Repeated observations, for example weekly locomotion scores over a period of time can also be considered as a level. A feature of data with this structure is clustering; for example, management practices on a particular farm will mean cows on that farm are more likely to be similar in some respects e.g. mean BCS due to the composition of the diet fed. It is important that statistical analyses recognises this structure and multilevel modelling is therefore a useful statistical tool. By not recognising hierarchical structure where it is present, this introduces bias and parameter estimates will not be estimated correctly (Rabash et al., 2009). The data used throughout this thesis was structured at the cow-week level and multilevel modelling was used to recognise this hierarchical structure.

1.6.2 Bayesian framework

Whilst the frequentist approach (likelihood-based) is concerned with the frequency with which data is observed to test a hypothesis, the Bayesian approach is concerned with the probability of the hypothesis given knowledge about the model parameters. In the Bayesian framework a probability distribution is given to the parameters (the prior distribution) as well as the data. When the data and prior distribution are combined this provides the posterior distribution, which summarises knowledge about the parameters after observing the data (Dohoo et al., 2003). The Bayesian approach is therefore sequential; for example posterior predictions can be used as prior distributions and by incorporating this new data a new posterior prediction will be given.

It has been demonstrated that with multilevel logistic regression models, likelihood-based methods can result in biased estimates, despite being computationally faster (Browne and Draper, 2006). Markov chain Monte Carlo (MCMC) methods provide an alternative by fitting multilevel models within a Bayesian framework and has been used as an approach throughout this thesis. MCMC is a simulation-based procedure; given initial starting values for model parameters, an estimation for each

parameter is produced at each iteration. Therefore with each iteration the estimate is updated. The final converged posterior predictions are used to generate summary statistics (e.g mean) with accurate interval estimates. It is important to ensure that model convergence has taken place and samples from the burn-in period are discarded (Green et al., 2004). Gelman and Shirley (2011) summarises some of the approaches to this including running multiple chains.

1.6.3 Stochastic models

Whilst the predictions from deterministic models are determined by specified values for the initial conditions and input parameters (point estimates), stochastic models reflect uncertainty through model parameters being estimates with a specified distribution (probabilistic). Wilkinson (2009) provides a description of stochastic models as *‘A model that contains an element of unpredictability or randomness specified in a precise mathematical way. Each run of a given model will produce different results, but the statistical properties of the results of many such runs are pre-determined by the mathematical formulation of the model’*. When investigating the effect of changes in a factor in a complex and dynamic biological system, incorporating this uncertainty into a model can help to quantify these effects by taking into account the variation that other factors will have. For example, in Chapter 6 the effect of herds adopting different culling rates is investigated using a stochastic model. This model incorporates the variability that may be present in terms of the risk associated with previous lameness and the baseline risks of lameness that will change over time in a herd. These models therefore provide increased flexibility by capturing multiple sources of heterogeneity in order to improve the evaluation of complex biological scenarios. Stochastic modelling has a long history of use in biology (Galton and Watson, 1875, Goel and Richter-Dyn, 1974), however with increased computational capacities its use has increased dramatically. Stochastic models have also been increasingly used to investigate dairy herds including exploring; farm efficiencies (Kumbhakar et al., 1991, Bravo-Ureta and Rieger, 1991), management decisions (Dijkhuizen et al., 1986) as well as control strategies and/or economic consequences for infectious diseases (Kudahl et al., 2007, Sørensen et al., 1995), lameness (Ettema et al., 2010, Bruijnis et al., 2010) and mastitis (Østergaard et al., 2005, Down et al., 2013, Hudson et al., 2015). One important element to stochastic modelling is the choice of input distributions as these will impact on the predictions and inferences drawn. Previously published research,

expert opinion or use of experimental datasets can help to inform these distributions in terms of the type of distribution (for example uniform or Gaussian) and the ranges (Down et al., 2013, Østergaard et al., 2005).

1.7 Conclusions and aims

A review of the current literature highlights major knowledge gaps surrounding the risk factors for lameness and in particular those related to the development of CHDLs. The ‘laminitis’ hypothesis, which has historically been the predominant hypothesis for the pathogenesis of CHDLs, has been challenged more recently. Alternative hypotheses related to the increased risk of these lesions in high producing dairy cows and the timing of their occurrence after calving has led to recent research investigating the influence of BCS and sole soft tissue thickness on lameness. In addition it is apparent that pathological changes to the distal limb anatomy are associated with CHDL and lameness and therefore the potential importance of lameness in heifers is highlighted. As lameness in dairy cattle is a multifactorial disease statistical modelling that controls for confounding factors is critical. In addition, the longitudinal nature of the relationships being investigated frequently necessitates the use of multilevel modelling. It is increasingly recognised that in order to understand the complexities of biological systems, whether at a molecular level or at the level of the dairy herd, simulation-based approaches to modelling can provide a better representation of reality. By incorporating uncertainty, the dynamics of how changing certain factors will impact on lameness within the herd, can be explored. A greater understanding of the risk factors for lameness and quantifying their impacts at the herd level is critical if progress is to be made in controlling this disease. This is important from health and welfare perspectives, as well as for an economically and environmentally sustainable future for the dairy industry.

The overall aims of this thesis were to use statistical modelling to investigate risk factors for lameness with a particular focus on CHDLs; primarily BCS and early life events and evaluate their impacts at the herd level. A rich longitudinal dataset was used to investigate hypotheses formulated throughout this thesis. Chapter 2 describes the major data handling aspects including structuring and selection of data and the construction of categories for variables. The specific details that are relevant to individual chapters are discussed within respective chapters. Chapter 3 investigates the relationship between BCS, BW and lameness, exploring repeated lifetime events as well

as first lifetime events. An important aspect of this was to assess the relationship between BCS and first lifetime event where prior lameness events could not have a confounding effect. Chapter 4 explored the associations between hoof lesions occurring in early life around the time of first calving and future lameness, milk yield and culling. Chapter 5 and 6 aimed to use simulation-based models to quantify the impacts of BCS and previous lameness at the herd level and to then explore how different culling strategies may alter lameness levels in the herd. A summary of the overall conclusions and discussion is included as Chapter 7.

Furthering our understanding of the risk factors for lameness and CHDLs could help to identify where management interventions could be targeted to reduce the risk of lameness in dairy herds. Further to this, quantifying the contribution that risk factors make towards the total amount of lameness in herds is an important step forward from identifying risk factors; by identifying control measures that will have the most impact at the herd level. This is also important for informing where future research work should be targeted, as well supporting individual farm decision making in terms of resource allocation. Controlling lameness in dairy herds will help to achieve a sustainable dairy industry through improved health, welfare and production.

CHAPTER 2: General Introduction to the Study Herd, Data and Data Structuring

2.1 Introduction

The primary dataset used for this thesis was derived from the Crichton Royal research herd held at the Scotland's Rural College (SRUC) Dairy Research and Innovation Centre in Dumfries, Scotland. The SRUC centre has two pedigree research herds which are based at the same site; the 'Langhill' systems herd and 'Acrehead' herd. The Langhill herd were managed on a long-term genetic breeding and feeding system, with systematic recording of health, production and management data. Cows remained within the Langhill herd for typically 3 lactations, after which they were moved to Acrehead. The Acrehead herd was managed as a separate research and experimental herd with no long-running feed or management groups and data was not systematically recorded. This chapter describes the study herds as well as the process of data handling that was carried out to create the primary dataset that was used throughout this thesis. This process aimed to construct a dataset structured at the cow-week level (i.e. each cow had 421 weeks of records for the total study period), in order to conduct multilevel statistical analysis. Further details of data handling that are specific to individual chapters will be described within those chapters.

2.2 Study herd

2.2.1 Overview

Between 2002 and 2011 the Langhill herd was maintained as a closed herd with a replacement rate of approximately 25% and aiming for an average of 200 cows. The herd comprised Holstein Friesians managed on a long-term 2 x 2 factorial genetic and feeding system study; select (S) and control (C) genetic lines (as described in section 2.2.4); managed as one herd, divided equally into low-forage (LF) and high-forage (HF) groups. Heifers were served at approximately 15 months or 350 kg by artificial insemination; aiming for calving at 24 months of age. No oestrus synchronisation programme was used within the herd. Prior to the start of the transition period before first calving, heifers were separated according to the feeding system to which they had been allocated (LF or HF) for entry to the Langhill herd. The herd was all-year round calving and milked three times daily (0415 (am), 1245 (md) and 1945 (pm)) through a 14/14 herringbone parlour. It was intended that cows remain in the Langhill herd for an

average of 3 lactations before moving to the Acrehead herd, however if no replacement heifers were due to calve within 2 months, animals could remain at Langhill for one or more additional lactations (Roberts and March, 2013).

2.2.2 Lameness control

Footbathing was carried out at 3 consecutive milkings per week for lactating cows, monthly for young stock and weekly for dry cows using 5% copper sulphate solution. A professional foot trimmer attended bi-annually to trim the whole herd (all hind feet and front feet if they were considered overgrown). Cows considered lame (locomotion score (LS) 4 or 5 on a single occasion or two successive assessments of LS 3) were examined and treated by a veterinarian on a weekly basis prior to 2006 and every two weeks after this time. Acutely lame cows (LS 5) were treated by trained farm staff within 24 hours of observation.

2.2.3 Feeding system

LF cows were continuously housed and fed a complete diet containing between 45% and 50% forage in the dry matter (DM). HF cows were grazed during summer months provided sufficient grass was available and then housed during winter months (typically November to March) and fed a complete diet containing 70% to 75% forage in the DM. The forage component of the diet for both groups included grass silage, maize silage and whole crop wheat. Concentrates were included in the LF and HF diets at approximately 1,200 kg and 3,000 kg per cow per year respectively. Table 2.1 presents a summary of the target composition of the diets offered to the two groups. Target yields for LF and HF cows were 13,000 kg and 7,500 kg per cow per year respectively.

Table 2.1 Target composition for the diets offered to low forage and high forage groups at the Langhill herd at Scotland's Rural College (SRUC) Dairy Research and Innovation Centre.

Variable	Low forage			High forage		
	Average	Minimum	Maximum	Average	Minimum	Maximum
ME (MJ/kg of DM)	12.3	12.1	12.5	11.5	11.3	11.7
CP (g/kg of DM)	185	180	190	180	175	185
Oil (g/kg of DM)	60	55	65	50	45	55
Starch (g/kg of DM)	180	160	200	130	110	150
Sugar (g/kg of DM)	70	60	80	50	40	60
NDF (g/kg of DM)	345	330	360	390	360	420
DM (%)		45			30	

2.2.4 Genetic lines

S line cows were selected for maximum milk fat plus protein kilograms; cows were bred to bulls with the highest Predicted Transmitting Abilities (PTA) for kg fat plus protein yield in the UK. Whilst C line cows were maintained at the national average for milk fat plus protein; sires were of about UK average genetic merit for kg fat plus protein yield (Pryce et al., 1999). The genetic lines were established as a long-term experiment at the University of Edinburgh/Scottish Agricultural College Langhill Dairy Cattle Research Centre in 1973 to investigate the effect of selection on production, feed intake, health, fertility and profitability (Pryce et al., 1999). Heifers were selected mainly on the basis of their pedigree index ($0.50 \times \text{sire's PTA for fat plus protein plus } 0.25 \times \text{maternal grandsire's PTA for fat plus protein}$).

2.2.5 Housing and environment

All cows were housed in cubicle housing during winter months and had the potential to graze for varying period throughout summer months (Rioja-Lang et al., 2009). Housing was the same for cows in LF and HF groups; cubicles with mattresses installed in 2004 (mats were present prior to this) and sawdust bedding. Cubicles were 2.1m long and 1.1m wide with a slatted passageway between them, and a solid passageway adjacent to the feed face which was automatically scraped every 2 hours. Stocking density never exceeded one cow per cubicle and regularly ran at less than this.

Cow tracks were maintained with mainly crushed recycled concrete dressed with a layer of 10mm Whinstone Dust that was re-dressed every 2 years. A camber from the centre of the track encourages natural drainage. In some areas recycled plastic gridding filled with whinstone was used.

2.2.6 Data recording

Locomotion scores and body condition scores were collected weekly by experienced, trained assessors following standard protocols. Assessors alternated every week to reduce the impact of operator bias and regular training was carried out with the same veterinarian during the whole study period. Locomotion scores were measured using a 1 to 5 scale (LS 1 to 5) based on descriptions provided by Manson and Leaver (1988) (Table 2.2). BCS was measured using a 0 to 5 categorical scale with increments of 0.25 (Mulvany, 1977).

Body weights were recorded using an automatic weighing system (Stepmetric) after milking three times daily. Milk yield was recorded automatically three times daily. Milk composition was recorded weekly including somatic cell count, fat and protein content. All health, production and management data were recorded in a database.

Table 2.2 Descriptions of the locomotion scoring system used at Scotland's Rural College (SRUC) Dairy Research and Innovation Centre based on Manson & Lever (1988).

Locomotion score	Description	
	Manson & Lever	SRUC modification
1	Minimal ab/adduction, even gait	Even gait, tracking, no ab/adduction
1.5	Slight ab/adduction present, even gait	
2	Ad/adduction, uneven gait, perhaps tenderness of feet	Even gait, tracking, ab/adduction present
2.5	Ab/adduction, uneven gait, tenderness of feet	
3	Slightly lame, not affecting behaviour	Uneven gait, short strides
3.5	Obviously lame, difficulty turning, behaviour not affected	
4	Obviously lame, difficulty turning, behaviour affected	Lame
4.5	Some difficulty getting up walking, behaviour affected	
5	Difficult getting up, walking, behaviour adversely affected	Difficulty turning

2.3 Data selection and structuring

2.3.1 Introduction

Records were obtained for the period 1st September 2003 to 30th September 2011. This time period was selected as no major management changes to the herd had been implemented. The aim of the data selection and structuring process was to achieve a homogenous set of data for the herd over the defined time period, with minimal loss of data whilst ensuring data quality was maintained. An initial phase of exploration by scanning datasets and the use of descriptive statistics allowed selection criteria to be applied alongside assessment of missing data. This section describes the processes involved in constructing the final dataset that was used throughout this thesis.

2.3.2 Materials and methods

2.3.2.1 Data extraction

All records were obtained from a relational database accessed at SRUC. Initially data were exported as Comma Separated Values (CSV) files into Microsoft Excel (2013) or Microsoft Access (2013) with unique cow identification (eartag number) and date included in order to link data when constructing the final dataset. Table 2.3 summarises the initial data extracted including the type of data.

Table 2.3 Initial data extracted from the database at Scotland's Rural College (SRUC) Research and Innovation Centre for the period 1st September 2003 to 30th September 2011.

Dataset name	Field name	Data type	Description
BASIC LACTATION	EARTAG	Number	Unique cow ID
	LACT_NO	Number	Lactation number
	CALVING_DATE	Text	Date of calving
	LACT_START_DATE	Text	Date started that lactation
	DRYING_DATE	Text	Date of dry off
GENETIC GROUP	EARTAG	Number	Unique cow ID
	DOB	Text	Date of birth
	GENETIC_GROUP	Text	Genetic group (C or S)
	NO_OF_LACT	Number	Lactation number
FEED GROUP	EARTAG	Number	Unique cow ID
	LACT_NO	Number	Lactation number
	FEED_TYPE	Text	XE: Low forage, XM: High forage, DR: dry, NT: non-trial, AH: Acrehead, ZZ: hospital pen
	FEED_GROUP	Number	1: XE, 2: XM, 5: AH/non-trial
	START_DATE	Text	Date started in feed group
	STOP_DATE	Text	Date left feed group
MILK YIELD	EARTAG	Number	Unique cow ID
	LACT_NO	Number	Lactation number
	MILK_DATE	Text	Date milked including time
	YIELD_1	Number	Yield recorded at am milking
	YIELD_2	Number	Yield recorded at md milking
	YIELD_3	Number	Yield recorded at pm milking
CULLING	EARTAG	Number	Unique cow ID
	LACT_NO	Number	Lactation number
	CULLING_DATE	Text	Date culled
	CULLING_TYPE	Number	Reason for culling
	CULLING_TYPE2	Number	Additional reason for culling

BODY CONDITION SCORE	EARTAG	Number	Unique cow ID
	LACT_NO	Number	Lactation number
	BCS_DATE	Text	Date BCS recorded
	BCS	Number	BCS recorded
BODY WEIGHT	BCS_RECORDER	Number	Code to identify assessor
	EARTAG	Number	Unique cow ID
	LACT_NO	Number	Lactation number
	WEIGHT_DATE	Text	Date weighed including time
LOCOMOTION SCORE	LIVE_WGT	Number	Body weight recorded
	EARTAG	Number	Unique cow ID
	LACT_NO	Number	Lactation number
	LS_DATE	Text	Date locomotion score recorded
	LOCO_SCORE	Number	Locomotion score recorded
	LS_RECORDER	Number	Code to identify assessor

2.3.2.2 Data manipulation

Each dataset was individually explored in Microsoft Excel to identify missing data, impossible data or errors. Descriptive statistics were used to describe the distributions and aid in developing selection criteria where relevant. Data were manipulated to create a structure based on a cow-week format i.e. each cow had 421 weeks for the whole study period. A new column for EARTAG_WEEK was created for each dataset using the unique eartag cow identifier and the date of record/observation so that data could be linked. Summaries of the initial and final datasets are presented in Table 2.5 and Table 2.6.

Milk yield

Total daily yield (TDY) was calculated as a sum of the three values for milk yield recorded on a daily basis. A total of 538,142 records for TDY from 749 cows were included in the initial dataset. 3.3% of these were calculated from 2 or less records per day and these were recorded as missing data to avoid introducing error. An average daily yield for the week was then calculated from TDY. Where there were 3 or less TDY records within the week milk yield was recorded as missing for that week. 1.4% of average weekly TDY were missing and a further 3.7% were excluded as 3 or less records in the week; 75,638 average weekly TDY from 724 cows were available (3,927 missing records).

Body condition score

Data obtained for BCS was in 4 separate datasets depending on when BCS was recorded; weekly, at service, at dry-off and at calving. As these had been obtained using the same method of BCS scoring and by the same assessors all data was included. Duplicate records were removed (6,588 records). 79,543 records from 724 cows for BCS were available.

Bodyweight

Initial exploration of BW data highlighted erroneous records, for example the minimum BW recording was 100 kg, which was biological implausible for adult lactating cows. These were likely generated as a systematic error by the electronic weigh scales. Distribution of the data was normal, range was 100 kg to 990 kg (Figure 2.1). Both minimum and maximum values were examined and in communication with staff on the farm a minimum BW of 350 kg was defined as an acceptable cut-off for a biologically plausible weight. All data outside of these criteria were recorded as missing. The maximum bodyweight was deemed correct and therefore it was decided to keep all data in the upper ranges.

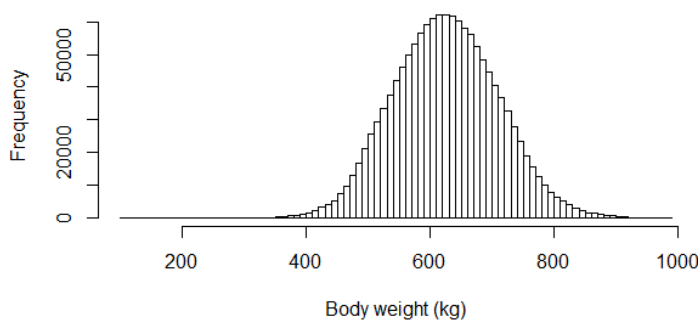


Figure 2.1 Frequency distribution for body weight (BW) from records obtained from the Scotland's Rural College (SRUC) Research and Innovation Centre.

An assumption was made that if the average dry matter intake (DMI) for a 650 kg cow was approximately 25 kg, BW may vary in the region of approximately 50 kg due to ingested feed within a day for an individual cow. BW may also vary with milking, however as all BW were recorded following milking, yield should not significantly

impact on variation in daily BW in individual animals. An average BW for 7 days' worth of recordings was calculated for each cow. The difference between each individual recording and this 7 day average was then calculated. A frequency distribution for the calculated differences was evaluated to determine how much variation from the 7 day average might be plausible for individual recordings (Figure 2.2). A BW of 40 kg appeared to be an appropriate threshold for the data, that was also biologically plausible. All BW records that were > 40 kg from the 7 day average were removed from the dataset.

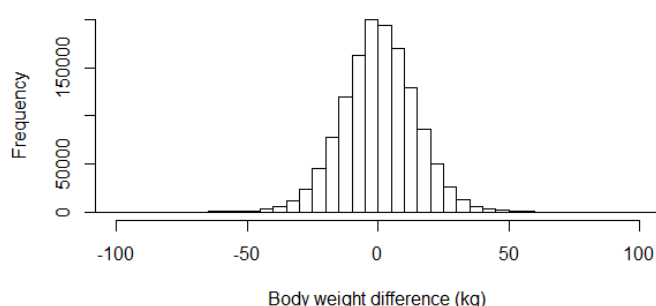


Figure 2.2 Frequency distribution for the difference between individual body weight (BW) recordings from the 7 day average BW for that cow.

As the number of recordings taken within the 7 days varied, a plot using count as an integer was used to show the variation of the weekly average recordings depending on the number of records within the 7 days (both including the data outside of the range of 40 kg difference from 7 day average and excluding this data). Figure 2.3 and Figure 2.4 show that including or excluding this data had little impact due to the number of cows outside of the range being very small. There was a larger degree of variation where there were ≤ 4 BW recordings over the 7 days per cow. Therefore this was an additional criteria applied that all records with cow weeks with ≤ 4 BW recordings over the 7 days were removed from the dataset.

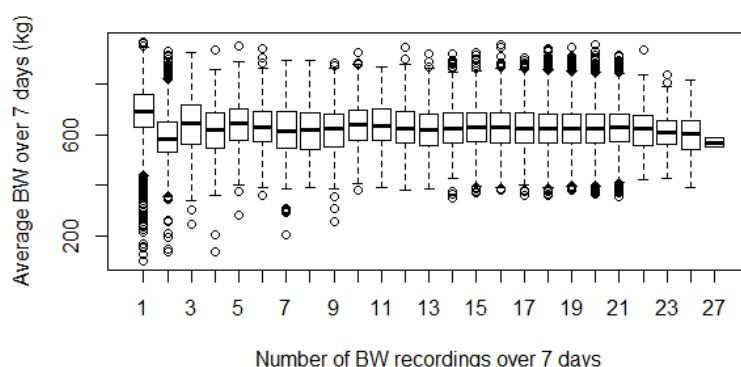


Figure 2.3 Box plot showing count of the number of body weight (BW) recordings over 7 days per cow against average weekly BW, with all data points > 40kg difference between test day BW and weekly average BW included. The median and interquartile ranges of average weekly BW are shown by the horizontal line and whiskers accordingly and outliers are shown by circles.

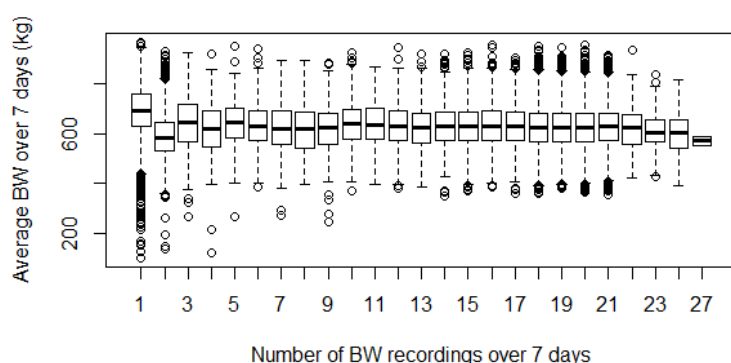


Figure 2.4 Box plot showing count of the number of BW recordings over 7 days per cow against average weekly BW, with all data points > 40kg difference between test day BW and weekly average BW removed. The median and interquartile ranges of average weekly BW are shown by the horizontal line and whiskers accordingly and outliers are shown by circles.

As there is no time influence on the BW of cows other than variation within the day, where there were missing records within the day (i.e. < 3 records/day) and within the week (i.e. < 7 days / week) data was still included. Frequency distributions for each of these criteria were also explored and used to justify this decision.

Average BW for the day for each cow was calculated and from this the daily average for the week was calculated generating 72,185 records. Table 2.4 summarises the number of records available for BW and those which met the selection criteria.

Table 2.4 Summary of body weight (BW) data available from records obtained from Scotland's Rural College (SRUC) Research and Innovation Centre.

Data	Number of records
Total BW records	1,341,163

Number of records not matching criteria set	41,786
% not matching criteria set	3.1%
Number of records for average daily BW	483,550
Number of records for average weekly BW	72,185

Locomotion score

84,079 records were extracted from the database of which 4202 were recorded as missing data. 3 records with values outside of the scoring system were removed, leaving 79,874 cow weeks with locomotion scores available. These scores included the full range from 1 to 5. Locomotion score was categorised as lame where $LS \geq 3$ (i.e. milder lameness as well as more severe lameness) or ≥ 4 (i.e. more severe lameness).

2.3.2.3 Final dataset

Separate datasets were combined in Microsoft Access to create one dataset including all available data structured at the cow-week level i.e. each cow had 421 week risk periods reflecting the duration of the study period. The minimum requirement for each row of data was a record for locomotion score as this was the primary outcome of interest. Table 2.5 summarises the data available before and after selection criteria were applied during the data handling process. Descriptive statistics were used to explore the final dataset, including visual inspection of frequency distributions to check for normality. Finally, data for BW, BCS and milk yield were lagged by 1 through to 16 weeks so that temporal associations could be investigated. Categories were also created for previous lameness; these are described further in individual chapters as well as categorisation of all other explanatory and outcome variables.

Table 2.5 Summary of data available before and after selection criteria applied during the data handling process for records obtained from the Scotland's Rural College (SRUC) Research and Innovation Centre.

Data set	Variable	Before selection criteria applied		After selection criteria applied		Summary	
		No. records	Range	No. records	Range	No. data points missing (%)	No. data points removed (%)
Milk Yield	TDY ¹	538,142	0.1 – 75.5	520,118	0.5 – 75.5	18,024 (3.3)	
	Av. weekly TDY	79,603	0.63-65.8	75,638	1.15-65.8	1,051 (1.4)	2,914 (3.7)
BCS ²	BCS	89,856	0.75-4.25	83,267	0.75-4.25	1	6,588 (7.3)
BW ³ (kg)	Av. daily BW	1,341,163	100-999	1,299,377	350-976		41,786 (3.1)
	Av. weekly BW			72,185	355.6 - 956.4		
LS ⁴	LS	84079	1-5	79874	1-5	4202 (5)	3 (0.0036)

¹TDY = Total daily yield

²BCS = Body condition score

³BW = Body weight

⁴LS = Locomotion score

2.4 Results

A total of 79,565 risk period weeks were available for 724 cows in the final dataset. A summary of the data available for the main variables of interest are described in Table 2.6. The main explanatory variables of interest (BW, BCS and milk yield) were considered approximately normally distributed on visual inspection of frequency distributions (Figure 2.5).

Table 2.6 Summary of the data available in the final dataset (79,565 records) for records obtained from a total of 724 cows.

	Lower quartile	Median	Mean	Upper quartile	Min	Max	No. missing records
Av. TDY (kg)	22.93	28.64	29.38	35.04	1.16	65.81	13,887
BCS	2.0	2.25	2.20	2.50	0.75	4.25	22
Av. BW (kg)	562.9	618.1	619.5	673.8	355.6	956.4	16,922
LS	2	2	2.17	2	1	5	0

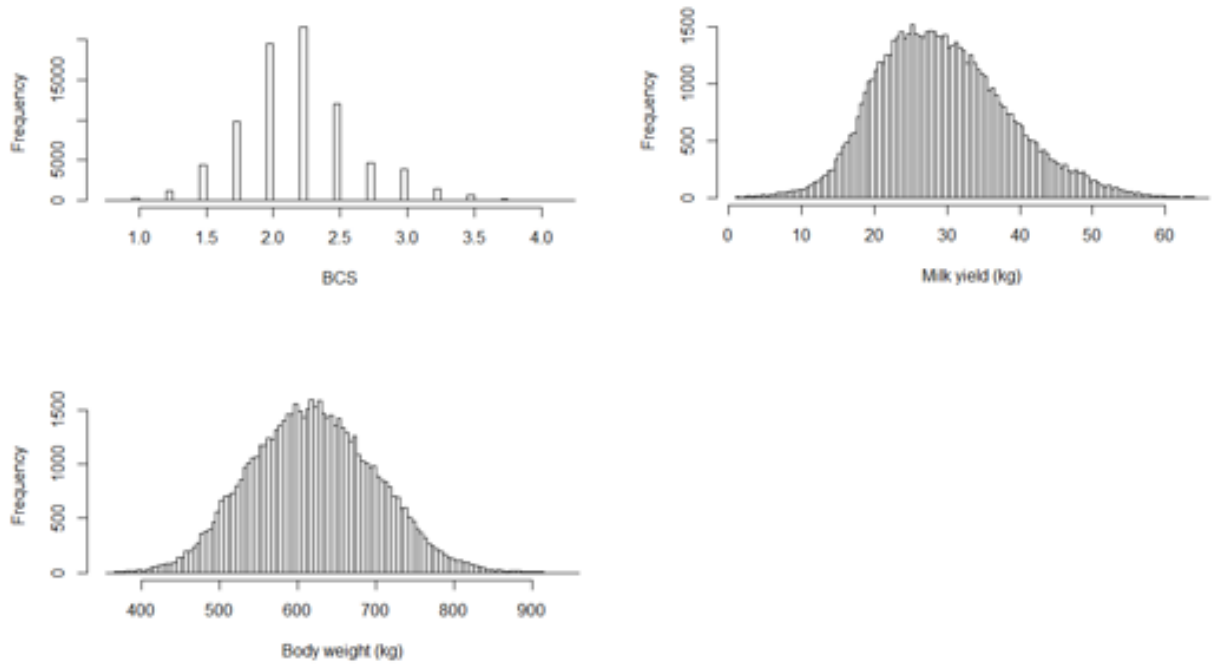


Figure 2.5 Frequency distribution for body condition score (BCS), milk yield (kg), and body weight (kg) data in the final dataset constructed.

There were a total of 17,114 lameness events (LS 3, 4 or 5); 8,799 of which were mildly lame (LS 3) and 8,315 of which were severely lame (LS 4 or 5). Frequency distribution for the number of lameness events per cow, defined as $LS \geq 3$ or ≥ 4 show that a small number of cows had a very high number of lameness events (Figure 2.6 and Figure 2.7, respectively). The effect of this on model fit is explored further in Chapter 4 (section 4.2.2) and Chapter 5 (section 5.2.2). The mean lameness prevalence (based on $LS \geq 3$) was 21.3% over the 421 weeks of the study period, ranging from 2.5% to 53.0% (Figure 2.8). Of the 724 cows included in the study, 93.0% had at least one or more locomotion score ≥ 3 and 83.7% had at least one or more locomotion score ≥ 4 .

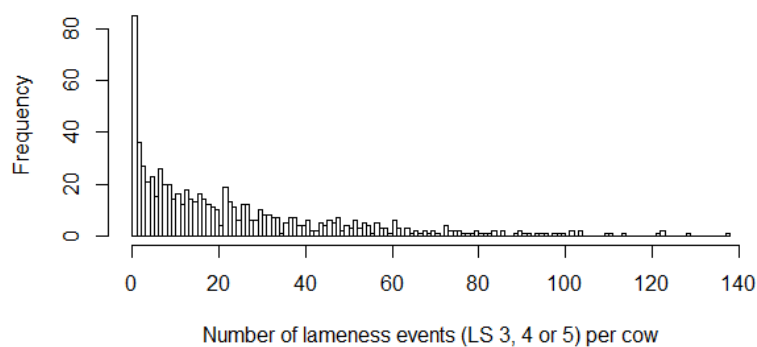


Figure 2.6 Frequency distribution for the number of lameness events per cow where lameness event was defined as locomotion score (LS) 3, 4 or 5.

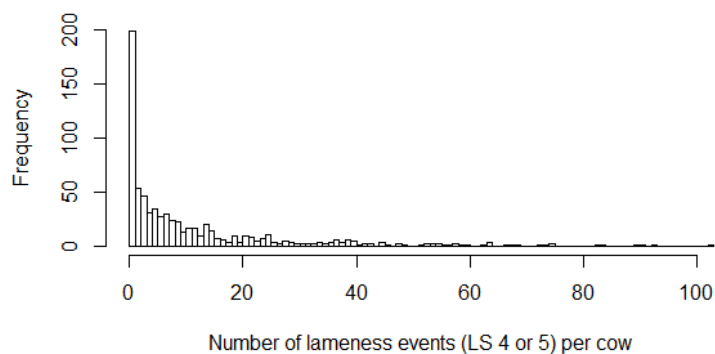


Figure 2.7 Frequency distribution for the number of lameness events per cow where lameness event was defined as locomotion score (LS) 4 or 5.

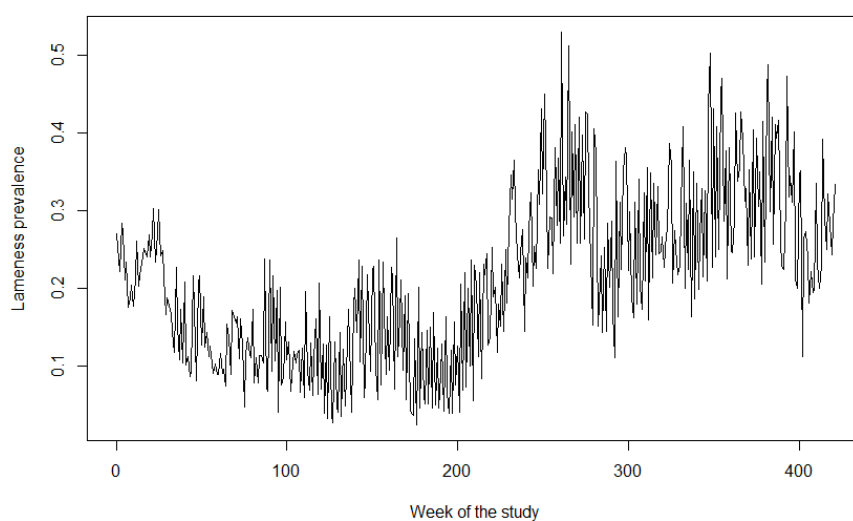


Figure 2.8 Lameness prevalence (based on locomotion score ≥ 3) over 421 weeks of the study period, 1st September 2003 to 30th September 2011.

Mean age at first calving for the herd over the 8 year period was 25.8 months (range, 20.5 to 37.8 months). The majority of cows were parity 1 to 3 during the study period; mean parity over the study period was 2.1, with 38% parity 1, 28% parity 2, 21% parity 3 and 13% parity 4+ cows. The proportion of cows with locomotion score 3 decreased with increasing parity, and the proportion of cows with locomotion score 4 and 5 increased with increasing parity.

2.5 Discussion

The retrospective data obtained from the SRUC Research and Innovation Centre enabled detailed analyses to be carried out using complex statistical methods due to the high number of data points and the number of parameters recorded. Although this data was collected from only one research herd where management differences may exist compared to commercial dairy herds (such as frequent assessments of locomotion score that may mean interventions are introduced sooner), a dataset of this detail and richness and over this period of time is currently not available from commercial herds, due to the resources required to collect and store this amount of data. As a rich dataset from one population, the results of statistical modelling conducted will be highly relevant to the study population. In terms of generalising findings to the target population, i.e. the national dairy herd, considerations need to be taken into account such as similarities in environmental and management factors compared with the study population. This data however provides an excellent research tool to investigate hypotheses formulated in this thesis.

Through the process of investigating the initial datasets extracted from the SRUC Research and Innovation Centre database a number of decisions were made regarding exclusion criteria. Although these were based on ensuring biological plausibility and inspection of the data using descriptive statistics, it is possible that this may introduce bias. For example, BW data was excluded based on several criteria (e.g. < 350 kg, < 4 BW recordings in one week and > 40 kg difference for individual records compared with the weekly average) and it is possible that one or more of these criteria may have selected for animals in the dataset that are more or less likely to become lame. As the proportion of records that were removed or recorded missing were very low this was considered unlikely. In addition, where possible, missing records remained within the final dataset so that categories for missing data could be created during the data analysis stages of each chapter with the aim of minimising loss of data. No thresholds

were used for the number of lameness events because these were deemed to be possible real events happening through time. However, during the modelling process, the effect of a high number of repeat lameness events were checked and explored to see if there was undue influence on model parameters. This is discussed in further detail in relevant chapters (Chapter 4 and 5).

Following on from this data handling process, Chapter 3 through to Chapter 6 will utilise this dataset to investigate a number of hypotheses, primarily focussed on evaluating the risk factors associated with BCS and early life events, for lameness in dairy cattle. Further data manipulation that was carried out, such as creating categories for explanatory variables and outcomes, specific to the hypothesis being tested are described in each of the individual chapters where relevant.

CHAPTER 3: Low Body Condition Predisposes Cattle to Lameness: An 8 Year Study of One Dairy Herd

3.1 Introduction

Lameness is considered by the dairy industry to be one of the most significant current challenges (Huxley, 2012). Extensive impacts on herd performance are reported, including milk yield losses (Green et al., 2002, Amory et al., 2008), increased culling (Booth et al., 2004), and impaired reproductive performance (Garbarino et al., 2004). The significance of these impacts extends beyond the financial implications. The importance of sustainable food production and welfare, and the potential effects of lameness on these are increasingly being recognised (Huxley, 2012, Herrero and Thornton, 2013). As a consequence of intensification of dairy production, there has been an associated increased risk of lameness (Huxley, 2012). The prevalence of lameness in high-producing, housed dairy herds have been reported at between 13% and 55% (Keyserlingk et al., 2012, Cook et al., 2016). In the UK, one estimate of incidence was approximately 50 cases/100 cows/year (Archer et al., 2010a).

A number of cow level factors have been associated with an increased incidence of lameness. Higher yielding cows are more likely to become lame whilst also still producing more milk over a lactation than cows that are never lame, even though lameness can result in 305-day yield losses in the region of 350 kg (Green et al., 2002, Bicalho et al., 2008, Archer et al., 2010b). Cows with low BCS ≤ 2 (on a scale 0 to 5) are more likely to be treated for lameness in the 2 or > 2 to 4 months following such a score (Green et al., 2014). Similarly, Lim et al. (2014) reported findings from a study demonstrating cows with low BCS (< 2.5) had a higher probability of becoming lame and lower probability of recovery compared to cows with BCS > 2 . It has been hypothesised that this association may be due to reduced digital cushion thickness which has been correlated with low BCS (Bicalho et al., 2009). The digital cushion is thought to function to dissipate concussive forces within the hoof during claw strike, therefore with a reduction in thickness damage to the corium may occur resulting in CHDLs.

The aim of the current chapter is to explore the hypothesis that cows in low body condition are at increased risk of lameness (measured as repeat locomotion scores) and identify associations between detailed BCS categories and the risk of lameness of

differing severity. An improved understanding of the role of BCS will help to support decision making in the control of lameness.

3.2 Materials and methods

3.2.1 Study herd and data

The study herd from which data were obtained and descriptions of the data are detailed in Chapter 2.

3.2.2 Statistical Analysis

3.2.2.1 General approach to modelling.

In addition to the data handling described in Chapter 2, outcome and explanatory variable categories were constructed. Locomotion score (LS) was the outcome variable and categorised as not lame (LS 1 or 2), mildly lame (LS 3) or severely lame (LS 4 or 5). Categories for the explanatory variables were explored and defined in terms of similarity in risk and number of data points within each category. Categorical explanatory variables tested included categories for parity, feed-genetic group (low forage-control: LF-C; low forage-select: LF-S; high forage-control: HF-C; high forage-select: HF-S; dry-control: D-C; dry-select: D-S; other-control: O-C; other-select: O-S), milk yield, age at first calving, BCS at calving, BCS change 0 to 4 weeks post-calving, BW, and BCS. Weeks in milk (WIM) was tested as an explanatory variable with polynomials up to the power 5. Week of the study was included as an explanatory variable with categories or polynomials to account for changes in background risk of lameness over time. Season was also tested as an explanatory variable (categorised as January to March, April to June, July to September and October to December). Categories for milk yield, BW and BCS were lagged by 2, 3, 4, 8, 12 and 16 weeks to explore their longitudinal association with lameness outcomes. Locomotion score and BCS assessor was included as an explanatory variable (Locomotion score recorder; 1 to 5) to control for possible inter-observer variability. Kendall's correlation coefficient (Kendall, 1955) was used to determine the correlation between BCS and BW.

Mixed effects hierarchical models were used to explore the relationship between explanatory variables and lameness outcomes. The models were constructed in MLWiN 2.28 (Rabash et al., 2009), and initially model parameters were estimated by the

iterative generalised least square procedure (Goldstein, 2003). Lagged yield, BW and BCS were explored systematically to identify the time periods that had significant impact on the response variable. Where lagged weeks were correlated, those with the largest effect size were left in the model. Final parameter estimates for each model investigated were made using Markov chain Monte Carlo (MCMC) to reduce biased estimates (Rabash et al., 2009). A minimum burn-in of 1,000 iterations was used during which model convergence occurred; these iterations were discarded to ensure confidence in the posterior distributions (Green et al., 2004). Parameter estimates were based on a further 50,000 iterations and chain mixing and stability were evaluated visually. Explanatory variables remained if the 95% credible interval of the odds ratio did not include 1 and as such these terms were considered ‘significant’.

Model fit was assessed by visual comparison of posterior predictions to observed data as described by Gelman et al. (1996). Where traditional methods, such as inspection of residual plots are useful for assessing model fit for linear regression model, these are considered inadequate for mixed effects logistic regression models (Green et al., 2009). Model fit was also explored using the Hosmer-Lemeshow (HL) test (Hosmer and Lemeshow, 1989) to assess goodness-of-fit; the χ^2 statistic was used to compare groups (for example deciles) of fitted risk values (i.e. posterior predictions at the repeated measures level) to the matched observed risk. A low p-value indicates poor model fit and vice-versa, although the number of groups can also influence this due to the number of predicted and observed events in each of the grouping. For this reason, a number of methods were used to assess model fit. Posterior predictions were also used to compare the predicted cow level lameness (total number of events) to the observed data.

3.2.2.2 Modelling

Modelling was conducted in two stages;

Stage 1: Multinomial model for repeated lameness events (Model 3.1). A mixed effects multinomial logistic regression model (Goldstein, 1995) with unordered categories was used to analyse the data as a frailty model, where each cow could have repeated lameness events over time. Cow was included as a random effect and time since the last lameness event as a fixed effect.

The model took the form:

$Lame_{ij} \sim \text{Multinomial}(\text{probability} = \pi_{ij})$

$\text{Log}(\pi_{1ij} / \pi_{0ij}) = \alpha + \beta_1 wk_{ij} + \beta_2 wk_{2ij} + \beta_3 wk_{3ij} + \beta_4 X_{ij} + \beta_5 X_j + v_{0j}$

$\text{Log}(\pi_{2ij} / \pi_{0ij}) = \alpha + \beta_1 wk_{ij} + \beta_2 wk_{2ij} + \beta_3 wk_{3ij} + \beta_4 X_{ij} + \beta_5 X_j + v_{1j}$

$\begin{bmatrix} v_{0j} \\ v_{1j} \end{bmatrix} \sim N(0, \Omega_v)$

Where subscripts i and j denote the i th observation of the j th cow respectively. π_{1ij} = probability of mildly lame outcome for the i th observation of the j th cow, π_{2ij} = probability of severely lame outcome for the i th observation of the j th cow. α = intercept value, wk_{ij} = week of the study for the i th observation of the j th cow, β_1 to β_3 = vector of coefficients for wk_{ij} , X_{ij} = the vector of covariates associated with each observation, β_4 = vector of coefficients for X_{ij} , X_j = vector of covariates associated with each cow, β_5 = the coefficients for covariates X_j . v_{0j} and v_{1j} were random effects to account for residual variation between cows (assumed to have a multivariate normal distribution of mean = 0 and variance-covariance matrix = Ω_v). Explanatory variables tested and the approaches to model building and fit are described above. Briefly, lagged (2 weeks to 16 weeks previously) BCS categories tested were; < 2, 2, 2.25, 2.5, 2.75, 3, > 3 and lagged (2 weeks to 16 weeks previously) BW categories tested were; < 550, 550 to 700, > 700 kg. The Pearson correlation coefficient was used to assess the amount of cow level variability explained by the model prediction (Dohoo et al., 2003).

Stage 2: Binomial models for survival to first lifetime lameness event (Models 3.2a, 3.2b, 3.3a and 3.3b). To allow previous BCS to be investigated without the confounding effect of a previous lame event, discrete time survival models were used with the outcome survival to a first lifetime lameness event for each cow.

Two different subsets of these data were used; i) first lifetime lameness events in heifers (first lactation) (Models 3.2a and 3.2b; first mild lameness event and severe lameness event respectively) ii) first lifetime lameness event in cows in their second lactation or greater (Models 3.3a and 3.3b; mild lameness event and severe lameness event respectively). For each subset of data, two separate models were constructed with

lameness outcomes categorised as mildly lame (LS 3), and severely lame (LS 4 or 5). The baseline for all models was cows that had not been lame. Once a cow had been recorded lame all subsequent records were removed and therefore only events prior to a first lameness case were modelled.

Models took the form:

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

$\text{Logit}(\pi_{ij}) = \alpha + \beta_1 \text{wk}_{ij} + \beta_2 \text{wk}_{2ij} + \beta_3 \text{wk}_{3ij} + \beta_4 X_{ij} + \beta_5 X_j + u_j$

$u_j \sim N(0, \sigma_v^2)$

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, wk_{ij} = week of the study for the i th observation of the j th cow, β_1 to β_3 = vector of coefficients for wk_{ij} , X_{ij} = the vector of covariates associated with each observation, β_4 = vector of coefficients for X_{ij} , X_j = vector of covariates associated with each cow, β_5 = the coefficients for covariates X_j . u_j = random effect to account for residual variation between cows (assumed to be normally distributed with mean = 0 and variance = σ_v^2). Explanatory variables tested and the approaches to model building and fit are described above. Briefly, lagged (2 weeks to 16 weeks previously) BCS categories tested were; < 2 , 2 , 2.25 , 2.5 , ≥ 2.75 (Models 3.2a and 3.2b) and < 2 , 2 , 2.25 , ≥ 2.5 (Models 3a and 3b). Lagged (2 weeks to 16 weeks previously) BW categories tested were; < 505 , 505 to 560 , > 560 kg (Models 3.2a and 3.2b) and < 600 , 600 to 650 and > 650 kg (Models 3.3a and 3.3b respectively). Biologically plausible interactions were explored including between BCS and BW.

3.3 Results

3.3.1 Stage 1: Multinomial model for repeated lameness events (Model 3.1)

The dataset included a total of 79,565 cow weeks at risk. There were 17,114 lameness events identified by weekly locomotion scoring; 8,799 of which were mildly lame (LS 3) and 8,315 BW of which were severely lame (LS 4 or 5). There were 79,543 and 62,643 recordings for BCS and weekly average BW respectively. The median BCS was 2.25 (range, 0.75 to 4.25) and mean ($\pm$ SD) BW was 619.5 ($\pm$ 80.5) kg (range, 355.6

to 956.4 kg). Mean age at first calving for the herd over the 8 year period was 25.8 months (range, 20.5 to 37.8 months). The majority of cows were parity 1 to 3 during the study period; mean parity over the study period was 2.1, with 38% parity 1, 28% parity 2, 21% parity 3 and 13% parity 4+ cows. The proportion of cows with locomotion score 3 decreased with increasing parity, and the proportion of cows with locomotion score 4 and 5 increased with increasing parity. The proportion of lame (mild and severe) observations in each lagged BCS category (3 weeks through to 16 weeks) had a trend of decreasing percentage of lame events from BCS < 2 to BCS >3, with BCS 2.5 having the lowest percentage (Table 3.1). BCS and BW were moderately correlated (kendalls tau = 0.26, $P \leq 0.05$), as also shown in Figure 3.1.

Table 3.1 Proportions of lame (mild and severe) observations in each lagged BCS category (3, 8, 12, and 16 weeks previously) for 724 cows.

BCS	Number of weeks previous to the risk period week							
	3 weeks		8 weeks		12 weeks		16 weeks	
	% Lame	% Not Lame	% Lame	% Not Lame	% Lame	% Not Lame	% Lame	% Not Lame
< 2	26.6 ¹	73.4	25.7	74.3	26.0	74.0	25.9	74.1
2	21.3	78.7	21.8	78.2	21.8	78.2	21.4	78.6
2.25	19.9	80.1	20.3	79.7	20.8	79.2	21.1	78.9
2.5	19.9	80.1	20.0	80.0	20.0	80.0	20.7	79.3
2.75	20.6	79.4	21.5	78.5	21.3	78.7	21.6	78.4
3	20.4	79.6	21.6	78.4	22.3	77.7	21.8	78.2
> 3	24.1	75.9	24.5	75.5	22.9	77.1	24.9	75.1

¹ For example, 26.6% of all the observations that had a BCS of < 2 three weeks previously were recorded as lame.

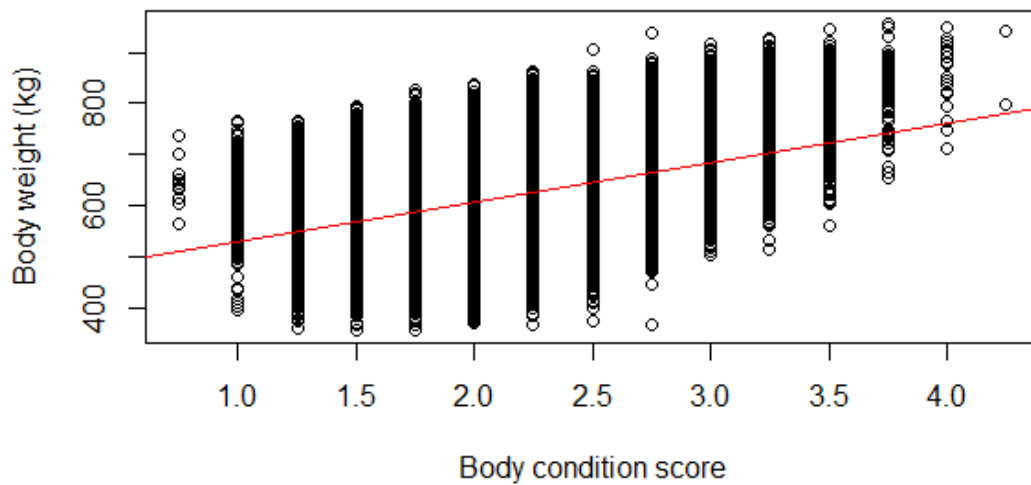


Figure 3.1 Scatter plot and regression line (red line) created using 'abline' function in R (R Core Team, 2016) for body weight and body condition scores for 724 cows.

Table 3.2 shows the results from Model 3.1. Lagged explanatory variables for BCS and BW were correlated from 2 through to 16 weeks. A significantly increased risk of mild or severe lameness was observed in cows with BCS < 2 three weeks prior to the lameness event compared with other BCS categories. Although there appeared to be a pattern of decreasing risk of lameness with increasing BCS, as illustrated in Figure 3.2, the odds ratios were similar for each BCS category, with overlapping credible intervals (Table 3.2), showing this increase in risk was not significantly different to the BCS category below (apart from BCS 2 versus < 2). Effect sizes for all BCS scores were larger for severely compared with mildly lame cows. Low BW cows were at significantly increased risk of mild or severe repeated lameness events compared with cows in heavier BW categories. Compared to the baseline category of < 550 kg, cows in the 550 to 700 kg category were less likely to become mildly lame (OR (95% credible interval) = 0.88 (0.81 to 0.97)) and severely lame (OR (95% credible interval) = 0.82 (0.74 to 0.92)). The risk of lameness decreased as BW increased, Figure 3.2. Interactions investigated were not found to be significant.

Age at first calving < 24 months was associated with the least risk of subsequently becoming being mildly lame or severely lame (OR (95% credible interval) = 0.77 (0.64 to 0.91) and 0.75 (0.56 to 0.99) respectively) when compared with a baseline of 24 to 27 months. Whilst cows with age at first calving > 33 months were at

greatest risk of subsequently being mildly lame or severely lame (OR (95% credible interval) = 2.14 (1.21 to 3.80) and 2.87 (1.16 to 7.12) respectively), as seen in Figure 3.2.

Cows losing body condition in the first 4 weeks after calving were at significantly higher risk of future lameness (OR (95% credible interval) = 1.21 (1.03 to 1.42) for severely lame cows) compared with a baseline of no change in BCS. This association was significant for severely lame cows only. Cows which had previously been lame (LS 3 to 5) were at higher risk of being mildly or severely lame. This effect was significant when lameness occurred any time between 1 and 16 weeks previously, with the previous 1 to 4 weeks having the largest effect (OR (95% credible interval) = 2.09 (1.96 to 2.23) and 3.85 (3.56 to 4.18)). A previous lameness event > 16 weeks previously also increased the risk of severe lameness.

Higher parity cows were at greater risk of lameness compared with parity 1 cows; parity 4+ OR (95% credible interval) = 2.09 (1.82 to 2.41) and 4.93 (4.14 to 5.88) for mildly and severely lame categories respectively. With LF:C as the baseline category, grazed cows (HF:S and HF:C) were significantly less likely to be severely lame (OR (95% credible interval) = 0.73 (0.60 to 0.89) and 0.67 (0.53 to 0.86) for HF:C and HF:S categories respectively). HF:S cows were significantly less likely to be mildly lame (OR (95% credible interval) = 0.69 (0.59 to 0.82)). Cows with lower yields 3 weeks previously and cows with higher yields 16 weeks previously were at significantly greater risk of severe lameness. Season was not found to be significant; likely due to week of the study being included as an explanatory variable and therefore correcting for any seasonal effect.

The Pearson correlation coefficient (r^2) was estimated from Model 3.1 was 0.62 and 0.60 for mildly and severely lame outcomes respectively.

Table 3.2 Model 3.1: Final multinomial model of risk of repeated lameness events for 724 cows with outcomes mildly lame and severely lame^{1,2}

Variable	Not lame vs. mildly lame (Model 3.1)				Not lame vs. severely lame (Model 3.1)			
	Coefficient: 1.92; SE: 0.30				Coefficient: 3.62; SE: 0.29			
	No. Lam ³	Odds ratio	Lower 95% CrI	Upper 95% CrI	No. Lam ³	Odds ratio	Lower 95% CrI	Upper 95% CrI
Weeks ⁴								
Week^1		0.98	0.97	0.98		0.98	0.98	0.99
Week^2		1.00	1.00	1.00		1.00	1.00	1.00
Week^3		1.00	1.00	1.00		1.00	1.00	1.00
Weeks in milk (WIM) ⁴								

WIM^1		1.00	1.00	1.01		1.02	1.01	1.03
WIM^2		1.00	1.00	1.00		1.00	1.00	1.00
Parity								
1	2,916	Baseline			2,179	Baseline		
2	2,342	1.13	1.05	1.23	1,960	1.49	1.36	1.65
3	2,293	1.57	1.42	1.73	2,273	2.58	2.27	2.93
4+	1,248	2.09	1.82	2.41	1,903	4.93	4.14	5.88
Feed-genetic group ⁵								
LF:C	2,061	Baseline			1,877	Baseline		
LF:S	1,889	1.01	0.86	1.81	1,755	1.13	0.90	1.43
HF:C	1,664	0.87	0.76	1.00	1,064	0.73	0.60	0.89
HF:S	1,589	0.69	0.59	0.82	1,342	0.67	0.53	0.86
Dry:C	428	0.77	0.66	0.89	356	0.65	0.54	0.77
Dry:S	390	0.76	0.63	0.93	324	0.61	0.47	0.79
Other:C	68	1.16	0.84	1.61	167	3.45	2.60	4.57
Other:S	51	1.25	0.87	1.80	221	4.59	3.34	6.30
Age at first calving (months)								
24-27	803	Baseline			635	Baseline		
< 24	5,951	0.77	0.64	0.91	5,279	0.75	0.56	0.99
28-30	956	1.48	1.18	1.84	775	775	0.94	2.00
31-33	150	1.08	0.67	1.74	76	0.88	0.39	1.97
> 33	133	2.14	1.21	3.80	171	2.87	1.16	7.12
Locomotion score recorder ⁶								
1	1,537	Baseline			699	Baseline		
2	1,867	0.22	0.20	0.23	4,296	0.94	0.84	1.04
3	3,010	0.89	0.82	0.98	2,203	0.95	0.85	1.06
4	2,296	0.81	0.74	0.88	1,093	0.91	0.81	1.02
5	89	1.10	0.83	1.47	24	0.53	0.33	0.86
Previous lameness (LS 3,4 or 5)								
None		Baseline				Baseline		
1 to 4 wks	5,857	2.09	1.96	2.23	6,338	3.85	3.56	4.18
5 to 8 wks	5,429	1.55	1.46	1.65	5,686	1.77	1.64	1.91
9 to 12 wks	5,089	1.25	1.17	1.33	5,243	1.28	1.19	1.38
13 to 16 wks	4,846	1.24	1.16	1.32	4,819	0.99	0.92	1.06
> 16 weeks	2,58	1.00	0.72	1.39	389	1.35	1.00	1.82
BCS 3-weeks previously								
< 2	1,963	Baseline			1,965	Baseline		
2	2,086	0.91	0.84	0.98	1,884	0.80	0.73	0.87
2.25	2,130	0.90	0.82	0.98	1,994	0.73	0.67	0.80
2.5	1,208	0.90	0.81	1.00	1,075	0.63	0.56	0.71
2.75	464	0.82	0.71	0.94	439	0.64	0.55	0.74
3	385	0.80	0.69	0.94	352	0.61	0.51	0.72
> 3	248	0.79	0.65	0.95	246	0.69	0.56	0.84
BW 3-weeks previously (kg)								
< 550	1,491	Baseline			1,119	Baseline		
550 - 700	4,156	0.88	0.81	0.97	4,206	0.82	0.74	0.92
> 700	1,159	0.84	0.74	0.97	1,232	0.72	0.62	0.84
Milk yield 3-weeks previously (kg)								

< 12	105	Baseline				109	Baseline		
12 – 24	2,076	0.87	0.68	1.11	} *	1,884	0.82	0.63	1.07
25 – 37	3,536	0.81	0.63	1.05		3,258	0.68	0.52	0.89
38 – 50	1,312	0.81	0.63	1.06		1,239	0.62	0.47	0.83
> 50	227	0.81	0.59	1.11		180	0.47	0.33	0.66
Milk yield 16-weeks previously (kg)									
< 12	75	Baseline				44	Baseline		
12 – 24	1,784	0.95	0.72	1.26	} *	1,515	1.49	1.03	2.15
25 – 37	3,324	0.98	0.75	1.30		3,118	1.58	1.09	2.29
38 – 50	1,159	0.95	0.71	1.26		1,262	1.66	1.14	2.42
> 50	190	0.92	0.66	1.29		203	1.49	0.97	2.26
BCS change 0 to 4 weeks post-calving									
No change	605	Baseline				491	Baseline		
Loss $\geq$ 0.25	1,600	1.13	0.98	1.30	} *	1,462	1.21	1.03	1.42
Gain $\geq$ 0.25	236	0.91	0.73	1.12		256	1.15	0.90	1.46
Random effects	Variance (σ_0): 0.45		Standard error: 0.04			Variance (σ_1): 0.58		Standard error: 0.05	
	Covariance ($\sigma_{0,1}$):1.36		Standard error: 0.10						

¹Table 3.2 refers to the single multinomial model (Model 3.1) comparing two outcomes (mildly lame and severely lame). CrI = credible interval.

²Some nonsignificant results are shown as they represent part of the multinomial model (e.g. results marked *).

³Number of lameness events in each category.

⁴Explanatory variables weeks (week of the study) and WIM included as a polynomial to power 3 ($\wedge 3$) and 2 ($\wedge 2$).

⁵Feed-genetic groups include low forage (LF), high forage (HF), control (C), and select (S). Dry refers to dry cows, and other refers to all other management groups outside of LF, HF, and dry.

⁶Locomotion score recorder = individual locomotion score/BCS observer identification.

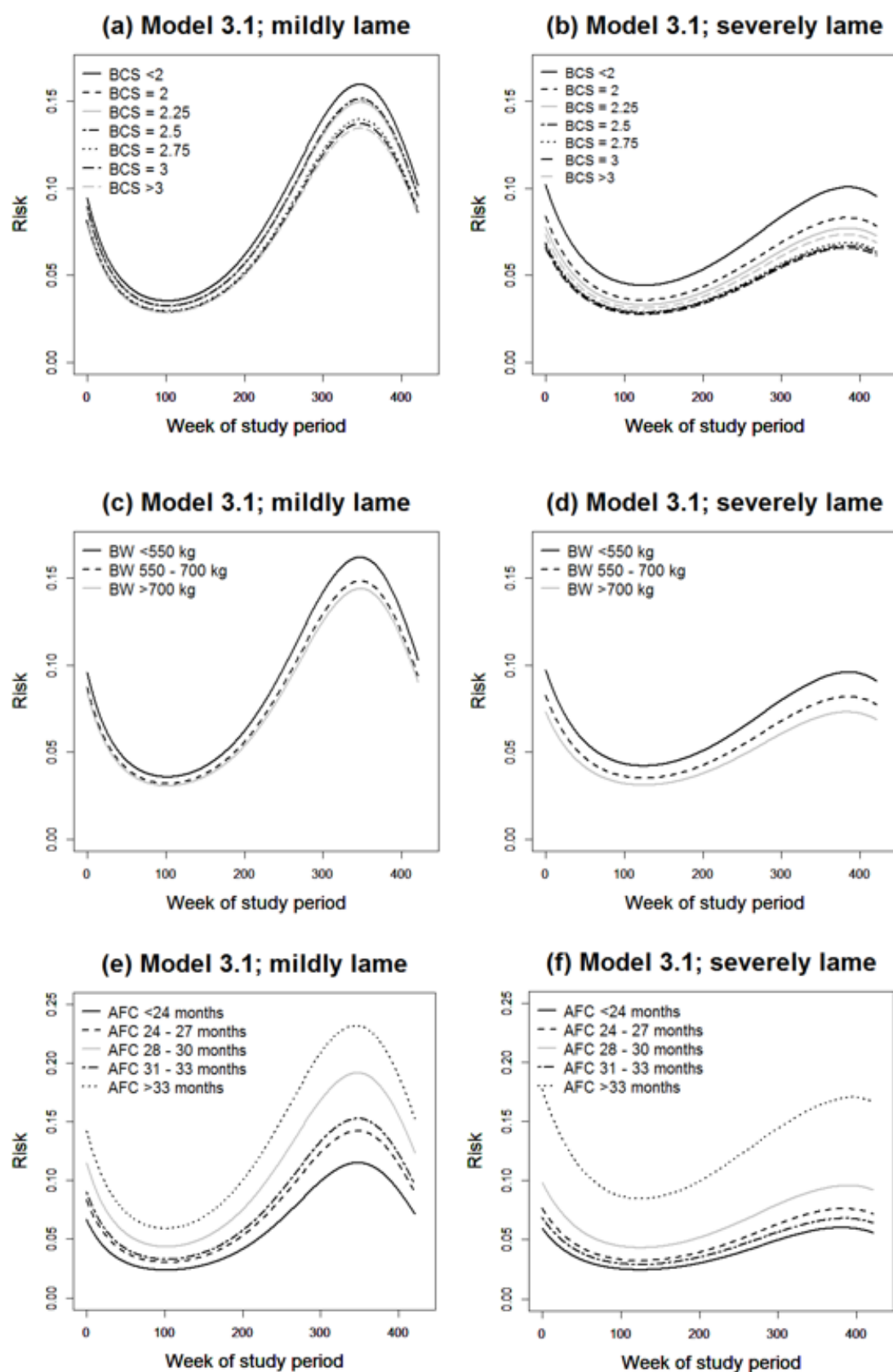


Figure 3.2 Median predicted risk of lameness from multinomial model parameters (model 3.1) for 724 cows. (a) Shows the median predicted risk for BCS categories 3 weeks previously with the outcome mildly lame and (b) severely lame. (c) Shows the median predicted risk for BW categories 3 weeks previously with the outcome mildly lame and (d) severely lame. (e) Shows the median predicted risk for age at first calving categories with the outcome mildly lame and (f) severely lame.

Model fit for the multinomial model was assessed by comparing posterior predictions to observed risk. The majority of observed data points were within the 95% credible interval of posterior predictions (Figure 3.3 and Figure 3.4). The model appeared to under-predict for the outcome $LS \geq 4$. This may be because the categories or values for parameters in the model being used for the posterior predictions were not representative of the mean effect. To test this, different categories/values were selected for two of the parameters (parity and weeks in milk; parity = 4 and weeks in milk = 20). When these categories/values were used the posterior predictions fitted the observed data better (Figure 3.5). This was repeated for the outcome $LS = 3$ (Figure 3.6) and model fit also improved. Overall, model fit was considered good.

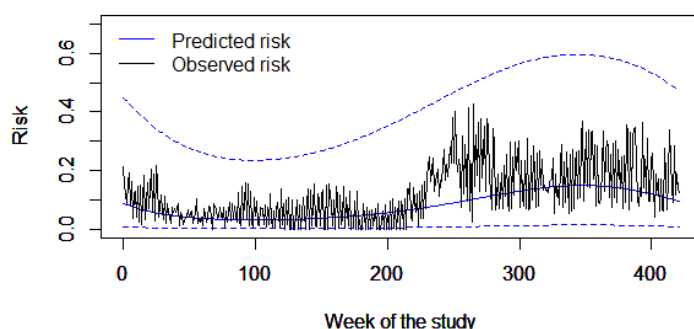


Figure 3.3 Predicted risk for repeated lameness events ($LS = 3$) using observed and predicted data including 95% credible intervals for level 3 (cow) (dashed blue line).

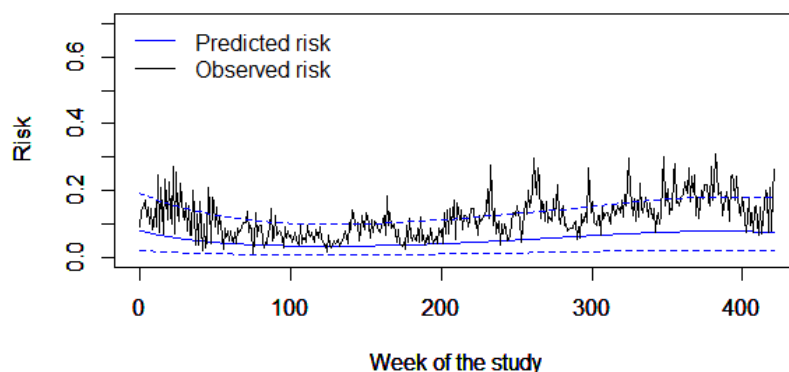


Figure 3.4 Predicted risk for repeated lameness events ($LS \geq 4$) using observed and predicted data including 95% credible intervals for level 3 (cow) (dashed blue line).

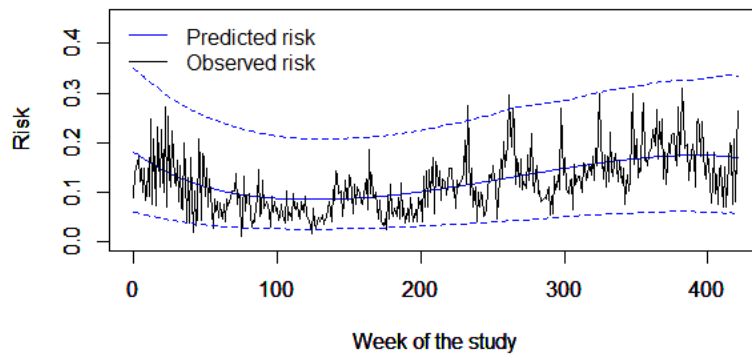


Figure 3.5 Predicted risk for repeated lameness events ($LS \geq 4$) using observed and predicted data including 95% credible intervals for level 3 (cow) (dashed blue line). Predictions were made for weeks in milk = 20 and parity = 4.

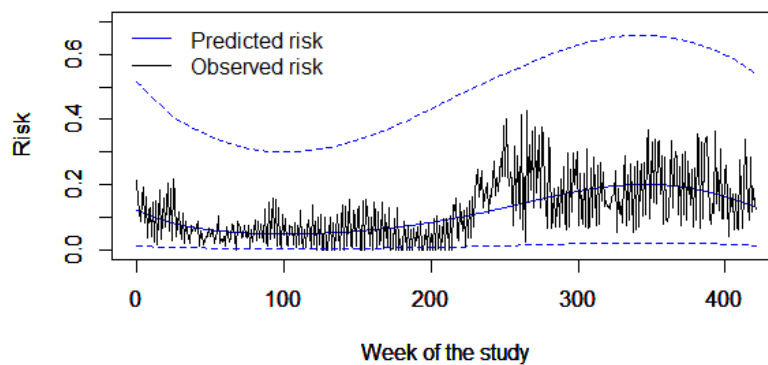


Figure 3.6 Predicted risk for repeated lameness events ($LS = 3$) using observed and predicted data including 95% credible intervals for level 3 (cow) (dashed blue line). Predictions were made for weeks in milk = 20 and parity = 4. Stage 2: Binomial models for survival to first lifetime lameness event (Models 3.2a, 3.2b, 3.3a and 3.3b)

3.3.2 Stage 2: Binomial models for first lifetime lameness events (Model 3.2a, 3.2b, 3.3a and 3.3b)

Datasets used in the analysis for first lifetime lameness events, Models 3.2a and 3.2b, included 4,983 (375) and 3,070 (252) cow weeks at risk (number of cows) respectively. There were 333 first lifetime lameness events with the outcome mildly lame and 211 with the outcome severely lame. Results from Models 3.2a and 3.2b are summarised in Table 3.3. $BCS \geq 2.75$ two weeks previous to a first mild lame event was associated with the lowest risk of lameness compared to the baseline $BCS < 2$ (OR (95% credible interval) = 0.40 (0.19 to 0.85)). This association was not significant

for severe lameness. Other variables tested including BW, BCS at calving, BCS change 0 - 4 weeks post-calving and milk yield were not significant. Season was not found to have a significant effect in Model 3.2a but was found to in Model 3.2b. This may be due to the week categories in Model 3.2a controlling for season effect, but there not being sufficient week categories to control for this effect in Model 3.2b. Based on visual comparison of posterior predictions compared with observed number of lameness events in each week category (Figure 3.7 and Figure 3.8) and the HL test; $\chi^2 = 0.33$, $p = 1.0$ and $\chi^2 = 0.80$, $p = 0.94$ for Models 3.2a and 3.2b respectively, model fit was considered to be good.

Datasets used in the analysis for first lifetime lame events in second or greater lactation animals (i.e. excluding parity 1 cows), Models 3.3a and 3.3b, included 1,961 (91) and 1,503 (59) cow weeks at risk (number of cows) respectively. There were 81 first lifetime lameness events with the outcome mildly lame and 49 with the outcome severely lame. Results from Models 3.3a and 3.3b are summarised in Table 3.4. Lagged explanatory variables for BCS were correlated and significantly associated with risk of lameness at 12 and 16 weeks previously for first mild lameness event and 8 and 12 weeks previously for first severe lameness event. Cows with $BCS \geq 2.5$ 16 weeks previous to a mild lameness event were at reduced risk of lameness compared to the baseline ($BCS < 2$) with OR (95% credible interval) = 0.25 (0.07 to 0.91). Cows with $BCS = 2.25$ 8-weeks previous to a severe lameness event were at reduced risk of lameness compared to the baseline (OR (95% credible interval) = 0.23 (0.05 to 0.96)). HF:S cows were at significantly less risk of a first mild lameness event in second lactation and greater cows compared with the baseline category of LF:C (OR (95% credible interval) = 0.14 (0.02 to 0.88)). Season was significant for Model 3.3a, but was not found to be significant for Model 3.3b. This may be due to loss of power with smaller sample sizes in Model 3.3b such that a significant effect could not be identified or that there was no significant effect of season on this outcome. Other variables tested were not significant. Based on visual comparison of the posterior predictions and observed number of lameness events for Model 3.3a and 3.3b (Figure 3.9 and Figure 3.10) and the HL test; $\chi^2 = 0.29$, $p = 0.96$ and $\chi^2 = 0.28$, $p = 0.87$ respectively, model fit was considered good. Week categories were grouped to ensure the observed and expected frequencies were adequate for the HL test (i.e. not < 1 and not less than 5 in $> 20\%$ of the categories).

Table 3.3 Models 3.2a and 3.2b: Final binomial models of risk of first lifetime lameness event in heifers (first lactation) with outcomes mildly lame (Model 3.2a) and severely lame (Model 3.2b)

Not lame vs mildly lame (Model 3.2a)					Not lame vs severely lame (Model 3.2b)				
Intercept	Coefficient: -2.25		Standard error: 0.59		Coefficient: -3.49		Standard error: 1.55		
Variable	No. Lam ¹	Odds ratio	Lower 95% CrI	Upper 95% CrI	Variable	No. Lam ¹	Odds ratio	Lower 95% CrI	Upper 95% CrI
Week category									
0 – 60	50	Baseline			0 – 60	33	Baseline		
61 – 120	26	0.83	0.33	2.08	61 – 120	23	0.61	0.08	4.89
121 – 180	30	0.50	0.20	1.22	121 – 180	23	0.25	0.03	2.43
181 – 240	53	0.72	0.32	1.64	181 – 240	38	0.49	0.05	4.74
241 – 300	57	2.78	1.11	6.96	241 – 421	94	8.81	0.85	91.49
301 – 360	54	1.81	0.70	4.66					
361 – 421	63	2.21	0.89	5.48					
					Season				
					Jan – Mar	68	Baseline		
					Apr – Jun	54	0.54	0.23	1.24
					Jul – Sept	50	0.23	0.08	0.67
					Oct – Dec	39	0.24	0.09	0.66
Weeks in milk (WIM) ³									
WIM ^{^1}		1.16	1.06	1.26	WIM ^{^1}		1.43	1.18	1.74
WIM ^{^2}		1.00	0.99	1.00	WIM ^{^2}		1.00	0.99	1.00
WIM ^{^3}		1.00	1.00	1.00	WIM ^{^3}		1.00	1.00	1.00
Feed-genetic group ⁴									
LF:C	79	Baseline			LF:C	50	Baseline		
LF:S	79	0.95	0.50	1.36	LF:S	47	0.15	0.02	1.02
HF:C	80	0.92	0.49	1.73	HF:C	40	0.65	0.11	3.83
HF:S	67	0.77	0.39	1.52	HF:S	35	0.06	0.01	0.49
Dry	11	4.62	1.51	14.14	Dry & other	28	11.73	2.30	59.90
Other	3	0.92	0.15	5.77					
Locomotion score recorder ⁵									
1	51	Baseline			1	10	Baseline		
2	84	0.25	0.14	0.42	2	112	1.51	0.41	5.47
3	142	0.71	0.40	1.23	3	72	1.16	0.31	4.31
4	52	0.87	0.47	1.63	4	17	0.82	0.18	3.70
5	4	0.54	0.12	2.39					
BCS 2 weeks previously									
<2	41	Baseline			<2	10	Baseline		
2	62	0.44	0.27	0.74	2	33	0.66	0.22	1.96
2.25	87	0.45	0.27	0.76	2.25	51	1.11	0.37	3.34
2.5	45	0.43	0.23	0.81	2.5	31	1.16	0.34	3.98
≥ 2.75	25	0.40	0.19	0.85	≥ 2.75	10	0.82	0.22	1.96
Random effect	Variance (σ ₀)	Standard error				Variance (σ ₀)	Standard error		
	3.22	1.28				22.92	11.92		

¹Number of lameness events in each category. CrI = credible interval.

³Explanatory variable weeks in milk (WIM) included as a polynomial to the power 3 (^3).

⁴Feed-genetic groups include low forage (LF), high forage (HF), control (C) Feed-genetic groups include low forage (LF), high forage (HF), control (C), and select (S). Dry refers to dry cows and other refers to all other management groups outside of LF, HF and dry.

⁵Locomotion score recorder = individual locomotion score/BCS observer identification.

Table 3.4 Models 3.3a and 3.3b: Final binomial models of risk of first lifetime lameness event in cows second lactation or greater with outcomes mildly lame (Model 3.3a) and severely lame (Model 3.3b).

Not lame vs mildly lame (Model 3.3a)					Not lame vs severely lame (Model 3.3b)				
Intercept	Coefficient: 1.90		Standard error: 1.23		Coefficient: -2.01		Standard error: 1.91		
Variable	No. Lam ¹	Odds ratio	Lower 95% CrI	Upper 95% CrI	Variable	No. Lam ¹	Odds ratio	Lower 95% CrI	Upper 95% CrI
Week category									
0 – 60	53	Baseline			0 – 120	34	Baseline		
61 – 180	8	0.11	0.02	0.54	121 - 180	8	1.74	0.33	9.26
181 – 240	9	0.25	0.05	1.27	181 - 421	7	2.67	0.31	23.13
241 – 421	11	0.70	0.08	6.12					
Week 0 ²									
No	62	Baseline			No	43	Baseline		
Yes	19	12.16	3.26	45.38	Yes	6	1.54	0.22	10.64
Season									
Jan – Mar	19	Baseline							
Apr – Oct	41	0.27	0.10	0.72					
Nov - Dec	21	0.62	0.23	1.68					
Weeks in milk (WIM) ³									
WIM ⁴		0.97	0.81	1.15			0.90	0.76	1.08
WIM ²		1.00	0.99	1.01			1.00	1.00	1.01
WIM ³		1.00	1.00	1.00			1.00	1.00	1.00
Feed-genetic group ⁴									
LF:C	11	Baseline			LF	7	Baseline		
LF:S	10	0.78	0.13	4.63	HF	12	0.73	0.09	5.89
HF:C	11	0.32	0.06	1.60	Dry	5	1.36	0.08	24.36
HF:S	6	0.14	0.02	0.88					
Dry	3	0.75	0.09	6.41					
Locomotion score recorder ⁵									
1	6	Baseline			1	1	Baseline		
2	35	0.11	0.02	0.59	2	37	0.67	0.04	12.44
3	35	0.16	0.03	0.86	3	11	0.12	0.01	2.53
4	5	4.34	0.44	42.96					
BCS 16-weeks previously					BCS 8-weeks previously				
< 2	10	Baseline			< 2	7	Baseline		
2	10	0.39	0.13	0.96	2	5	0.38	0.09	1.73
2.25	11	0.59	0.19	1.80	2.25	5	0.23	0.05	0.96
≥ 2.5	10	0.25	0.07	0.91	≥ 2.5	10	0.67	0.12	3.70
Random effects	Variance (σ_0)		Standard error		Variance (σ_0)		Standard error		
	2.84		1.92		6.57		3.62		

¹Number of lameness events in each category. CrI = credibly interval.

²Explanatory variable as a binary outcome for week = 0.

³Explanatory variable weeks in milk (WIM) included as a polynomial to the power 3 (³).

⁴Feed-genetic groups include low forage (LF), high forage (HF), control (C) Feed-genetic groups include low forage (LF), high forage (HF), control (C), and select (S). Dry refers to dry cows.

⁵Locomotion score recorder: individual locomotion score/body condition score observer identification.

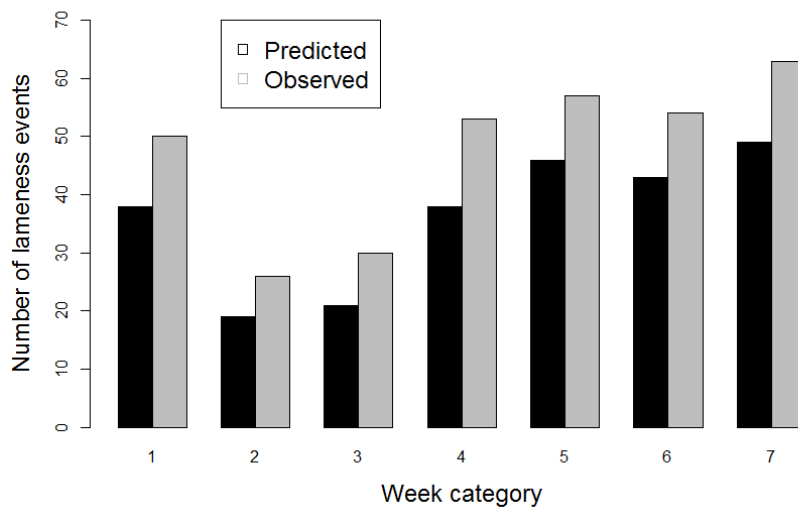


Figure 3.7 Observed and predicted number of first lifetime lameness events (LS = 3) in heifers. Where week category 1 to 7 represent weeks of the study in 60 week blocks from 0 to 421 weeks. Posterior predictions were from Model 3.2a.

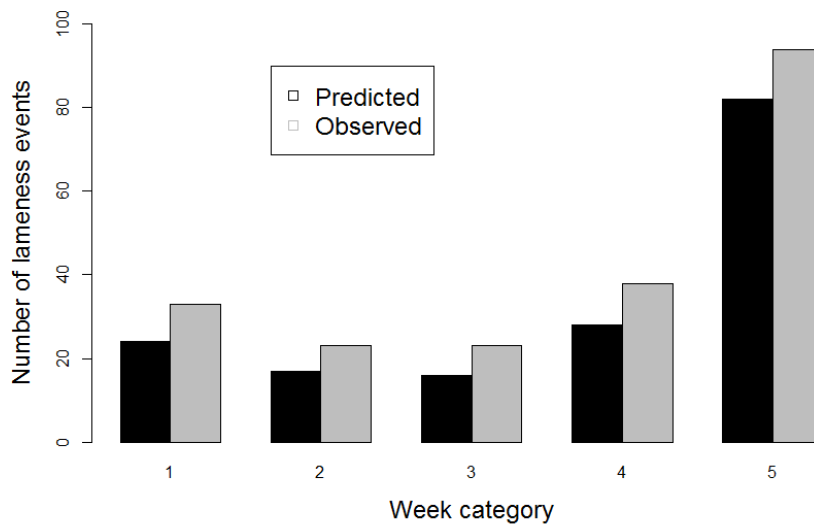


Figure 3.8 Observed and predicted number of first lifetime lameness events (LS ≥ 4) in heifers. Where week category 1 to 5 represent weeks of the study in 60 week blocks from 0 to 240 and one block from 241 to 421 weeks. Posterior predictions were from Model 3.2b.

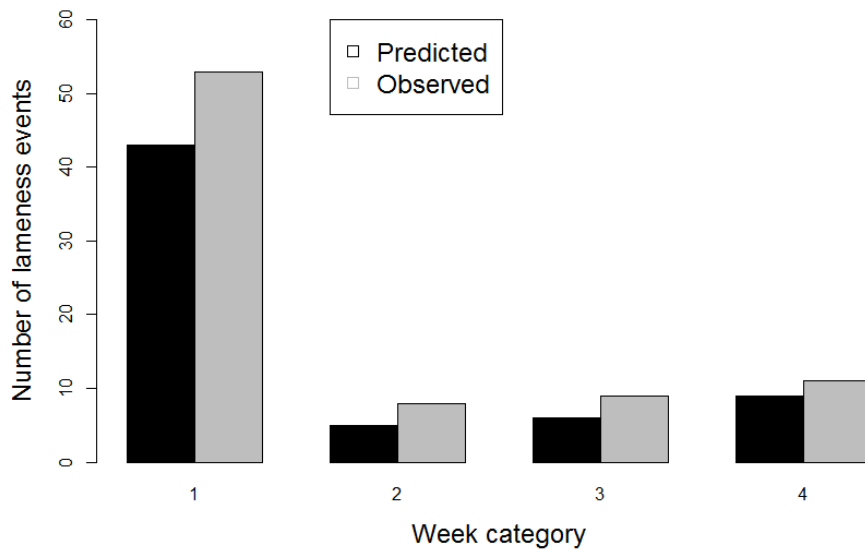


Figure 3.9 Observed and predicted number of first lifetime lameness events (LS= 3) in cows 2nd lactation and greater. Where week category 1 to 4 represent weeks of the study; 1: 0 to 60, 2: 61 to 120, 3: 121 to 240, 4: 241 to 421. Posterior predictions were from Model 3.3a.

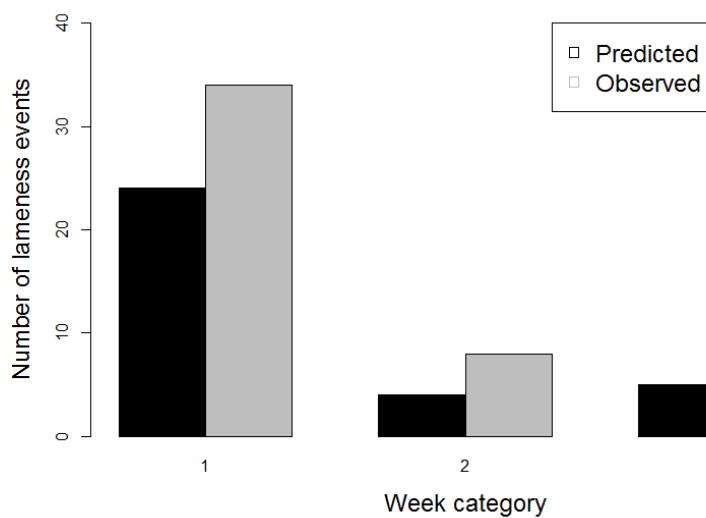


Figure 3.10 Observed and predicted number of first lifetime lameness events (LS ≥ 4) in cows 2nd lactation and greater. Where week category 1 to 3 represents weeks of the study; 1: 0 to 120, 2: 121 to 180, 3: 181 to 421. Posterior predictions were from Model 3.3b.

3.4 Discussion

This work describes the analysis of a rich longitudinal data set, investigating risk factors for both repeated lameness events and first lifetime lameness event. Whilst previous papers have described low body condition score as a risk factor for future lameness, this is the first research carried out to identify fine thresholds of individual BCS as predictors for mild and severe lameness. Cows with BCS < 2 were at greatest risk of future mild or severe lameness, including first lifetime lameness events in second

or higher lactation dairy cows, and long term repeated lameness events in all ages of cattle. The results suggest that maintaining $BCS \geq 2.5$ is optimal for reducing the risk of a lameness event. Results from this chapter also provides evidence that low BW (independent of BCS), and age at first calving over 24 months are important factors for long term risk of repeated lameness events. Overall, Model 3.1 explained 62% and 60% of the variability at cow level for mild and severe lameness respectively, highlighting the importance of the variables investigated as risk factors, and which if managed, could have significant impacts on controlling lameness on farm.

3.4.1 Body condition score

BCS is thought to be associated with development of CHDLs due to decreased thickness of the digital cushion and therefore reduced protective function (Bicalho et al., 2009, Green et al., 2014). The weekly locomotion scores over the 8 year time period provided sufficient data and power to allow analysis of individual body condition scores in increments of 0.25, from 2 through to 3, only scores outside of this range needed to be merged (for multinomial analysis). Based on the results of the current chapter, having controlled for stage of lactation, the minimum BCS to reduce the risk of mild or severe lameness was 2 but the risk reduced further as BCS increased. The results suggest that maintaining $BCS \geq 2.5$ is optimal for reducing the risk of lameness. Although higher BCS were associated with a reduced risk of mild lameness, this needs to be balanced with maintaining an optimum BCS depending on stage of lactation, for the control of conditions such as metabolic disease and dystocia. BCS has been reported as a risk factor for lameness in a number of studies (Hoedemaker et al., 2009, Green et al., 2014, Lim et al., 2014). Green et al. (2014) reported that in a longitudinal study of 1137 cows, those with $BCS \leq 2$ were more likely to be treated for lameness in the following 2 or 2 to 4 months compared with cows with $BCS > 2$. The results from that study support the findings of the current chapter, suggesting an association between low BCS and lameness that could be causal.

Notable in this analysis is that in the subset of cows with their first lifetime lameness event in the second lactation or higher, BCS had a significant effect 12 to 16 weeks previously for mild lameness and 8 to 12 weeks previously for severe lameness. Other lagged explanatory variables for BCS may also have had an effect, but due to low numbers in this subset of data and therefore reduced power, were not detectable. In general the models constructed to explore the associations between BCS and first

lameness events were using a much smaller subset of data compared with the model constructed to explore repeated lameness events and this needs to be taken into consideration when interpreting results. BCS did not appear to have a significant effect on the first lifetime lameness event in first lactation animals across the lagged explanatory variables for BCS tested, other than at 2 weeks for mild lameness. The results support the hypothesis that BCS is an important risk factor for lameness, but that factors other than BCS may be important for risk of first lameness events in heifers. Possible explanations for this include; the immature digital cushion at this age (Raber et al., 2006) or a different lameness aetiology in heifers compared with later parity cows. Raber et al. (2006) identified in a study comparing the composition of the digital pad in 6 heifers and 6 cows that the lipid content was significantly less in heifers compared with cows up to 4th lactation. It was inferred from these findings that lipid deposition occurs during first lactation; changes in BCS may therefore have less impact on the digital cushion of heifers. Body condition of animals in that study were not reported which may have influenced results; further work is required to understand these apparent differences between heifers and older cows. Alternatively, it may be that in growing cattle (such as heifers) body condition scoring, which has been developed for mature animals, is less informative. Unfortunately, the cause of lameness was not defined in this data set and risk factors that may be significant for first lameness events in heifers in this herd were not identified.

Periparturient BCS loss was also investigated as part of this chapter. Mobilization of lipid reserves in early lactation resulting in loss of body condition postpartum has been shown to contribute to increased risk of lameness (Hoedemaker et al., 2009). The results from this chapter support these previous findings; cows with loss in BCS 0 to 4 week post-calving were more likely to be severely lame in the future. These results highlight the importance of avoiding BCS loss in early lactation and maintaining an appropriate BCS throughout a cow's lifetime as an aid to preventing lameness.

3.4.2 Body weight

The association between BW and lameness, having adjusted for other factors including BCS was a novel finding. Norring et al. (2014) reported in a study of 70 dairy cows that severely lame cows had lower BW (713 ± 7 kg vs 718 ± 7 kg (BW $\pm$ S.E) for

lameness score 4 and 5 vs 1 and 2 respectively). In that study however, BW was not adjusted for BCS and therefore the reported difference in BW may have been influenced by the condition of the cows. The mechanisms by which BW influences the risk of lameness could be related to a number of different factors, such as biomechanical (Capion et al., 2008), nutritional or social factors. Of these, it is possible that low BCS (nutritional) and social/behavioural factors may be possible explanations for the association between low BW and increased risk of lameness. Having controlled for BCS, it is possible that BW is an indicator of condition within each individual BCS category (for example, low BW score 2 cows may be thinner than high BW score 2 cows). Therefore the association between lameness and low BW could have occurred because the risk of lameness may increase in cows that are lighter (and hence thinner), within each BCS category. Alternatively, behavioural and social function may explain the association between low BW and risk of lameness. Studies have shown that lameness is associated with lying times and lying times may impact on recovery. Ito et al. (2010) found that severely lame cows spent 1.6 hours per day longer lying down than cows that were not severely lame on farms with deep bedded stalls, indicating a change in behaviour that could aid recovery. Increased standing times or exposure to hard flooring surfaces have been shown to be risk factors for lameness and suggested to have a negative effect on recovery in lame cows (Galindo and Broom, 2000, Cook and Nordlund, 2009). If lying times are reduced, for example where behaviour has changed in lame cows or low social ranking cows, this could therefore potentially impact on the risk of repeated lameness events. Galindo et al. (2000) investigated the relationships between social and individual behaviour and incidence of lameness in 3 Holstein-Friesian dairy herds and demonstrated reduced lying times and reduced survival rate to lameness in low-ranking cows. It is possible therefore, that in this herd, low social ranking and associated stresses could have contributed to the increased risk of long term repeated lameness observed in the < 550 kg BW category cows compared to higher BW category cows. Further research is required to understand the interactions between BCS, BW and lameness.

3.4.3 Age at first calving

The current chapter provides evidence for the effect of age at first calving on the lifetime risk of lameness in dairy cows. Previous literature has only examined short term effects within one lactation or effects on individual lesions. Sanders et al. (2009)

reported that increased age at first calving decreased the hazard for thin sole-induced toe ulcers and white line disease, but increased the hazard for heel ulcers. Whilst, Ettema and Santos (2004) demonstrated a greater incidence of lameness in the first lactation with older age at first calving (≥ 25 months). Increased growth rates during the rearing period have been associated with younger age at first breeding and age at first calving (Brickell et al., 2009). Therefore, one hypothesis explaining the relationship between age at first calving and lifetime risk of repeat lameness is that an increased age at first calving may reflect animals that are less robust, with reduced ability to cope with their environment. As a consequence, these heifers may have had poorer growth rates, immune function or disease status, which may then be reflected long term with an increased susceptibility to lameness. Further research is required to investigate this hypothesis. Age at first calving has already been demonstrated to be an important factor influencing future health and productivity in dairy cows, with earlier age at first calving resulting in improved survivability, milk yield and fertility (Bach, 2011, Cooke et al., 2013). The findings of the current chapter add weight to the evidence for earlier age at first calving in benefiting lifetime health and productivity of dairy cows.

3.4.4 Previous lameness events

Previous lameness events were significantly associated with the risk of current lameness. Green et al. (2014) and Reader et al. (2011) also reported this finding. One explanation is that treatments are not effective or long lasting or that the underlying risk, such as thin digital cushion associated with low body condition score, is not removed by treatment. This association could also be explained by a lameness event resulting in damage that alters the structure or function of the claw, thereby increasing the susceptibility to future lameness, as suggested by Knott et al. (2007). These results indicate that managing herds to minimise the number of first cases of lameness in individual cows is an important factor in controlling lifetime risk of lameness.

3.4.5 Feed and genetic group

Inclusion of feed-genetic groups within the model allowed the effect of genotype and environment on lameness to be accounted for alongside other risk factors. This is important in reflecting the multifactorial nature of lameness and allowed the effect of the cow factors described above to be distinguished from the environmental factor associated with duration of housing.

3.5 Conclusions

The findings from this chapter indicate that a BCS < 2 was associated with the greatest risk of mild or severe lameness in dairy cows, and that the risk of lameness decreases with increased BCS. The results suggest that maintaining BCS ≥ 2.5 may be optimal for reducing the risk of lameness in dairy cows. These findings were relevant for long term repeated lameness events and first time lameness events in seconded lactation and greater cows. Results from this chapter also provides evidence for low BW and increased age at first calving, over 24 months, being important risk factors for lifetime risk of repeated mild or severe lameness events in dairy cows. Other variables associated with increased risk of lameness included higher parity, loss in BCS 0 to 4 weeks post-calving, higher milk yield 16 weeks previously, feed-genetic group and time from occurrence of previous lameness events. The combination of risk factors explored in this data analysis explained 62% and 60% of the variability for mild and severe lameness respectively, highlighting their importance and where management interventions could be targeted to impact significantly on reducing the risk of lameness in dairy herds.

CHAPTER 4: Lameness in Dairy Heifers: Impacts of Hoof Lesions Present Around First Calving on Future Lameness, Milk Yield and Culling Risk

4.1 Introduction

It is widely reported that clinical lameness in any lactation increases the risk of future lameness (Green et al., 2014, Hirst et al., 2002b); a finding that was also reported in Chapter 2. The impact of foot lesions during the first lactation on long-term lameness risk, is however less clear. Lameness in dairy heifers has the potential to have a severe impact on their overall lifetime performance within the herd. The significance of this is most pronounced when considering the high prevalence of lesions in heifers (Capon et al., 2009, Maxwell et al., 2015, Manske et al., 2002). Capon et al. (2009) found the prevalence of moderate to severe sole haemorrhage and white line lesions in 147 Danish Holstein heifers was 55% and 72% at 1 - 100 days in milk (DIM) respectively and the prevalence of digital dermatitis (DD) peaked at 39% at 0-100 DIM. Similarly, Maxwell et al. (2015) reported that 95% of a cohort of 139 Holstein dairy heifers being trimmed at between 50 and 80 days post-partum had some pathology on at least one claw. Lameness in the first lactation has been associated with a doubling of the hazard for lameness in the second lactation (Hirst et al., 2002b). Consequently, Bell et al. (2009) suggested that a critical control point for lameness in dairy cattle should aim to prevent CHDLs and digital dermatitis in heifers. The transition period, around the time of calving, has been identified as an important risk period, with increased stress related to physiological changes, social factors and changes in housing that impact on the risk of lameness occurring in heifers (Tarlton et al., 2002, Bergsten et al., 2015). Webster (2002) reported that heifers housed in straw yards for eight weeks after calving before being moved to cubicle housing resulted in less severe sole haemorrhages compared to heifers introduced to cubicle housing four weeks before calving. This finding demonstrated that housing practices around the time of calving affect the development of foot lesions in dairy heifers. The impact of lesions in heifers on long-term lameness is not yet known and could have major implications for the future health and welfare of the dairy herd.

Lameness in dairy cows has also been demonstrated to be associated with significant impacts on performance, such as reduced milk yield and increased culling risk (Booth et al., 2004, Amory et al., 2008). For other diseases, such as mastitis, it has been shown that disease occurring in heifers affects lifetime performance, for example

an increase in somatic cell count in heifers in early lactation negatively impacts on lifetime milk yield (Archer et al., 2013). This relationship may also be true for lameness, but has not yet been fully explored.

The aim of the current chapter is to investigate the long-term impacts of hoof lesions that occur around the time of first calving in heifers, on lameness, daily milk yield and culling risk. Mixed effects logistic regression and linear regression models were constructed to test the null hypothesis that hoof lesions occurring around the time of first calving in heifers have no impact on future lameness risk, average daily milk yield and culling risk.

4.2 Materials and methods

4.2.1 Study herd

The study herd from which data were obtained and descriptions of the life-time data for the Langhill and Acrehead herds are detailed in Chapter 2. Cows spent on average 3.9 years (3.5 lactations) within the Langhill herd, and is referred to throughout this chapter as ‘herd lifetime’. Descriptions for young-stock management and hoof lesion scoring are detailed in sections 4.2.1.1 to 4.2.1.3.

4.2.1.1 Young-stock management prior to first calving

Young-stock were reared in stable groups of approximately 25 animals from weaning to the start of their first transition period at approximately eight weeks before calving. As calves, they remained with the dam until at least 24 hours of age, and were fed 2 litres of colostrum by stomach tube. Following removal from the dam, calves were housed individually indoors in straw-bedded pens and received 2 litres of pooled colostrum twice daily for up to 7 days, followed by 6 litres per day of calf milk replacer. After ten days, calves were housed in group pens with deep straw bedding; the UK minimum recommended space allowance (Defra, 2003) was exceeded at all times. Fresh water was available from drinking bowls fitted to the wall of the building and calf milk replacer was fed via automatic feeders. Calves were weaned at approximately 50 to 60 days and managed as one group of dairy replacement young-stock; they were reared indoors until their second summer. Heifers were grazed during their second summer and then fed a young-stock ration when housed during winter. Table 4.1 presents a summary of the typical formulation for the young-stock ration. Housing was

straw bedded pens until 12 to 15 months of age, at which time all heifers were moved to cubicle housing with mattress and sawdust until the transition period. Passageways were grooved concrete. Target age at first calving was 24 months; first service was scheduled at approximately 350kg of BW and 15 months of age. All inseminations were artificial. At the Langhill site, heifers calved all year round. No routine foot trimming was performed prior to first calving. Footbathing was carried out monthly for young-stock using 5% copper sulphate solution. Live weight was recorded monthly using walk-in weigh scales. Prior to the start of the transition period before first calving, heifers were separated according to the feeding system to which they had been allocated (described in section 2.2.3) and were fully housed in straw bedded pens until calving. The same management protocols were applied by the same stock persons and technicians throughout the study period.

Table 4.1 Example young-stock ration fed to dairy heifers calving for the first time during the period August 2003 to March 2006 at the Scotland's Rural College (SRUC) Dairy Research and Innovation Centre.

Description	DM (%)	Actual Weight (kg/animal)	
Young-stock ration	54.66	16.10	
Actual			
		kg/ animal	% Load
Straw	80.00	6.00	37.27
Distillery co-product	30.00	8.00	49.69
General purpose minerals	100.00	0.10	0.62
Molasses	75.00	2.00	12.42

4.2.1.2 Lesion scoring around first calving

During the period 1st September 2003 to 31st January 2006, all four feet of heifers were lifted and lesions were recorded on standardised hoof maps (Greenough and Vermunt, 1991). Examinations were carried out by the same veterinary surgeon at regular intervals, approximately two months apart, with an average of 37 heifers being examined each time over the three year period 2003 to 2006. Lesions were severity scored on a 1 to 10 scale for sole or white line lesions (1 to 5 for haemorrhage and 6 to 10 for sole ulcers or white line separation) and a 1 to 3 scale for digital dermatitis (1 for mild or 3 for severe) as described by Offer et al. (2000) and Leach et al. (2005) (Table 4.2).

Table 4.2 Description of severity scores for hoof lesions recorded for heifers calving during the time period August 2003 to March 2006 and lesion scores recorded during the period September 2003 to March 2006 (Offer et al, 2000; Leach et al, 2005).

Visual appearance of lesion	Lesion score	Number of heifers with maximum lesion score recorded in each examination period		
		0-2 months pre-calving	0-2 months post-calving	2-4 months post-calving
Sole lesion				
Diffuse red or yellow in horn	1	40	57	21
Defined red in hoof horn	2	8	20	32
Stronger red colouration	3	4	8	31
Deep dense red	4	0	5	17
Port coloured	5	0	5	7
Mild sole ulcer, possible fresh blood	6	0	0	2
Corium exposed	7	0	2	0
Corium exposed with some loss of horn	8	0	0	1
Deep sole ulcer with major horn loss	9	0	0	0
Infected sole ulcer	10	1	0	0
White line lesion				
Diffuse red or yellow in white line	1	32	45	44
Defined red in white line	2	12	26	32
Stronger red colouration	3	2	13	16
Deep dense red	4	1	5	1
Port coloured	5	0	0	0
Separation of the white line, possible fresh blood	6	0	1	0
Corium exposed with separation	7	0	0	0
Corium exposed and loss of horn with separation	8	0	0	0
Deep separation of the white line	9	0	0	0
Infection present in the white line	10	0	0	0
Digital Dermatitis				
Lesion present in a small area	1	5	9	5
Larger lesion with slight exudate	2	4	4	11
Deeper lesion with exudate reddening and swelling	3	4	5	4

4.2.1.3 Management subsequent to first calving

As heifers calved they were introduced into the Langhill milking herd, remaining within the feeding system to which they had been allocated prior to calving. The detailed diet and management systems for the herd have been described in Chapter 2 (section 2.2.3). Cows were moved to Acrehead, typically after 3 lactations, according to Langhill research herd protocol requirements. Housing and general management was the same for cows in the Acrehead herd as it was for Langhill. Cows were milked three times daily and fed a grass-silage based total mixed ration, formulated to provide adequate nutrients for maintenance and milk production. All cows were housed in cubicle housing during winter months and had the potential to graze for varying periods throughout summer months (Rioja-Lang et al., 2009).

4.2.2 Statistical methods

Data recorded from heifers calving during the period August, 1, 2003 to March, 31, 2006 were obtained which included; lesion data recorded during the period September, 1, 2003 to January, 31, 2006 and lifetime health and production data recorded as these animals were followed through from September, 1, 2003 to August, 31, 2011. Microsoft Excel 2010 (Microsoft Corp.) was used for data handling and manipulation including identification and removal of unusual or anomalous data and constructing categorical variables. Where possible, missing observations were included as a categorical variable and fitted within each of the models to minimise the loss of data. Three examination periods were assigned to the data according to when heifers had lesions scored in relation to calving; 0 to 2 months pre-calving, 0 to 2 months and 2 to 4 months post calving.

As it was possible for a heifer to have multiple lesions on more than one claw, the maximum, sum and mean of the scores for each of the lesions (sole lesions, white line lesions and digital dermatitis) were calculated. As the majority of lameness is recorded in the hindlimbs, these were calculated for the hind feet only to give a cow-level lesion score. Lesion score data were added to the lifetime data records for the study population using the dataset described in Chapter 2. Outcome variables of interest were; lameness (based on locomotion score) categorised as ‘not lame’ (LS 1 or LS 2), or ‘lame’ (LS 3, LS 4 or LS 5), average daily milk yield (kg) as a continuous variable and culling as a binary variable (0 or 1 for not culled or culled respectively). Lesion score categories with a similar effect on the outcome variable were grouped together to ensure the minimum number of cows within a category was 10. Average daily milk yield was calculated for the time from first calving through to removal from the Langhill herd. Kendall’s correlation coefficient (Kendall, 1955) was used to determine the correlation between sole lesions and white line lesions during different time periods.

All models were constructed in MLWin 2.28 (Rabash et al., 2009). Multilevel models were used to explore the relationship between lesion scores and the outcomes of repeated locomotion scores and survival to culling. Data were structured at the cow week level. Initial assessment of model parameters was carried out using the iterative generalised least square procedure for parameter estimation (Goldstein, 2003) with forward selection of explanatory variables. Biologically plausible interactions were investigated. Final parameter estimates for each model were made using Markov chain

Monte Carlo (MCMC) to reduce the potential for biased estimates (Rabash et al., 2009). A minimum burn-in of 5,000 iterations was used during which model convergence occurred; these iterations were discarded to ensure confidence in the posterior distributions (Green et al., 2004). Explanatory variables remained within the model if the 95% credible interval of the odds ratio did not include 1 and as such were considered ‘significant’. To explore the relationship between lesion scores and average daily milk yield, a linear regression model was used, with data structured at the cow level (Dohoo et al., 2003). Model parameters were estimated using the iterative generalised least square procedure (Goldstein, 2003) and explanatory variables remained within the model if $P \leq 0.05$. A forward selection procedure was used for model build. Methods for assessing model fit and posterior predictions are described in detail below.

4.2.2.1 Model 4.1: Impacts of lesions present around first calving on lameness.

The data were analysed as a frailty model using a mixed effects binomial logistic regression framework (Goldstein, 2003), where each cow could have repeated lameness events over time. Cow was included as a random effect and time since last lameness event as a fixed effect. This model equates to a multilevel survival model with random effects (Goldstein, 1995).

The model took the form;

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

$$\text{Logit}(\pi_{ij}) = \alpha + \beta_1 \text{wk}_{ij} + \beta_2 X_{ij} + \beta_3 X_j + u_j$$

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

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, wk_{ij} = categorical variable to represent week of the study for the i th observation of the j th cow, β_1 = vector of coefficients for wk_{ij} , X_{ij} = vector of covariates associated with each observation, β_2 = coefficients for covariates X_{ij} , X_j = vector of covariates associated with each cow, β_3 = coefficients for covariates X_j . u_j = random effect to account for residual variation between cows (assumed to be normally distributed with mean = 0 and variance = σ_v^2).

Lesion scores were included as a categorical explanatory variable. Other potentially confounding explanatory variables tested included; categories for parity (1 to 4 +), previous LS 3, 4 or 5 (yes or no in two month intervals; 0 to 2 months previously, 2 to 4 months previously and > 4 months previously), age at first calving (< 24 months, 24 to 27 months, 28 to 30 months, 31 to 33 months and greater than 33 months), feed-genetic group (low-forage control: LF-C, low-forage select: LF-S, high forage control: HF-C, high forage select: HF-S, dry-control: D-C, dry-select: D-S, other-control: O-C, other-select: O-S, where other represents all management groups outside of LF, HF and Dry). Locomotion score assessor was included as an explanatory variable to control for possible inter-observer variability (Locomotion score recorder; 1 to 4). Weeks in milk (WIM) was categorised in five 8-week intervals from 0 to 40 weeks and a separate category for > 40 weeks. Week of the study was included as a categorical variable to account for background changes in risk over time. Within the dataset there were a small number of cows with a high number of weeks recorded 'lame', which influenced model parameters (see Figure 2.6 in section 2.3). Therefore a term was included for cows with greater than 40 lame weeks, with the threshold value being selected based on examination of the frequency distribution of the number of lame weeks per cow.

Posterior predictions were used to assess model fit by visual comparison to the observed data (Gelman et al., 1996) and the Hosmer-Lemeshow test (Hosmer and Lemeshow, 1989) was used as a statistical test for goodness-of-fit for mixed effects models by comparing deciles of fitted risk values to the matched observed risk, as explained in further detail in section 3.2.2.1. Posterior predictions were also used to calculate relative risks for each of the lesion categories to aid interpretation of results.

4.2.2.2 Model 4.2: Impacts of lesions present around first calving on milk yield

A linear regression model was used to analyse the data with animal average daily milk yield for time spent at Langhill, as the outcome.

The model took the form;

$$\text{Yield}_i \sim N(XB, \Omega)$$

$$\text{Yield}_i = \alpha + \beta_1 X_i + e_i$$

$$[e_i] \sim N(0, \Omega_e)$$

Where $Yield_i$ is the average daily yield for the i th cow. α = intercept value, β_1 = vector of covariates associated with each cow and e_i represents the residual error (assumed to be normally distributed, with mean = 0 and variance = Ω_e).

Lesion scores were included in the model as a categorical variable. Other explanatory variables tested included; feed-genetic group (low-forage control: LF-C, low-forage select: LF-S, high forage control: HF-C, high forage select: HF-S), maximum age at first calving (< 24 months, 24 to 27 months, 28 to 30 months, 31 to 33 months and > 33 months).

Model fit was evaluated using plots of standardized residuals and by examining the influence and leverage of data points (Rabash et al., 2009).

4.2.2.3 Model 3: Impacts of lesions present around first calving on culling

A discrete time survival model was used to explore the relationship between lesions and survival to culling.

The model took the form;

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

$\text{Logit}(\pi_{ij}) = \alpha + \beta_1 wk_{ij} + \beta_2 X_{ij} + \beta_3 X_j + u_j$

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

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, wk_{ij} = categorical variable to represent week of the study for the i th observation of the j th cow, β_1 = vector of coefficients for wk_{ij} , X_{ij} = vector of covariates associated with each observation, β_2 = coefficients for covariates X_{ij} , X_j = vector of covariates associated with each cow, β_3 = coefficients for covariates X_j , u_j = random effect to account for residual variation between cows (assumed to be normally distributed with mean = 0 and variance = σ_v^2).

Lesion scores were included as a categorical explanatory variable. Other potentially confounding explanatory variables tested were the same as described above for Model 3.1. The only difference being that parity was not included and WIM was categorised in two 16-week intervals from 0 to 32 weeks and another category for > 32 weeks.

Posterior predictions were used to assess model fit by visual comparison to the observed data (Gelman et al., 1996) and the Hosmer-Lemeshow test (Hosmer and Lemeshow, 1989) was used as a statistical test for goodness-of-fit for logistic regression models.

4.3 Results

Data were available for a total of 158 heifers calving for the first time between August 2003 and March 2006; parity number ranged from 1 to 7 for animals in the complete dataset.

4.3.1 Descriptive analysis

Sole lesions were the most commonly observed lesion at each of the examination points and the proportion of heifers with sole lesions increased from pre-calving to 2 to 4 months post-calving, such that by 2 to 4 months post-calving 97% of heifers had some degree of sole lesion recorded (Table 4.3). A similar pattern of increasing proportions of heifers having a lesion recorded for each of the time periods was also observed for white line lesions; at the 2 to 4 month post calving observation 81% of heifers had a lesion recorded (Table 4.3). Score severity also increased over the time periods 0 to 2 months pre-calving through to 2 to 4 months post-calving for white line (Figure 4.1 and Appendix; supplement to Figure 4.1) and sole lesions (Figure 4.2 and Appendix; supplement to Figure 4.2).

Sole lesion and white line lesion scores were moderately correlated at each examination point (Kendall's tau = 0.24, 0.35 and 0.13 for the time period 0 to 2 months pre-calving, 0 to 2 months post-calving and 2 to 4 months post-calving respectively, $P \leq 0.05$).

Table 4.3 Summary of the number of hoof lesions recorded for each examination point and examination point and lesion cumulative incidence in 158 heifers calving during the time period August 2003 to March 2006 and lesion scored during the period September 2003 to January 2006.

Lesion	Examination point	Total number of heifers observed ¹	Number of heifers with lesions on at least one claw	Lesion cumulative incidence
White line lesion	0 to 2 months pre-calving	145	57	0.39
	0 to 2 months post calving	128	96	0.75
	2 to 4 months post-calving	118	95	0.81
Sole lesion	0 to 2 months pre-calving	145	59	0.41
	0 to 2 months post calving	128	103	0.80
	2 to 4 months post-calving	118	115	0.97
Digital dermatitis	0 to 2 months pre-calving	145	14	0.10
	0 to 2 months post calving	128	19	0.15
	2 to 4 months post-calving	118	20	0.17

¹A total of 158 heifers were included in the data set, however actual examination periods and categorised examination periods are likely to not always coincide, therefore resulting in missing observations. There is no other information to suggest these data were anything other than missing at random.

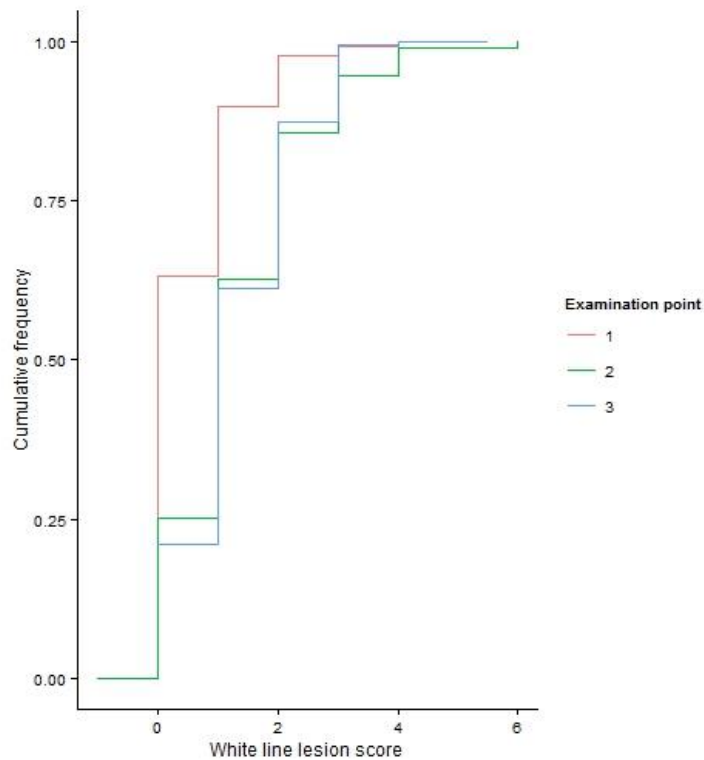


Figure 4.1 Cumulative frequency plot for white line lesion scores within each examination point for 158 heifers calving during the time period August 2003 to March 2006 and lesion scored during the period September 2003 to January 2006. Examination points 1 to 3 represent; 1 = 0 to 2 months pre-calving, 2 = 0 to 2 months post-calving and 3 = 2 to 4 months post-calving.

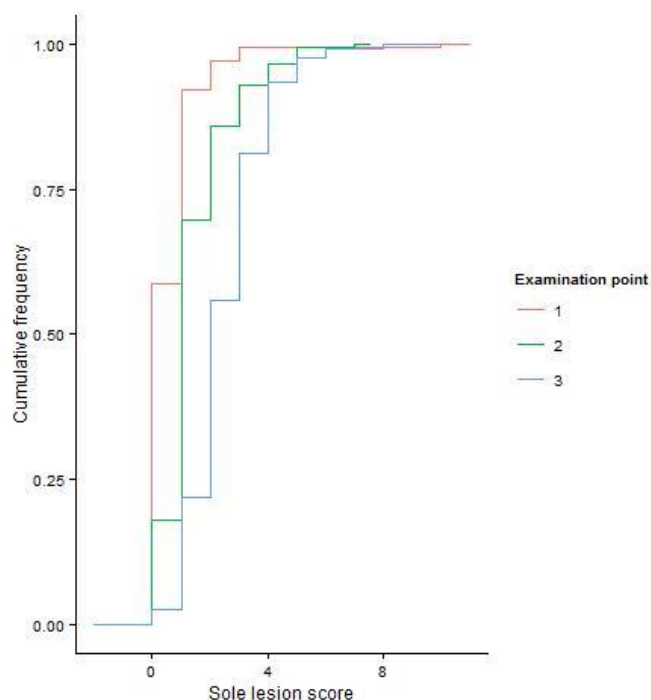


Figure 4.2 Cumulative frequency plot for sole lesion scores within each examination point for 158 heifers calving during the time period August 2003 to March 2006 and lesion scored during the period September 2003 to January 2006. Examination points 1 to 3 represent; 1 = 0 to 2 months pre-calving, 2 = 0 to 2 months post-calving and 3 = 2 to 4 months post-calving.

4.3.2 Modelling

For all models, maximum lesion scores were included as a categorical variable in the final models. This was because the maximum score was considered biologically most likely to have an impact on subsequent health. Results for each of the models are described in detail below;

4.3.2.1 Model 4.1: Impacts of lesions present around first calving on lameness.

The dataset included a total of 24,335 cow weeks at risk for 158 heifers with lesion score data. There were 4,093 lame events recorded in a total of 146 animals over the period September, 1, 2003 to August, 31, 2011. Table 4.4 shows the results from Model 4.1.

Heifers with white line lesion scores between 2 and 4 in the 0 to 2 months pre-calving period had a decreased risk of future lameness events compared with heifers with lesion score zero or 1 at this examination point (OR (95% credible interval) = 0.34 (0.13 to 0.86) for lesion score = 2 to 4). White line lesions with a score of ≥ 2 in the 2 to 4 months post-calving period were associated with a significantly increased risk of future lameness compared with a baseline of score of zero to 1 (OR (95% credible interval) = 1.48 (1.07 to 3.12) and 3.48 (1.34 to 9.07) for score 2 and 3 to 4 respectively). Compared with the baseline white line lesion score of zero to 1, a score ≥ 3 at this examination point had a predicted increased relative risk of future lameness of 1.6.

More severe sole lesions (score of 4 to 8) in the 2 to 4 months post-calving were similarly associated with an increased risk of future lameness compared with a baseline score of 2 (OR (95% credible interval) = 1.53 (0.87 to 2.67) and 2.90 (1.54 to 5.46) for scores 3 and ≥ 4 respectively). Heifers with lesion score zero or 1 in the 2 to 4 months post-calving were also at increased risk of future lameness compared with those with a mild lesion of score 2 (OR (95% credible interval) = 2.00 (1.14 to 3.51)). Compared with a baseline sole lesion score of 2, more severe sole lesions (score 4 to 8) at this time point had a predicted increased relative risk of future lameness of 2.6, whilst a score of zero or 1 had a predicted increased relative risk of future lameness of 2.1. Interactions between feed-genetic group and white line lesion score 2 to 4 months post-calving were significant including, Dry:C and score 2 (OR (95% credible interval) = 2.05 (1.08 to 3.90)), HF:S and score 3 to 4 (OR (95% credible interval) = 0.21 (0.05 to 0.88)) and Other:C and score 3 to 4 (OR (95% credible interval) = 0.06 (0.01 to 0.50)). The

variance at the cow level was 0.85; inclusion of random effects improved model fit. Model fit was good based on inspection of posterior predictions for number of lameness events compared with observed number of lameness events for deciles of fitted risk values (Figure 4.3). $\chi^2 = 11.95$, $p = 0.22$.

Table 4.4 Model 4.1: Binomial model for repeated lameness events in 158 heifers calving during the time period August 2003 to March 2006 and lesion scored during the period September 2003 to January 2006.

Intercept			Coefficient: - 4.49		
Variable	N ¹	Lame ²	Odds ratio	Lower 95% CrI ³	Upper 95% CrI
White line lesion score (0 to 2 months pre-calving)					
0 to 1 ^{4a}	20,058	0.17	Baseline		
2 to 4 ^{4b}	2,272	0.13	0.34	0.13	0.86
White line lesion score (2 to 4 months post-calving)					
0 to 1 ^{4c}	11,781	0.15	Baseline		
2 ^{4d}	5,035	0.16	1.48	0.70	3.12
3 to 4 ^{4e}	2,434	0.19	3.48	1.34	9.07
Sole lesion (2 to 4 months post-calving)					
2 ^{4f}	6,522	0.14	Baseline		
0 to 1 ^{4g}	4,226	0.18	2.28	1.16	4.48
3 ^{4h}	4,897	0.14	1.53	0.87	2.67
4 to 8 ⁴ⁱ	3,605	0.20	2.90	1.54	5.46
Feed – genetic group ⁵					
LF:C	4,758	0.15	Baseline		
LF:S	4,757	0.21	1.04	0.50	2.16
HF:C	5,462	0.13	0.99	0.62	1.58
HF:S	5,680	0.17	1.52	0.73	3.13
Dry:C	1,436	0.14	0.90	0.53	1.52
Dry:S	1,273	0.21	2.33	1.03	5.27
Other:C	137	0.55	7.62	3.17	18.30
Other:S	183	0.62	12.82	4.79	34.29
Locomotion score assessor (1 to 4)					
1	1,150	0.42	Baseline		
2	11,331	0.11	0.38	0.30	0.48
3	11,116	0.17	1.12	0.88	1.41
4	738	0.69	1.93	1.34	2.79
Week category ⁶					
0 – 30	1,285	0.09	Baseline		
31 – 60	1,889	0.06	0.72	0.50	1.02
61 – 90	2,595	0.07	0.64	0.46	0.89
91 – 120	3,031	0.08	0.66	0.48	0.92
121 – 150	3,596	0.11	1.05	0.76	1.46
151 – 180	3,351	0.13	1.36	0.98	1.89
181 – 210	2,864	0.13	1.71	1.21	2.43
211 – 240	2,367	0.25	4.27	3.03	6.00
241 – 300	2,507	0.43	6.10	4.26	8.74
301 – 360	733	0.59	8.53	5.24	13.90
> 360	117	0.69	6.21	2.85	13.52
Weeks in milk					
0 – 8	3,765	0.14	Baseline		
9 – 16	3,457	0.15	1.16	0.95	1.42
17 – 24	2,963	0.17	1.53	1.24	1.89
25 – 32	2,496	0.18	1.56	1.25	1.95
32 – 40	2,876	0.20	1.59	1.27	1.97

> 40	5,117	0.14	1.33	1.10	1.61
Previous lameness event (0 to 2 months) ⁷					
None	10,230	0.03	Baseline		
Lameness event	12,628	0.28	3.96 ^c	3.37	4.65
Previous lameness event (2 to 4 months)					
None	9,737	0.06	Baseline		
Lameness event	11,854	0.27	1.51	1.31	1.73
Number lame weeks per cow ⁸					
≤ 40 lameness events	17240	0.09	Baseline		
> 40 lameness events	7095	0.36	3.70	2.30	5.97
Feed – genetic group x White line lesion score (2 to 4 months post-calving)					
LF:S x 2			1.12	0.37	3.40
HF:C x 2			1.00	0.47	2.11
HF:S x 2			1.00	0.34	2.94
Dry:C x 2			2.05	1.08	3.90
Dry:S x 2			2.23	0.74	6.75
Other:C x 2			0.39	0.12	1.27
Other:S x 2			1.63	0.33	8.06
LF:S x 3 to 4			0.58	0.15	2.27
HF:C x 3 to 4			0.38	0.11	1.33
HF:S x 3 to 4			0.21	0.05	0.88
Dry:C x 3 to 4			0.99	0.33	2.97
Dry:S x 3 to 4			0.35	0.08	1.46
Other:C x 3 to 4			0.06	0.01	0.50
Other:S x 3 to 4			0.58	0.03	12.60
Random effect			Variance: 0.85		

¹N = Total number of observations (cow weeks) within each category.

²Proportion of observations recorded lame within each category.

³CrI = credible interval.

^{4a to i}Number of cows with lesions observed within each lesion score category; a = 118, b = 15, c = 65, d = 32, e = 17, f = 32, g = 2, h = 31, i = 27.

⁵Feed-genetic groups include low forage (LF), high forage (HF), control (C), and select (S). Dry refers to dry cows, and other refers to all other management groups outside of LF, HF, and Dry.

⁶Week category = week of the study period, included as a categorical variable.

⁷Previous lameness event based on locomotion score recorded as 3, 4 or 5.

⁸Covariate for number of lame weeks per cow (>40) was added to the model to correct model over-dispersion and improve model fit.

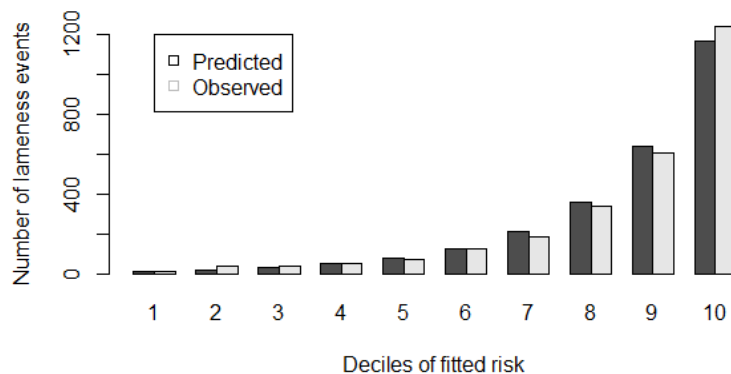


Figure 4.3 Observed and predicted repeat lameness events (LS ≥ 3) per decile of fitted risk values. Posterior predictions were from Model 4.1, constructed using records obtained from animals for the study period 2003 to 2011.

4.3.2.2 Model 4.2: Milk yield over lifetime within the Langhill herd

Milk yield data were available for 157 heifers, with an average time within the Langhill herd of 3.9 years. The mean (SD) average daily milk yield was 27.6 (5.9) kg with a range of 4.1 kg to 41.1 kg, and was approximately normally distributed. Table 4.5 shows the results for Model 4.2. The mean effect and mean number of days cows spent in the herd for each lesion category were used to calculate an adjusted yield loss for lesion categories.

Heifers with sole lesions score ≥ 4 in the 2 to 4 month post-calving period had a significantly reduced average daily milk yield of 2.68 kg compared with those with no lesion at this time point. Animals with sole lesions score ≥ 4 at this examination point remained within the herd a median of 326 days less (Figure 4.4 and Appendix; supplement to Figure 4.4), therefore the mean yield loss associated with these sole lesions equated to 9,928 kg over the animals' productive lifespan within the herd (calculated from the coefficient of the intercept multiplied by the mean number of days in the herd for cows in the baseline category minus the mean effect of the significant category multiplied by the mean number of days in the herd for cows in the significant category i.e. $(1631.88 \times 19.72) - (1305.89 \times (19.72 + -2.68))$). Digital dermatitis in the 2 to 4 months post-calving was associated with an increased average daily milk yield of 2.63 kg compared with no lesion. However, since animals with digital dermatitis lesions at this examination point remained in the herd for an average of 341 days less (Figure 4.5 and Appendix; supplement to Figure 4.5) than those with no lesion at this examination point, the adjusted yield difference associated with the presence of digital dermatitis lesions compared with no lesion was a net loss of 3,513 kg of lifetime production within the herd (calculated using the method described above). Residuals were normally distributed which indicated model fit was good.

Table 4.5 Linear regression model (Model 4.2) for average daily milk yield within the Langhill herd in 157 heifers calving during the time period August 2003 to March 2006, with herd lifetime data recorded from September 2003 to August 2011.

Intercept		Coefficient: 19.72		
Variable	N ¹	Mean effect	Lower 95% CI ²	Upper 95% CI
Sole Lesion (2 to 4 months post-calving)				
0 to 1	24	Baseline		
2	35	-0.76	-3.03	1.50
3	32	0.008	-2.29	2.30
4 to 8	27	-2.68	-5.05	-0.31
Digital dermatitis (2 to 4 months post-calving)				
0	98	Baseline		
1 to 3	20	2.63	0.51	4.75
Feed – genetic group ³				
LF:C	30	Baseline		
LF:S	36	10.57	7.99	13.14
HF:C	30	-1.41	-3.87	1.05
HF:S	32	2.737	0.27	5.21

¹N = Number of heifers with lesions observed within each category.

²CI = confidence interval. Parameter is 'significant' if the 95% confidence interval excludes 0.

³Feed-genetic groups include low forage (LF), high forage (HF), control (C), and select (S).

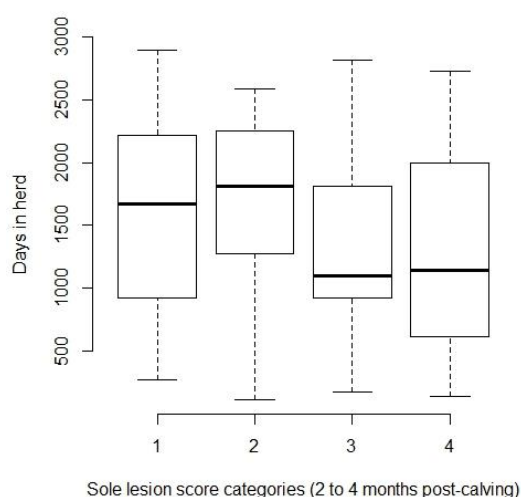


Figure 4.4 Box plot showing median (black line) and interquartile ranges (whiskers) for number of days in herd for each sole lesion category 2 to 4 months post-calving (lesion scores for categories; 1 = 0 to 1, 2 = 2, 3 = 3, 4 = 4 to 8) for 157 heifers calving during the time period August 2003 to March 2006 and lesion scored during the period September 2003 to January 2006.

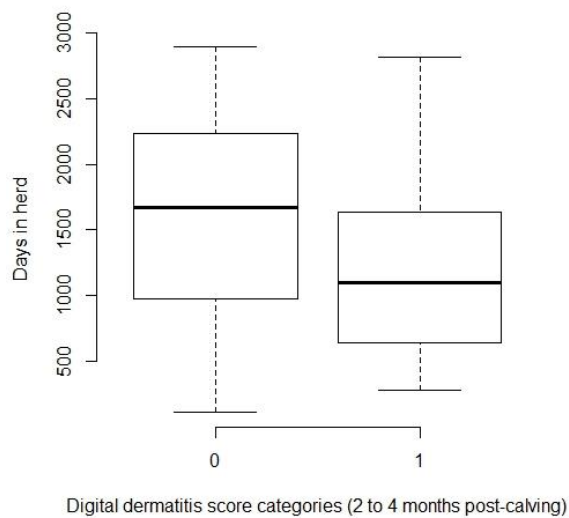


Figure 4.5 Box plot showing median (black line) and interquartile ranges (whiskers) for number of days in herd for presence or absence of digital dermatitis 2 to 4 months post-calving (lesion scores for categories; 0 = lesion absent, 1 = lesion present), for 157 heifers calving during the time period August 2003 to March 2006 and lesion scored during the period September 2003 to January 2006.

4.3.2.3 Model 4.3: Impacts of lesions present around first calving on culling.

Culling data were available for 157 heifers; 139 animals were culled within the study period September, 1, 2003 to August, 31, 2011. The data set included a total of 39,417 cow weeks at risk. Table 4.6 shows the results for Model 4.3.

Sole lesions in the 0 to 2 months pre-calving was the only lesion in any of the time periods investigated with a significant association with culling. Sole lesion of score 1 compared with no lesion was associated with a reduced risk of culling (OR (95% CI) = 0.52 (0.32 to 0.84)). The variance at cow level was 0.61; inclusion of random effects improves model fit. Tertiles of fitted risk values were used to compare posterior predictions with observed number of culling events to ensure a minimum frequency value of 5 in each group. Posterior predictions indicated good model fit (Figure 4.7), $\chi^2 = 0.55$, $p = 0.76$.

Table 4.6 Binomial model for survival to culling for 157 heifers calving during the time period August 2003 to March 2006 and lesion scored during the period September 2003 to January 2006.

Intercept		Coefficient: 9.18		
Variable	N ¹	Odds ratio	Lower 95% CrI ²	Upper 95% CrI
Sole lesion (0 to 2 months pre-calving)				
0 ^{3a}	17096	Baseline		
1 ^{3b}	10709	0.52	0.32	0.84
2 to 10 ^{3c}	2164	0.70	0.31	1.61
Feed – genetic group ⁴				
LF:C	4728	Baseline		
LF:S	4927	2.27	0.93	5.50
HF:C	5625	0.45	0.14	1.44
HF:S	5833	0.76	0.24	2.39
Dry:C	1801	0.37	0.13	1.08
Dry:S	1593	0.66	0.23	1.86
Other:C	5187	1.39	0.51	3.80
Other:S	1924	3.97	1.39	11.39
Week category ⁴				
0 – 60	3362	Baseline		
61 – 120	5923	1.08	0.31	3.72
121 – 180	7552	2.09	0.62	7.11
181 – 240	5825	2.11	0.57	7.81
241 – 300	4604	1.04	0.24	4.42
301 – 360	3262	1.48	0.32	6.77
> 360	1889	1.00	0.18	5.46
Weeks in milk				
0 – 16	8357	Baseline		
17 – 32	6239	1.89	0.69	5.16
> 32	9211	2.58	1.06	6.30
Random effect		Variance: 0.61		

¹N = Number of cow weeks.

²CrI = credible interval.

^{3a to c} Number of cows with lesions observed within each lesion score category; a = 78, b = 38, c = 11.

⁴Feed-genetic groups include low forage (LF), high forage (HF), control (C), and select (S). Dry refers to dry cows, and Other refers to all other management groups outside of LF, HF, and Dry.

⁵Week category = week of the study period, included as a categorical variable.

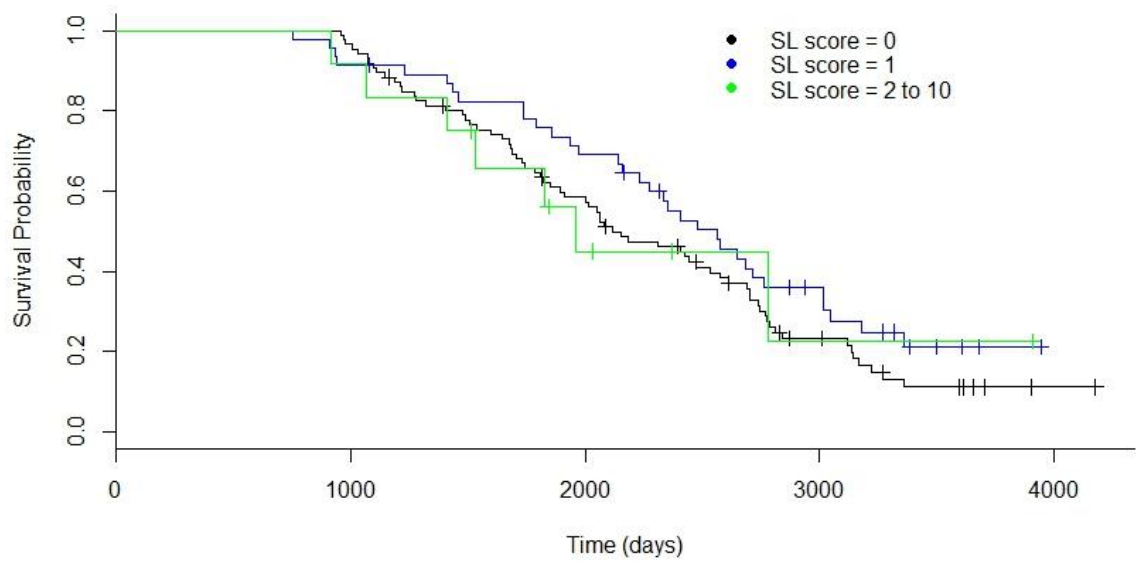


Figure 4.6 Kaplan-Meier survival plot for sole lesion (SL) categories 0 to 4 months pre-calving for 157 heifers calving during the time period August 2003 to March 2006 and lesion scored during the period September 2003 to January 2006.

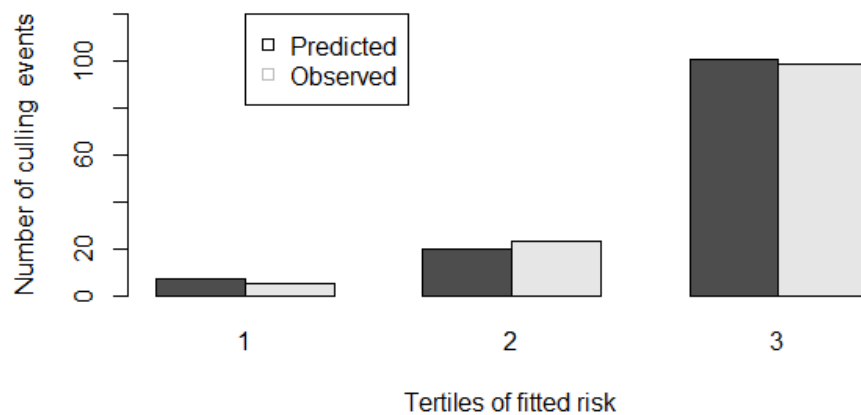


Figure 4.7 Observed and predicted number of culling events in heifers per tertile of fitted risk values. Posterior predictions were from Model 4.3, constructed using records obtained from animals for the study period 2003 to 2011.

4.4 Discussion

This chapter reports on findings demonstrating the long term impacts of foot lesions around the time of first calving in heifers, on future lameness risk, milk yield and culling risk in a dairy herd. Previous studies investigating the impacts of lameness in heifers on these outcomes have looked separately at impacts on future lameness

(Hirst et al., 2002b) or milk yield (Onyiro et al., 2008), or at impacts of digital dermatitis in pre-calving heifers on culling restricted to the first lactation (Gomez et al., 2015). As heifers represent the future of the dairy herd, understanding the overall effects of lesions occurring around the time of first calving could be important for improving lameness control in dairy herds. A particularly novel finding from the data analysis in this chapter was the reduced risk of culling associated with mild sole lesions (score 1) in the 0 to 2 months pre-calving.

4.4.1 Impacts of lesions present around first calving on culling

To the authors' knowledge, there are no previous studies reporting the impacts of lesions in heifers on long term survival within the dairy herd. Gomez et al. (2015) reported a numerically, but not statistically significant effect of digital dermatitis in pre-calving heifers on increased risk of culling before 60 days in milk (DIM) in their first lactation. Sogstad et al. (2007) used claw trimming data from 500 Norwegian herds to investigate the impacts of lameness and lesions on culling within the same lactation that claw trimming took place; lameness in lactation 1 was associated with earlier culling (hazard ratio = 4.2). Previous studies have reported significant negative effects of lameness in adult dairy cows on survival (Booth et al., 2004, Machado et al., 2010, Bicalho et al., 2007b). Whilst Barkema et al. (1994) et al. (1994) reported a lower culling rate associated with lameness, thought to be attributed to the retention of lame cows because of the higher milk production of these cows. Interpreting survival within the herd is complex due to the decisions behind culling, which may be a direct response to lameness or due to indirect effects of lameness on milk yield and fertility, alongside many other health and management reasons. In the current chapter the data were analysed using time to culling for all reasons due to this uncertainty. The findings of this chapter suggest that mild sole lesions occurring at a time when the animal is able to recover from and adapt to the insult may be beneficial for long term survival. Since the reasons for culling were not analysed it is not possible to identify possible underlying mechanisms, however this finding is consistent with some of the other outcomes explored; mild sole lesions and white line lesions were also associated with a reduced risk of lameness. Further research is required to understand the underlying mechanisms associated with this finding and to clarify the extent to which mild lesions may offer protection.

4.4.2 Impacts of lesions present around first calving on future lameness

More severe white line lesions and sole lesions were associated with a significantly increased risk of future lameness by 1.6 and 2.6 times respectively, across all future lactations within the herd. These results are similar to Hirst et al. (2002) who reported a positive association between claw horn lameness in heifers and future risk, but only for the second lactation; this association was not significant for the third lactation. Hirst et al. (2002b) also found that any type of lameness in the first lactation was associated with claw-horn lameness in the second lactation and hypothesised that this may be due to underlying pathology that is carried over from one lactation to the next. This hypothesis is supported by the findings of a study which used micro computed tomography and reported that claw horn lesions during life were associated with an increase in pathological changes to the bony architecture of the pedal bone (Newsome et al., 2016). This could explain the relationship between more severe CHDL and future lameness risk observed in this study. Further work is required to understand the longitudinal relationship between causal factors and the role of pathological changes to distal limb anatomy associated with CHDL.

The results of this chapter show that mild sole lesion and white line lesions occurring in the 2 to 4 months post-calving or 0 to 2 month pre-calving periods respectively, were associated with a reduced risk of lameness. This suggests that some degree of mild insult around the time of first calving may be beneficial to long term claw health; if adaptive changes occur in response to the insult during a time when the claw is able to recover and become more biomechanically resilient the animal may be less prone to lameness in the long term. Bergsten et al. (2015) reported findings that support this hypothesis; heifers reared on a hard flooring surface (cubicles with slatted concrete alleys) pre-calving and housed on a soft surface (slatted rubber alleys) post-calving resulted in the lowest prevalence and severity of sole and white-line haemorrhages in first-lactation. The authors suggested that the challenge from hard flooring during the rearing period resulted in traumatic sole haemorrhages, but as the heifers were able to cope at this time, this was ultimately beneficial for claw health. Previous studies have demonstrated that adaptive changes can take place in the bovine hoof and indicate that environment and exercise have a role in the development of the hoof support structures (Gard et al., 2015, Knott et al., 2007). Additional research is required to understand the mechanisms underlying these findings and therefore their clinical relevance. The findings from this chapter suggest that there may be a threshold

for severity of white line and sole lesions that is associated with an increased risk of future lameness, but that some degree of mild insult occurring at certain times may initiate adaptive changes within the hoof that are beneficial to long term claw health. Husbandry practices implemented during the pre- and post-calving period may have significant impacts on future lameness in adult cattle as a result of reducing the more severe lesions occurring during this time. Additionally, management of husbandry practices may also allow for adaptive changes to occur in the hoof that best prepare those heifers for their future life in the herd.

The interaction between the feed-genetic group HF:S and white line disease 2 to 4 months post-calving suggests that there may be environmental or nutritional factors mitigating the impacts of sole lesions occurring during this time period on the risk of future lameness. It may therefore be possible that the effects of CHDLs are different in herds with different management systems, for example grazed vs continuously housed.

The high prevalence of lesions in heifers has previously been highlighted in a number of studies (Manske et al., 2002, Capion et al., 2009, Maxwell et al., 2015), and at a similar level to that observed in the data analysed in this chapter. This is relevant not only for the health and welfare of those animals affected at that time, but also when considering the impacts on future health and welfare.

4.4.3 Impacts of lesions present around first calving on milk yield

Results from the current chapter showed that severe sole lesions 2 to 4 months post-calving were associated with a reduction in average daily yield of 2.68 kg. A number of studies have demonstrated a reduction in milk yield associated with lameness in dairy cows of all ages (Archer et al., 2010b, Green et al., 2002, Amory et al., 2008). Amory et al. (2008) investigated the effect of lesion-specific causes of lameness on milk yield in 1824 UK dairy cows. Sole ulcer and white line disease were associated with a lactation milk yield loss of approximately 570 and 370 kg respectively, whilst a slight increase in yield was observed following treatment of digital dermatitis. Interpretation of milk yield losses associated with lameness can be difficult, as it is the higher yielding cows that are more likely to become lame (Archer et al., 2010b, Green et al., 2002). This association may also lead to retention of lame cows within the herd. Maxwell et al. (2015) reported that lame heifers produced significantly more milk over the first lactation (734 litres, $P = 0.02$) than those that were not lame. In a study carried out 2003 to 2005 on the same UK research herd as that investigated in the current chapter, no

association was found between lameness in heifers and their 305-day yield. The study did not however explore the longitudinal relationship between lameness in heifers and their long-term future milk yield (Onyiro et al., 2008). Results from the current chapter also demonstrate that severe sole lesions were associated with a long-term negative impact on milk yield; this population of animals remained within the herd on average nearly one year less than those with no lesion. Similarly, as animals with DD lesions remained within the herd for a shorter period, DD was associated with a net yield loss, despite positive associations between DD and yield reported in this chapter and as reported by Amory et al. (2008). Gomez et al. (2015) also demonstrated that animals with DD lesions during the rearing period produce significantly less milk during their subsequent lactation. The reasons for cows leaving the Langhill herd were not recorded, therefore it is not possible to explain why severe sole lesions and DD were associated with a shorter time period within the Langhill herd.

4.4.4 Study limitations and generalisability

One of the limitations associated with this research in demonstrating the impacts of early life lesions in heifers on future lameness and performance, was that the regular examination of claws may have increased the level of treatment interventions, compared with the situation more commonly observed on UK commercial dairy farms. This may have resulted in a reduced effect of lesions on future lameness, milk yield and culling in this herd.

The published literature includes supporting evidence for the reduction in milk yield and longevity reported here and the possible protective effects of mild foot lesions on future robustness. Whilst the findings of this chapter are from one UK dairy herd and the quantitative impact of severe foot lesions in heifers are likely to be specific to this herd and dataset, the qualitative impacts may be generic across dairy herds using similar management systems.

Whilst the lesion scoring systems are subjective and may be prone to inter-observer variability, lesion scoring within the study period was limited to one person and any variability should not undermine the conclusions of the research. It is acknowledged that there is currently lack of a widely accepted lesion scoring system and there are also issues associated with a lack of understanding of pathogenesis and therefore lesion progression associated with CHDLs. Consequently results should be interpreted with consideration of the approach taken. Finally, due to the difficulties in

defining the duration of a case of lameness (especially in situations where lameness scoring is conducted frequently), in this chapter the risk of a cow being lame in any one week was modelled and results should therefore be interpreted in this context.

4.5 Conclusions

Findings from this chapter demonstrated that mild sole lesions are associated with an overall reduced risk of premature culling in dairy cows. It is therefore hypothesised that a mild insult may be beneficial to claw health; if adaptive changes occur in response to the insult during a time when the claw is able to recover and become more biomechanically resilient. High and low scores for white line and sole lesions in heifers were associated with a greater risk of future lameness than medium scores. High sole lesion scores and digital dermatitis were associated with an overall reduction in milk yield when adjusted for mean time within the herd. It is concluded that the current high prevalence of more severe CHDLs in dairy heifers is likely to have a large impact on the health, welfare and productivity of these animals over their lifetime within the herd. Identifying and implementing husbandry practices which reduce the occurrence of severe CHDLs is essential for the future sustainability of dairy herd production.

CHAPTER 5: The Contribution of Previous Lameness Events and Body Condition Score to the Occurrence of Lameness in Dairy Herds: A Study of Two Herds

5.1 Introduction

Numerous risk factors for lameness in dairy cattle have been reported in the literature, including environmental risk factors such as flooring surfaces and standing times (Galindo and Broom, 2000, Bergsten et al., 2015) as well as animal-based factors such as milk yield, body condition score and previous lameness events (Randall et al., 2015, Green et al., 2014). 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 (Bicalho et al., 2009, Green et al., 2014, Randall et al., 2015, Randall et al., 2016, Hirst et al., 2002b). Results from Chapter 3 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. Findings from Chapter 4 demonstrated that dairy heifers with CHDLs were at greater risk of lameness in subsequent lactations; similar findings were reported by Hirst et al. (2002b). A recent study suggested that this 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. Reported odds ratios indicated that these risk factors were highly associated with lameness; ranging from 7.73 (2.37 – 17.71) for BCS 1.00 – 1.75 at calving versus 2.50 – 2.75 to 13.80 (10.58 – 17.78) for previous lameness 31 – 60 days previously versus no lameness.

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 methods to calculate PAF with different limitations, such as biases arising from some formulas when adjusted estimates of relative risk are used or the exposure is across different levels (Steenland and Armstrong, 2006, Benichou,

2001, Rockhill et al., 1998). 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 BCS 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 to be incorporated into estimates of PAF (Steenland and Armstrong, 2006) as well as allowing for more complex scenarios to be investigated (Hudson et al., 2014).

The aim of this chapter was to investigate the contribution of previous lameness and BCS to the occurrence of total lameness events in two UK dairy herds. A novel approach to estimating PAF for these lameness risk factors, using simulation, was taken.

5.2 Materials and methods

5.2.1 Study herds

Data were obtained from two UK dairy herds, where detailed and accurate herd records were available. The reason for investigating 2 herds was to compare PAF estimates between herds with different environments and managements systems and hence give an insight into whether results may be similar and to an extent generalisable. The study herd from which data were obtained and descriptions of the data for Herd A are detailed in Chapter 2. For Herd B, the data were obtained from previous research conducted to investigate the hypothesis that cows in low BCS are more likely to become lame (Green et al., 2014). Herd B was 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. Cows were milked twice daily in a 60 point rotary parlour and continuously housed all year around, apart from summer when grazed during the last 2 months of lactation. Rations were formulated with the aim of maximising yield whilst minimising feed costs and fed to milking cow groups (early, mid and late lactation) accordingly. Biotin was added at 20mg/cow/day. Housing was modern free stall accommodation with water mattresses in cubicles and solid concrete passageways with automatic scrapers. Mean yearly yield was approximately 10,000 kg per cow per annum. A professional foot trimmer attended the herd each month; typically cows at the end of lactation and with mis-shapen feet were trimmed, with a minimum routine foot trim once per year. Daily observations of the herd by senior herdsman identified lame cows which were treated under veterinary direction using standard protocols, generally within 2 to 3 days. Body

condition score was recorded at 60 day intervals throughout the study period, by the head herdsman with appropriate training to prevent drift in scoring, on a scale of 0 to 5 in 0.5 increments. Health, production, BCS and lameness treatments were recorded in Interherd (National Milk Records).

5.2.2 Statistical analysis

To account for the longitudinal nature of the data, risk factors where repeated events could occur were lagged (e.g. BCS, previous lameness and milk yield) and frailty models constructed to take into account repeated measures of the outcome (lameness events). The main difference between Herd A and Herd B was in defining lameness events; Herd A was based on weekly locomotion scoring whilst Herd B was based on treatment for lameness from the farmer's records. 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.

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

Data handling for Herd A are described in detail in Chapter 2. For Herd B, data handling had been conducted previously including removal of incorrect records or unusual data (< 1% of the data).

Binary outcomes investigated for Herd A were repeated lameness events; locomotion score = 3, 4 or 5 (Model 5.1a) and a higher lameness severity threshold with locomotion score = 4 or 5 for two consecutive weeks (Model 5.1b). The model outcome in Herd B was also binary; yes or no for treatment for of lameness (all causes included; sole haemorrhage (SH), sole ulcer (SU)/white line disease (WLD) and digital dermatitis (DD)) (Model 5.2). 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). A minimum burn-in of 1,000 iterations was used during which model convergence occurred; these iterations were discarded to ensure confidence in the posterior distributions (Green et al., 2004). Parameter estimates were based on a further 9,000 iterations and chain mixing and stability were evaluated visually.

Models took the form;

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

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

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

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_v^2) and residual error.

Explanatory variables for Herd A and B tested in the model are described in Table 5.1. Variables of interest for were time since previous lameness and lagged BCS.

Table 5.1 Description of explanatory variables for Herd A (724 cows) and Herd B (1,040 cows) tested in Models 5.1a, 5.1b and 5.2.

Explanatory variable	Continuous or categorical
Herd A	
Weeks in milk	Continuous; polynomial
Week of the study	Continuous; polynomial
Parity	Categorical; 1 to 4+
Age at first calving	Categorical; < 24, 24 to 27, 28 to 30, 31 to 33 and > 33 months
BCS change 0 to 4 weeks post-calving	Categorical; 0 = loss, 1 = no change, 2 = gain
BW lagged by 1 through to 16 weeks previously	Categorical; < 550, 550 to 700 and > 700 kg
Assessor of locomotion and body condition	Categorical; 1 to 5

Feed – genetic group	Categorical; LF-C, low-forage select: LF-S, high forage control: HF-C, high forage select: HF-S, dry-control: D-C, dry-select: D-S, other-control: O-C, other-select: O-S, where other represents all management groups outside of LF, HF and Dry
Average daily milk yield lagged by 1 through to 16 weeks previously	Categorical; < 12, 12 to 24, 25 to 37, 38 to 50 and > 50 kg
Time since previous lameness	Categorical; 4 week intervals from time t to > 16 weeks
BCS lagged by 1 week through to 16 weeks	Categorical; < 2, 2, 2.25, 2.5, 2.75, 3 and > 3
Herd B	
Parity	Categorical; 1 to 6 +
Year quarter	Categorical; January to March, April to June, July to September and Oct to December
Month in herd	Continuous
Days in milk (at the end of a 30-day period)	Continuous
Milk yield (kg per day measured at the most recent monthly milk recording) lagged by one month	Continuous
Time since previous episode of lameness	Categorical; 30 day intervals from time t to > 120 days
BCS lagged by 0 to 2 months and 2 to 4 months	Categorical: ≤ 2 , > 2

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). Cow level residuals were found to be over-dispersed and non-normal for Models 5.1a and 5.1b. This was due to a small number of cow having a high number of lame events (see Figure 2.6 for frequency distribution of lameness events ($LS \geq 3$) per cow and Figure 5.1 for frequency distribution of lameness events ($LS \geq 4$ on 2 consecutive weeks) per cow). The Hosmer-Lemeshow test (Hosmer and Lemeshow, 1989) was used as a statistical test for goodness-of-fit for posterior predictions at the repeated measures level (as described in section 3.2.2.1); model fit was poor, $\chi^2 = 88.2$, $p = 0.0$ for Model 5.1a (Figure 5.2) and $\chi^2 = 66.0$, $p = 0.0$ (Figure 5.4) for Model 5.1b. Removing random effects improved

model fit to the extent that it was considered very good ($\chi^2 = 5.0$, $p = 0.83$ for Model 5.1a (Figure 5.3), $\chi^2 = 8.7$, $p = 0.46$ for Model 5.1b (Figure 5.5 and Figure 5.6).

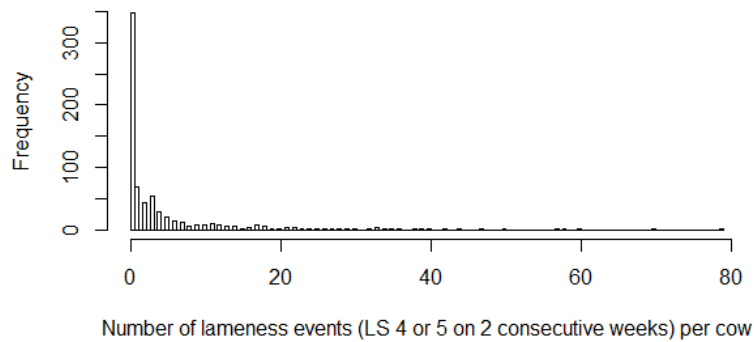


Figure 5.1 Frequency distribution for the number of lameness events per cow where lameness event is defined as locomotion score (LS) 4 or 5 on two consecutive weeks for records obtained from a total of 724 cows for the study period 2003 to 2011.

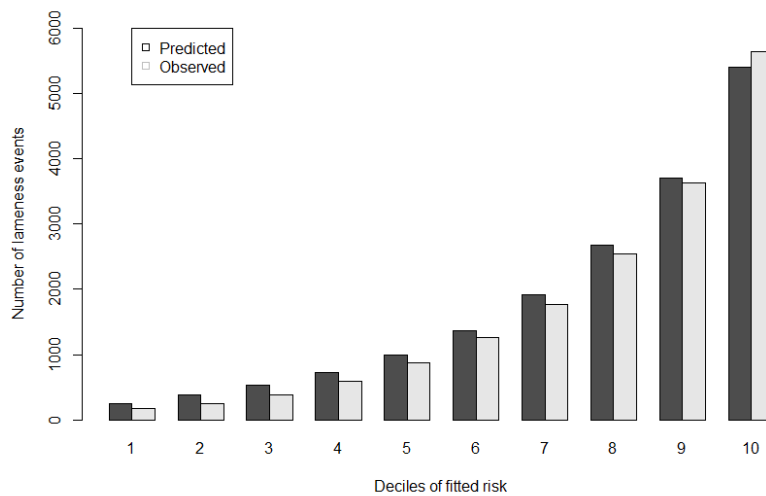


Figure 5.2 Observed and predicted repeat lameness events ($LS \geq 3$) per decile of fitted risk values. Posterior predictions were from Model 5.1a including random effects, constructed using records obtained from 724 cows for the study period 2003 to 2011.

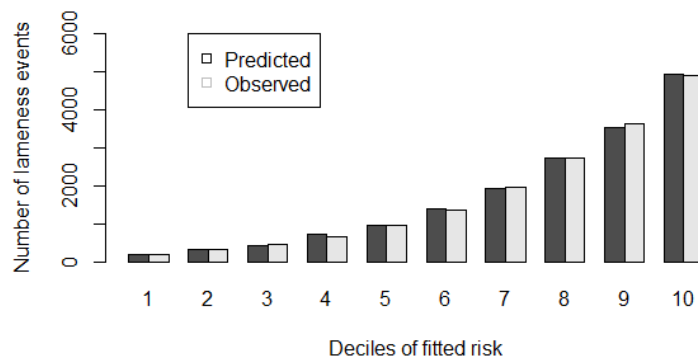


Figure 5.3 Observed and predicted repeat lameness events ($LS \geq 3$) per decile of fitted risk values. Posterior predictions were from Model 5.1a excluding random effects, constructed using records obtained from 724 cows for the study period 2003 to 2011.

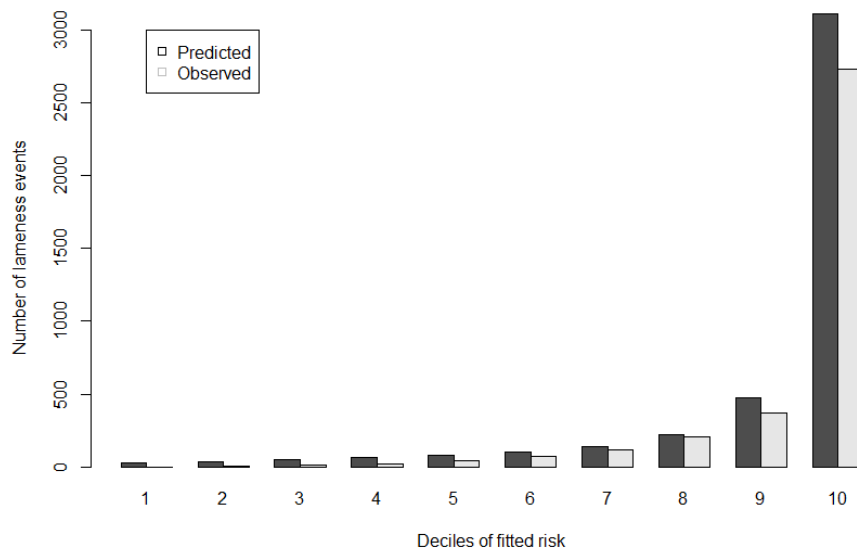


Figure 5.4 Observed and predicted repeat lameness events ($LS \geq 4$ on 2 consecutive weeks) per decile of fitted risk values. Posterior predictions were from Model 5.1a including random effects, constructed using records obtained from 724 cows for the study period 2003 to 2011.

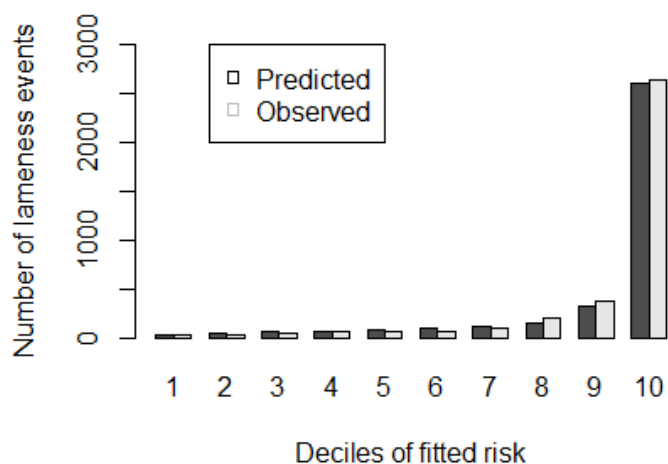


Figure 5.5 Observed and predicted repeat lameness events ($LS \geq 4$ on 2 consecutive weeks) per decile of fitted risk values. Posterior predictions were from Model 5.1b excluding random effects, constructed using records obtained from 724 cows for the study period 2003 to 2011.

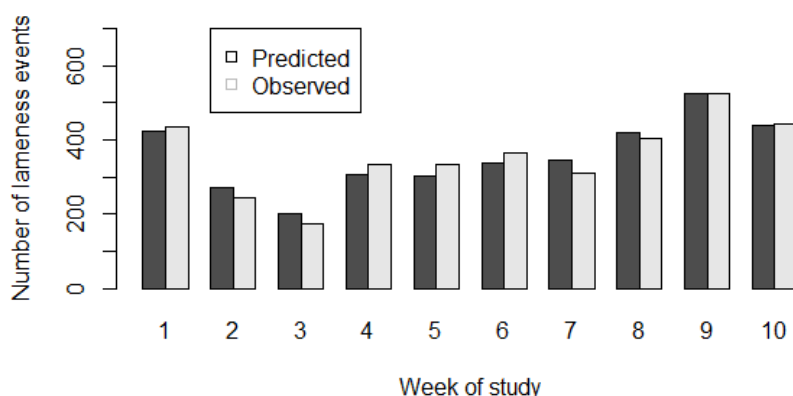


Figure 5.6 Observed and predicted repeat lameness events ($LS \geq 4$ on 2 consecutive weeks) per week of study category (categories 1 to 10 represent weeks 0 to 421 in blocks of 50 weeks). Posterior predictions were from Model 5.1b excluding random effects, constructed using records obtained from 724 cows for the study period 2003 to 2011.

Results from modelling

Herd A: Of the 724 cows in Herd A, 674 (93.0%) and 375 cows (51.8%) were ever lame in Model 5.1a ($LS \geq 3$) and 5.1b ($LS \geq 4$ on two consecutive weeks), respectively. There were a total of 79,565 and 78,698 cow weeks at risk in Model 5.1a and 5.1b, respectively. The number of lameness events was 17,114 and 3,572 respectively for Model 5.1a and 5.1b. 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 Model 5.1a and 5.1b. The proportion of observations where there was a previous lameness event 1 to 4 weeks 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 5.1a. For model 5.1b the proportion of observations where there was a previous lameness event 1 to 4 weeks 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. Final models are reported in Table 5.2 and Table 5.3 for Model 5.1a and 5.1b respectively. Assessment of model fit for both models was considered good. Table 5.5 summarises the odds ratios and 95% credible intervals for BCS and previous lameness from Models 5.1a, 5.1b and 5.2 for comparison. For Model 5.1a, BCS 3-weeks previously was positively associated with the lameness outcome $LS \geq 3$. 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 5.1b, BCS one week previously had the largest effect size and therefore left in the final model. As for Model 5.1a, 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) = 1.63 (1.28 to 2.07)). 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) = 18.71 (16.93 to 20.68)) 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. 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. Results from the final model are reported in Table 5.4. Model fit was good. Table 5.5 summarises the odds ratio for the explanatory variables of interest (BCS and previous

lameness) for all models including Model 5.2 **Error! Reference source not found..** For Model 5.2, BCS ≤ 2 in the last 0 to 2 months or 2 to 4 months was associated with an increased risk of lameness (all causes; SU, SH/WLD and DD) compared with BCS > 2 ; OR (95% credible interval) = 1.59 (1.38 to 1.85) and 1.36 (1.11 to 1.68) 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 (95% credible interval) = 19.51 (15.64 to 24.35).

Table 5.2 Final logistic regression model (Model 5.1a) with the outcome lameness events based on locomotion scores ≥ 3 .

Intercept	Coefficient: - 3.433		
Variable	Odds ratio	Lower 95% CI ¹	Upper 95% CI
BCS 3-weeks previously			
3	Baseline		
< 2	1.29	1.15	1.45
2	1.14	1.02	1.27
2.25	1.11	1.00	1.23
2.5	1.07	0.96	1.19
2.75	1.03	0.91	1.16
> 3	1.05	0.91	1.22
Previous lameness (LS 3,4 or 5)			
None	Baseline		
1 to 4 wk	3.65	3.48	3.83
None	Baseline		
5 to 8 wk	2.15	2.05	2.27
None	Baseline		
9 to 12 wk	1.64	1.53	1.77
None	Baseline		
13 to 16 wk	1.52	1.44	1.59
None	Baseline		
> 16 wk	1.21	1.12	1.31
Weeks in milk (WIM)			
WIM^1	1.031	1.00	1.01
WIM^2	1.00	1.00	1.00
Parity			
1	Baseline		
2	1.10	1.03	1.17
3	1.46	1.37	1.57
4 +	2.09	1.92	2.28
Age at first calving (months)			
< 25	Baseline		
24 to 27	1.27	1.18	1.35
28 to 30	1.59	1.45	1.74
31 to 33	1.28	1.08	1.53
> 33	2.32	1.93	2.79
Feed-genetic group ²			
LF:C	Baseline		

LF:S	1.03	0.97	1.10
HF:C	0.69	0.65	0.74
HF:S	0.67	0.63	0.72
Dry:C	0.68	0.61	0.76
Dry:S	0.69	0.61	0.78
Other:C	2.14	1.75	2.63
Other:S	2.97	2.40	3.67
Locomotion score assessor			
1	Baseline		
2	0.43	0.40	0.46
3	0.80	0.74	0.86
4	0.89	0.82	0.96
5	0.77	0.60	0.99
Milk yield 3-weeks previously (kg)			
< 12	Baseline		
12 to 24	0.88	0.74	1.05
25 to 37	0.80	0.67	0.96
38 to 50	0.77	0.64	0.93
> 50	0.65	0.52	0.82
Body weight 3-weeks previously (kg)			
< 550	Baseline		
500 to 700	0.89	0.84	0.96
> 500	0.81	0.74	0.90
BCS change 0 to 4 weeks post-calving			
No change	Baseline		
Loss $\geq$ 0.25	1.13	1.04	1.24
Gain $\geq$ 0.25	1.08	0.95	1.24
Logarithm week ³ (LOGwk)			
LOGwk^1	1.37	1.31	1.44
LOGwk^2	1.35	1.29	1.40
LOGwk^3	1.04	1.03	1.05

¹CI = credible interval

²Feed-genetic groups include low forage (LF), high forage (HF), control (C) and select (S). Dry refers to dry cows and other refers to all other management groups outside of LF, HF and Dry.

³Week refers to week of the study period.

Table 5.3 : Final logistic regression model (Model 5.1b) with the outcome lameness events based on 2 consecutive weekly locomotion scores 4 or 5.

Intercept	Coefficient: - 5.263		
Variable	Odds ratio	Lower 95% CI ¹	Upper 95% CI
BCS 3-weeks previously			
3	Baseline		
< 2	1.63	1.28	2.07
2	1.41	1.12	1.79
2.25	1.26	1.00	1.79
2.5	1.04	0.82	1.32
2.75	1.09	0.82	1.45
> 3	1.14	0.83	1.56
Previous lameness (LS 3,4 or 5)			
None	Baseline		
1 to 4 wk	18.71	16.93	20.68
None	Baseline		
5 to 8 wk	1.99	1.78	2.22
None	Baseline		
9 to 12 wk	1.51	1.34	1.69
None	Baseline		
13 to 16 wk	1.48	1.32	1.67
None	Baseline		
> 16 wk	1.62	1.47	1.80
Weeks in milk (WIM)			
WIM^1	1.03	1.02	1.04
WIM^2	1.00	1.00	1.00
Parity			
1	Baseline		
2	1.40	1.22	1.61
3	1.61	1.40	1.86
4 +	2.70	2.28	3.19
Age at first calving (months)			
< 25	Baseline		
24 to 27	1.36	1.15	1.62
28 to 30	1.40	1.12	1.75
31 to 33	1.24	0.77	1.99
> 33	2.24	1.59	3.14
Feed-genetic group ²			
LF:C	Baseline		
LF:S	1.12	0.98	1.28
HF:C	0.60	0.51	0.70
HF:S	0.68	0.59	0.79
Dry:C	0.62	0.49	0.80
Dry:S	0.63	0.49	0.40
Other:C	3.41	2.47	4.73
Other:S	3.51	2.55	4.83
Milk yield 1-week previously (kg)			
< 12	Baseline		
12 to 24	0.71	0.51	0.99
25 to 37	0.62	0.45	0.87
38 to 50	0.56	0.39	0.80
> 50	0.51	0.32	0.81

Body weight 1-week previously (kg)			
< 550	Baseline		
500 to 700	0.76	0.65	0.89
> 500	0.55	0.45	0.68
Logarithm week ³ (LOGwk)			
LOGwk^1	1.12	1.03	1.21
LOGwk^2	1.39	1.29	1.49
LOGwk^3	1.07	1.05	1.08

¹CI = credible interval

²Feed-genetic groups include low forage (LF), high forage (HF), control (C) and select (S). Dry refers to dry cows and other refers to all other management groups outside of LF, HF and Dry.

³Week refers to week of the study period.

Table 5.4 Final logistic regression model (Model 5.2) with the outcome lameness based on lameness treatment for all causes.

Intercept	Coefficient: - 2.706		
Variable	Odds ratio	Lower 95% CI ¹	Upper 95% CI
BCS last 0 to 2 months			
BCS > 2	Baseline		
BCS ≤ 2	1.59	1.38	1.85
BCS last 2 to 4 months			
BCS > 2	Baseline		
BCS ≤ 2	1.36	1.11	1.68
Previous lameness			
None	Baseline		
1 to 30 days ago	19.51	15.64	24.35
31 to 60 days ago	13.65	10.67	17.48
61 to 90 days ago	14.43	10.67	19.51
91 to 120 days ago	13.82	10.02	19.06
> 120 days ago	15.88	12.45	20.25
Year quarter			
January - March	Baseline		
April - June	1.13	0.96	1.32
July - September	1.30	1.10	1.52
October - December	1.04	0.87	1.24
Parity			
1	Baseline		
2	0.46	0.38	0.57
3	0.49	0.40	0.61
4	0.46	0.36	0.58
5	0.45	0.34	0.60
6 +	0.40	0.30	0.54
Yield (current)			
Kg per day	0.98	0.97	0.99
Yield (previous month)			
Kg per day	1.01	1.00	1.02
Days in milk			
305 DIM	1.00	1.00	1.00
Random term (variance and SE ²)		0.55 (0.07)	

¹CI = credible interval

²SE = standard error

Table 5.5 Summary of the results of Model 5.1a, 5.1b and 5.2 for explanatory variables body condition score (BCS) and previous lameness. Model 5.1a: Final model of lameness events based on locomotion score 3, 4 or 5 in the Scotland's Rural College (SRUC) Research and Innovation Centre dairy herd 2003 to 2011. Model 5.1b: Final model of lameness events based on 2 consecutive weekly locomotion scores 4 or 5 in the Scotland's Rural College (SRUC) Research and Innovation Centre 2003 to 2011. Model 5.2: Final model of lameness treated for all causes in a dairy herd in Somerset, UK.*

Variable	N ¹	Odds ratio	Lower 95% CrI ²	Upper 95% CrI
Model 5.1a; outcome = LS 3, 4 or 5				
Total N = 79565				
BCS 3-weeks 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 5.1b; outcome = 2 consecutive weeks LS 4 or 5				
Total N = 78698				
BCS 1-week previously				
3	3718	Baseline		
< 2	15122	1.63	1.28	2.07
2	18910	1.41	1.12	1.79
2.25	20990	1.26	1.00	1.79
2.5	11632	1.04	0.82	1.32
2.75	4481	1.09	0.82	1.45
> 3	2119	1.14	0.83	1.56
Previous lameness (2 consecutive LS 4 or 5)				
None	67770	Baseline		
1 to 4 wk	6181	18.71	16.93	20.68
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	1.47	1.80
Model 5.2; outcome = all causes of lameness (SH, SU/WLD and DD) ³				

BCS last 0 to 2 months			
BCS > 2	Baseline		
BCS ≤ 2	1.59	1.38	1.85
BCS last 2 to 4 months			
BCS > 2	Baseline		
BCS ≤ 2	1.36	1.11	1.68
Previous lameness			
None	Baseline		
1 to 30 days ago	19.51	15.64	24.35
31 to 60 days ago	13.65	10.67	17.48
61 to 90 days ago	14.43	10.67	19.51
91 to 120 days ago	13.82	10.02	19.06
> 120 days ago	15.88	12.45	20.25

¹N = Number of observations

²CrI = credible interval

* Only coefficients for explanatory variables BCS and previous lameness are reported here. Other covariates tested are reported in Table 5.3, 5.4 and 5.5 for Model 5.1a, 5.1b and 5.2 respectively

³SH, SU/WLD and DD = sole haemorrhage, sole ulcer/white line disease and digital dermatitis

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 a 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 5.1a, 5.1b and 5.2).

The simulation approach used posterior predictions of the number of lameness events to estimate PAF (Gelman, 2000). Models 5.1a, 5.1b and 5.2 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 for coefficients from Models 5.1a and 5.2 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 explored are summarised in Table 5.6.

Table 5.6 Description of scenarios investigated for two UK dairy herds to estimate PAF for BCS and previous lameness.

Herd	Model	Outcome (interval)	Scenario	description
A	5.1a	LS ¹ 3,4 or 5 (weekly)	BCS gain	Whole herd gains 0.5 BCS if < 3
			BCS loss No previous lameness	Whole herd loses 0.5 BCS Effect of all previous lameness events removed
	5.1b	2 consecutive LS 4 or 5 (weekly)	BCS gain	Whole herd gains 0.5 BCS if < 3
			BCS loss No previous lameness	Whole herd loses 0.5 BCS Effect of all previous lameness events removed
B	5.2	Clinical lameness; all causes (60 days)	No previous lameness	Effect of all previous lameness events removed

¹LS = locomotion score

Models 5.1a, 5.1b and 5.2 were imported into OpenBUGS alongside raw data from respective herds. For the BCS scenarios, additional categories were created for 0.5 added to the BCS score for each cow with BCS < 3 for each week in the herd (i.e. 0.5 BCS gain across the whole herd apart from cows with BCS 3 or above) and 0.5 taken away from each BCS score (i.e. 0.5 BCS loss across the whole herd) in the Herd A dataset; data were imported to OpenBUGS. Exposure distributions for the BCS categories are summarised in Table 5.7. 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 for the herd exposed to each of the scenarios (0.5 BCS gain and 0.5 BCS loss) and not exposed to these distributions (i.e. the BCS distribution of the raw herd data as a baseline) were predicted. Posterior predictions for PAF were calculated as the difference in number of lameness events as a proportion of the total number of lameness events. For the previous lameness scenario, the raw herd data was used as the baseline scenario, with exposure to previous lameness removed for the altered scenario (i.e. all coefficients for previous lameness categories equal to zero). The number of lameness events for the herd exposed and unexposed to previous lameness events were predicted. Posterior predictions for PAF were calculated as the difference in the number of lameness events as a proportion of the total number of lameness events.

To remove the effect of lameness that occurred just prior to a risk period and explore only the impact of lameness events that occurred earlier, Models 5.1a, 5.1b and 5.2 were used to estimate PAF of lameness events that occurred a minimum of 5 weeks before a current case and a minimum of 16 weeks before a current case. For models 5.1a and 5.1b the effect of previous lameness events that occurred in the 4 to 8, 9 to 12, 13 to 16 and > 16 weeks 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.

Table 5.7 Proportion of observations with body condition score (BCS) recoded in each category for the scenarios investigated for Herd A for the datasets used in Model 5.1a and 5.1b. 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

5.3 Results

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

The PAF estimated using both closed and simulation methods are presented in Table 5.8.

Model 5.1a. 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 5.1b. 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%). 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 5.2. 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.

Table 5.8 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 (Model 5.1a and 5.1b) and 1,040 cows from a 600-cow herd in Somerset (Model 5.2)*.

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. 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.

^aN: 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)

^bBCS 3 weeks previous to lameness events, ^cBCS 1 week previous to lameness events, ^dd = day

5.3.2 Estimating PAF for BCS and previous lameness.

Results of the scenarios investigated are presented in Table 5.9.

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 locomotion score 3, 4 or 5 (Model 5.1a). The median (95 % credible interval) PAF 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 increased to 2 consecutive locomotion scores 4 or 5 (Model 5.1b) there was a greater impact on the reduction in lameness levels; median (95 % credible interval) PAF was -8.06% (-13.12 - -2.22%). A loss in 0.5 BCS across all BCS score categories for Model 5.1a resulted in an additional 1030 predicted lameness events and the median (95% credible interval) PAF 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 (95% credible interval) PAF for this exposure using Model 5.1b, where lameness severity threshold was increased, was 11.2 % (5.52 - 17.33%).

Previous Lameness. When the effect of exposure to previous lameness was removed across the whole herd the predicted number of lameness observations was reduced by 7576 observations for Herd A where the outcome was locomotion score 3, 4 or 5 (Model 5.1a) and 2812 observations where outcome was locomotion score 4 or 5 on 2 consecutive weeks (Model 5.1b). 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 5.1a and 5.1b), respectively. When the effect of exposure to previous lameness was removed across the whole herd in Herd B (Model 5.2) 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 30.45% (17.88 to 42.03%), respectively for Models 5.1a, 5.1b and 5.2. 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 5.1a, 5.1b and 5.2.

Table 5.9 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 A							
Model 5.1a	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 5.1b	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 5.2	No previous lameness	1998	346	-1652	-82.69	-79.28	-85.61

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

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

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

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

5.4 Discussion

Findings demonstrated the potential importance of previous lameness events as a risk factor that can have an overwhelming impact on the total burden of lameness at the herd level. Using simulation to estimate PAF enabled the impacts of changes in BCS and occurrence of previous lameness events on the total lameness events within two herds to be explored. This novel approach to estimating PAF for lameness risk factors used longitudinal data with repeated measures.

5.4.1 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 may be attributable to exposure to previous lameness events (regardless of when they occurred), indicating that this may be an important risk factor.

When the effect of lameness events that occur > 4 weeks and > 16 weeks previously was investigated, the contribution from previous lameness reduced markedly, although it was still considerable. This finding suggests that lameness might last for some duration 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 may be 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 this. The distribution of lameness events show that some of the cows have a very high number of repeated lameness events whilst others have very few (Figure 2.6 and Figure 5.1). This could potentially be influenced by the duration of time spent 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 and/or animal-based factors that could explain the high number of repeat lameness events in some 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 and/or animal-based interaction which is important in consigning a cow to repeat lameness events. In addition, findings from this chapter 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 (Green et al., 2014, Randall et al., 2015, Hirst et al., 2002b). 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 in Chapter 4; more severe CHDLs 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 (Solano et al., 2015, Randall et al., 2015, Barker et al., 2009). 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, Woolf, 2011, Latremoliere and Woolf, 2009). 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 chapter 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.

5.4.2 Body Condition Score

The results of this chapter demonstrated the potential impact of changing BCS across the whole herd; 4% (1 – 6%) of all lameness events (weekly locomotion score 3, 4 or 5) may be prevented 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). Results from Chapter 3 showed that cows with BCS < 2 3-weeks previously 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 population attributable risks and 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% CI 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 chapter are similarly relatively small compared with the impact of previous lameness events. Although, in herds with fewer repeated lameness events, BCS could be relatively more important.

5.4.3 Comparison of closed and simulation-based approach 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 to epidemiology which focused 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. Formulas for estimating PAF require a number of assumptions to be fulfilled in order to obtain valid estimates. These include; a causal relationship between the risk factors and disease, that risk factors are independent i.e. changing the risk factors being considered does not influence other risk factors that may impact on disease risk, and that attainment of

unexposed disease risk occurs immediately after exposure to a risk factor is removed. Using a simulation approach mitigates some of these assumptions, for example the regression models provide adjusted conditionally independent risk estimates for the risk factors of interest after controlling for other risk factors, which propagates through the simulation to provide the estimate of PAF. Similarly, risk associated with previous exposure to low BCS and previous lameness are controlled for through modelling longitudinal data. The simulation approach to estimating PAF does however assume that there is a casual relationship between the risk factors BCS and previous lameness and the outcome lameness. An increasing body of evidence indicates that this association is likely to be causal and whilst this needs to be substantiated, it was considered that this assumption was entirely reasonable.

Use of simulation for estimating PAF with repeated measures data in this way for lameness risk factors is a novel approach, therefore estimates using a closed equation method were compared to those using simulation. In this study the formula applicable for when there are 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 are more realistic figures for PAF.

5.4.4 Study Limitations and Generalisability

The main findings of this chapter were demonstrated in two UK herds with different methods of lameness detection and different environments. The PAF of comparable scenarios were similar in both herds giving an indication for possible generalisability of these findings, in a qualitative way, 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 locomotion score ≥ 3 was 21.3% (section 2.4), which is similar to prevalence rates reported in other UK studies (section 1.3.2).

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 chapter.

5.5 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 (weekly locomotion score 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 weekly locomotion score 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 and/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 also potentially be important in preventing an escalation of repeated lameness events. A novel approach to estimating PAF for lameness risk factors using simulation enabled complex scenarios to be investigated whilst accounting for variability within the herds in this study.

CHAPTER 6: Using Simulation to Investigate the Effect of Adopting Different Culling Rates on Lameness Prevalence in Dairy Herds

6.1 Introduction

The risk of lameness in dairy cattle has been demonstrated to increase with both age and the occurrence of previous lameness (Randall et al., 2015, Green et al., 2014, Barker et al., 2009). Hirst et al. (2002b) reported an increased hazard in 2nd and 3rd lactation cows which had previously been lame in their first lactation, suggesting that underlying pathology may be carried over from one lactation to the next. This association was also demonstrated in Chapter 4; heifers with more severe CHDLs had an increased risk of lameness across all lactations investigated (Randall et al., 2016). Findings from Chapter 5 showed that previous lameness can potentially contribute up to approximately 80% of the total lameness events within the two herds investigated and is therefore an important risk factor to consider in controlling lameness. One study investigating the underlying pathological changes associated with lameness has recently shown that bone development, ‘exostosis’, on the caudal aspect of the distal phalanx present at slaughter is associated with a history of CHDL (Newsome et al., 2016). It is possible that irreparable anatomical damage may occur within the foot with the occurrence of CHDL, which is one explanation for why lameness risk increases with age in dairy cows. The rationale therefore exists that with an increasing age profile in a herd, lameness prevalence would also be expected to increase and vice versa.

The age profile of a herd is influenced by the herd culling rate, which describes the percentage of cows removed from a herd (Hadley et al., 2006); higher culling rates means a shorter herd lifespan. Average annual culling rates have been reported in the region of 22 to 25% for the UK (Whitaker et al., 2000, Bell et al., 2010b), 28.2% in Canada (CDIC, 2016) and 32 to 35% in the US (Hadley et al., 2006, De Vries et al., 2010). Tsuruta et al. (2005) highlighted that as a result of increased culling rates the age distribution of US Holsteins has shifted towards younger ages; average parities had fallen from 3.4 to 2.8 between 1989 to 2004 (Tsuruta et al., 2005, Nieuwhof et al., 1989) and a reported 83% of cows in large US herds are within their first three lactations (De Vries et al., 2010). In contrast, Maher et al. (2008) reported that in Irish dairy herds with an average culling rate of 21.3%, retention of cows beyond the third lactation was 65-68%. Brickell and Wathes (2011), in a prospective study of 18 English dairy farms reported a 19 and 24% culling rate for lactation 1 and 2, respectively. Fifty five percent

of replacement heifers calved for the third time, however this figure ranged from 32 to 80% across individual farms reflecting the wide variability in culling rates. Similarly, Sherwin et al. (2016) reported culling rates in primiparous heifers across 437 UK dairy herds at 15.9%. Reasons for culling have traditionally been defined into two categories; voluntary and involuntary. Voluntary culling refers to the removal of otherwise apparently healthy, low-yielding animals or selling of excess animals in the herd i.e. 'cows sold for dairy purposes' (Fetrow et al., 2006). This provides an opportunity to increase herd profitability and is considered normal apart from the low production. Whereas involuntary culling refers to the forced removal of cows from the herd due to death, disease or reproductive problems. Fetrow et al. (2006) highlighted issues with the use of these terms as not being reflective of culling decisions. Alternative definitions for culling reasons suggested were 'forced' and economic'. Beaudeau et al. (1993) estimated that more than half of all culling are associated with health disorders, whilst Bell et al. (2010a) Found that in a study investigating risk factors for culling using data from the SRUC Crichton Royal research herd, Scotland between 1990 and 2008, 68% of cows culled were classified as involuntary. There is often a trade-off between reasons for culling and although it is important to differentiate between them, the overall culling rate does not provide information on the culling situation on an individual farm (Weigel et al., 2003).

As the risk of lameness is influenced by age and the previous occurrence of lameness it is possible that with variations in culling rates this will have an impact on the level of lameness in herds. Benchmarking and targets for lameness levels may provide motivation to control lameness and act as a method to assess welfare on-farm (AssureWel, 2017, Whay and Shearer, 2017), however considerations regarding differences between farms may be appropriate. For example, a herd with a higher culling rate and younger age distribution may be able to more easily achieve lower lameness levels compared with a comparable herd with lower culling rates. In order to explore the effect of culling rates and the occurrence of previous lameness on lameness prevalence, simulation models were used to investigate a number of different scenarios. Simulation is a useful tool for investigating and assessing the impacts of inputs on the outputs of a complex system and can aid in the interpretation of results from complex statistical models (Hudson et al., 2015). For example, in this study, distributions for input parameters, such as baseline lameness risk over time and risk associated with a

previous lameness event, were explored in terms of their impact on the output (lameness prevalence) for different culling rate scenarios.

The aim of this chapter was to investigate the effect of herds adopting differing culling rates on lameness prevalence, by using simulation to investigate possible herd scenarios. Understanding how herds with varying culling rates differ in terms of lameness prevalence will aid in setting benchmarks and targets for lameness prevalence that take these herd differences into account as well as identifying herds where increased focus on lameness prevention and control may be necessary.

6.2 Materials and methods

6.2.1 Study herd

The study herd from which data were obtained and descriptions of the data are detailed in Chapter 2.

6.2.2 Statistical analysis

Data handling is described in detail in Chapter 2. The binomial logistic regression model (Model 5.1a) described in Chapter 5, was used as the basis for constructing a model to provide estimates for input parameters for simulation. Model 5.1a was simplified so that the only covariates included were those required as input parameters for simulation, with the model then averaging across all other covariates that were previously included in Model 5.1a. This meant that the predicted risk could then be calculated from the coefficients for these covariates.

6.2.3 Modelling

A binomial logistic regression model was constructed in MLWin 2.36 (Rabash et al., 2009) with the outcome lameness event, based on weekly locomotion score (LS) 3, 4 or 5. Parameter estimation for model parameters was carried out by iterative generalized least square procedures (Goldstein, 2003). Explanatory variables included in the model were those required as input parameters for the simulation model; the mean predicted risk and 95% credible intervals could then be calculated from model estimates for these parameters.

The model took the form;

$Lame_i \sim \text{Bernoulli}(\text{probability} = \pi_i)$

$$\text{Logit}(\pi_i) = \alpha + \beta_1 X_i$$

Where subscript i denotes the i th observation. π_i = probability of a lame outcome for the i th observation. α = intercept value, β_1 = vector of coefficients for X_i , X_i = vector of covariates associated with each observation. Explanatory variables included in the model were; parity (categorical variable from 1 to 5+), month in milk (as a polynomial to the power 2), lameness event 1 to 4 weeks previously (0 = no or missing, 1 = yes), lameness event 5 to 8 weeks previously (0 = no or missing, 1 = yes), lameness event 9 weeks or more previously (0 = no or missing, 1 = yes). Model coefficients (Table 6.1) were used to calculate estimates of predicted risk using Equation 6.1. Relative risk (RR) for previous lameness categories for each parity were also calculated using Equation 6.2.

$$\text{Risk} = \frac{\exp(\sum \log \text{odds of all covariates and the intercept})}{1 + (\exp(\sum \log \text{odds of all covariates and the intercept}))} \quad (6.1)$$

$$\text{RR} = \frac{\text{Predicted risk (previous lameness covariate = 1)}}{\text{Predicted risk for baseline (previous lameness covariate = 0)}} \quad (6.2)$$

Table 6.1 Logistic regression model constructed to estimate coefficients for previous lameness, parity and month in lactation using data obtained from 724 cows.

	Coefficient	S.E
Cons	-2.424	0.036
Previous lameness		
1 to 4 weeks (baseline = none or missing)	1.319	0.021
5 to 8 weeks (baseline = none or missing)	0.928	0.021
9 or more weeks (baseline = none or missing)	0.145	0.032
Parity		
1	Baseline	
2	-0.017	0.026
3	0.287	0.027
4	0.511	0.035
5 +	0.423	0.046
Month in lactation		
Month^1	-0.054	0.007
Month^2	0.002	0

Posterior predictions were used to assess model fit by visual comparison to the observed data (Gelman et al., 1996); Figure 6.1. The Hosmer-Lemeshow test (Hosmer and Lemeshow, 1989) was used as a statistical test for goodness-of-fit (as described in section 3.2.2.1); $\chi^2 = 16.48$, $p = 0.04$ indicating that model fit was adequate.

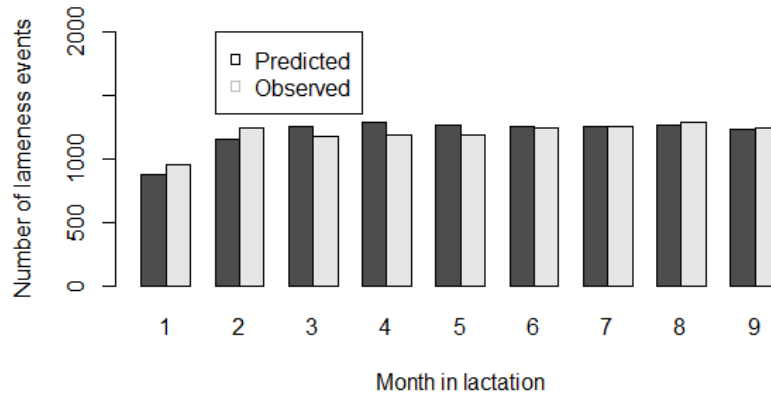


Figure 6.1 Observed and predicted repeat lameness events ($LS \geq 3$) per month in lactation. Model constructed using records obtained from 724 cows.

6.2.4 Simulation

The simulation model was constructed in OpenBUGS version 3.2.3 (Lunn et al., 2009) based on the structure shown in Figure 6.2 for heifers and Figure 6.3 for COWS.

Lactation 1

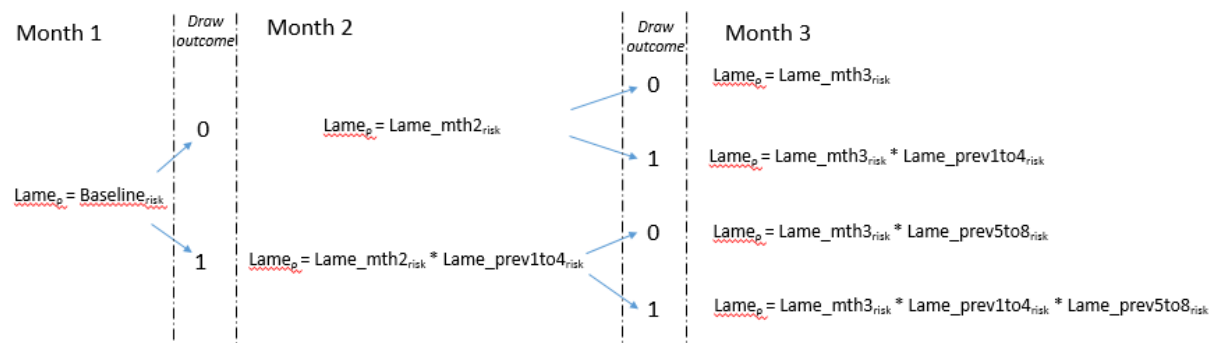


Figure 6.2 Flow diagram to demonstrate the structure of the simulation model to investigate the effect of culling rates on lameness prevalence. Blue arrow represents a draw from a binomial distribution with probability = $Lame_p$ to give a binary outcome for lameness. $Lame_p$ = lameness event probability. $Baseline_{risk}$ = lameness risk for month one for each parity. $Lame_{prev1to4}$ = lameness risk for previously lame in the previous 1 to 4 weeks. $Lame_{prev5to8}$ = lameness risk for previously lame in the previous 5 to 8 weeks.

Lactation 2

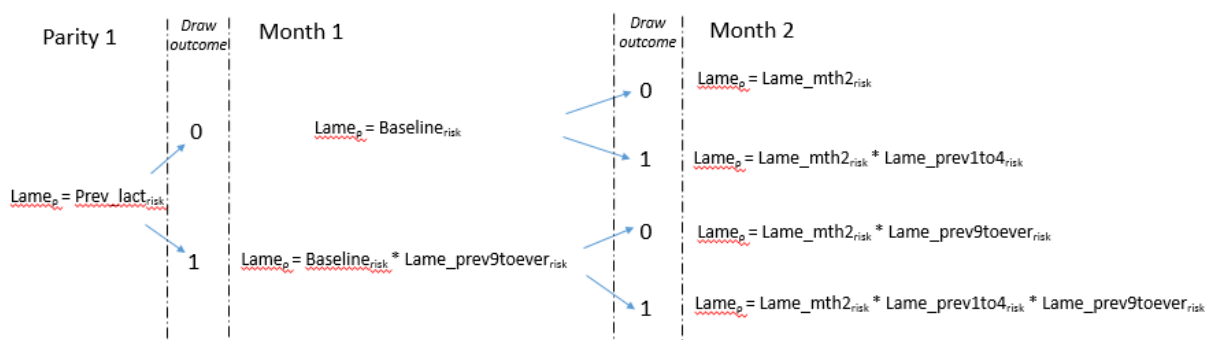


Figure 6.3 Flow diagram to demonstrate the structure of the simulation model to investigate the effect of culling rates on lameness prevalence. Blue arrow represents a draw from a binomial distribution with probability = $Lame_p$ to give a binary outcome for lameness. $Lame_p$ = lameness event probability. $Prev_lact_risk$ = lameness risk carried over from the previous lactation. $Baseline_risk$ = lameness risk for month one for each parity. $Lameness_prev1to4$ = lameness risk for previously lame in the previous 1 to 4 weeks. $Lameness_prev9toever$ = lameness risk for previously lame in the previous 9 weeks or more.

For each month of lactation a matrix of possible outcomes (binary yes or no for lameness event) were constructed taking into account outcomes from previous months as shown in Figure 6.2 and Figure 6.3. In this way, both changes in baseline risk over time and risk associated with previous lameness in previous months or lactations could be incorporated into the probability of the outcome being lame or not lame for each month. Distributions for these input parameters were derived from the logistic regression model described above in section 6.2.3 as well as the observed risk from raw herd data. Although this model and hence distributions were derived using data from one herd, the aim of simulation was to investigate how changes in culling rates applies to other herds. It is unknown where the mean effect for the parameters month 1 baseline risk and previous lameness risk in other herds might be, therefore a range of distributions were explored, including both normal and uniform distributions.

A total of 9 months were simulated for each lactation, for parity 1 through to 5 and greater. In lactation one the raw herd data was used to calculate the baseline risk in month 1 because the predicted risk for month 1 from the logistic regression model would not include any additional risk of having been previously lame. For each month of each lactation the number of lameness events were predicted. Input parameters included baseline risk for month one of each parity, risk for month of lactation and risk from previous lameness events (1 to 4 weeks, 5 to 8 weeks and 9 or more weeks previously). The distributions of input parameters that were investigated (baseline risk and previous lameness risk) are summarised in Table 6.2. For the baseline month 1 risk, a uniform distribution was selected to give an equal probability that the risk will be at

any value within the whole range given. Ranges explored included; a narrower range around the mean risk from model estimates or raw data and a wider range between the mean risk estimated from the model and the observed risk from raw data. For previous lameness risk, the distributions explored included; a uniform distribution between the 95% credible intervals and a normal distribution defined using the mean risk and precision (precision = 1 divided by variance). A uniform distribution around a narrower range of values were also explored using the upper and lower 95% credible intervals to explore what happens if the previous lameness risks were lower or higher respectively. The lameness probability for each month of each lactation was calculated from the number of lameness events divided by the number of animals in that parity. Lameness prevalence was calculated for mid-lactation (month 4) for each parity from the sum of the total number of lameness events across each lactation in month 4 divided by the number of animals in the herd. The model was run for a total of 1,000 iterations, with the first 400 iterations being discarded, during which model convergence occurred. The scenarios investigated for different culling rates are summarised in Table 6.3 and illustrated in Figure 6.4. Culling rates for each lactation were based on figures reported in the literature that are relevant to the UK average as well as exploring the impact of lower and higher culling rates that are observed on individual farms. (Brickell and Wathes, 2011, Sherwin et al., 2016, Whitaker et al., 2000, Bell et al., 2010b). Lameness prevalence in month 4 calculated from the simulation model was compared to the observed data using a culling rate of 25% to test how accurately the model could predict lameness prevalence.

Table 6.2 Distributions for input parameters that were investigated in the simulation model including source for the main variables of interest; baseline risk in month one for each parity and previous lameness risk 1 to 4 weeks previously, 5 to 8 weeks previously and 9 weeks or more previously.

Input variable	Source	Distributions
Baseline month 1 risk		
Parity 1	Observed data	Uniform (0.125,0.126)
Parity 2	Model (mean)	Uniform (0.080,0.0801)
	Observed data	Uniform (0.198,0.1981)
Parity 3	Model (mean) and observed data	Uniform (0.080,0.198)
	Model (mean)	Uniform (0.105,0.1051)
	Observed data	Uniform (0.216,0.2161)
Parity 4	Model (mean) and observed data	Uniform (0.105,0.216)
	Model (mean)	Uniform (0.128,0.1281)
	Observed data	Uniform (0.338,0.3381)
Parity 5	Model (mean) and observed data	Uniform (0.128,0.338)
	Model (mean)	Uniform (0.119,0.1191)
	Observed data	Uniform (0.278,0.2781)

Model (mean) and observed data		Uniform (0.119,0.278)
Previous lameness relative risk		
Parity 1		
Lame_prev1to4	Model (95% CI ¹)	Uniform (3.096,3.308); normal (3.201,4.320)
	Model (lower 95% CI)	Uniform (3.096,3.0961)
	Model (upper 95% CI)	Uniform (3.308,3.3081)
Lame_prev5to8	Model (95% CI)	Uniform (2.232,2.395); normal (2.312,4.913)
	Model (lower 95% CI)	Uniform (2.232,2.2321)
	Model (upper 95% CI)	Uniform (2.395,2.3951)
Lame_prev9toever	Model (95% CI)	Uniform (1.080,1.214); normal (1.145,5.400)
	Model (lower 95% CI)	Uniform (1.080,1.0801)
	Model (upper 95% CI)	Uniform (1.214,1.2141)
Parity 2		
Lame_prev1to4	Model (95% CI)	Uniform (3.103,3.316); normal (3.201,4.308)
	Model (lower 95% CI)	Uniform (3.103,3.1031)
	Model (upper 95% CI)	Uniform (3.316,3.3161)
Lame_prev5to8	Model (95% CI)	Uniform (2.234,2.398); normal (2.315,4.904)
	Model (lower 95% CI)	Uniform (2.234,2.2341)
	Model (upper 95% CI)	Uniform (2.398,2.3981)
Lame_prev9toever	Model (95% CI)	Uniform (1.080,1.214); normal (1.145,5.396)
	Model (lower 95% CI)	Uniform (1.080,1.0801)
	Model (upper 95% CI)	Uniform (1.214,1.2141)
Parity 3		
Lame_prev1to4	Model	Uniform (2.971,3.162); normal (3.066,4.561)
	Model (lower 95% CI)	Uniform (2.971,2.9711)
	Model (upper 95% CI)	Uniform (3.162,3.1621)
Lame_prev5to8	Model	Uniform (2.178,2.330); normal (2.252,5.098)
	Model (lower 95% CI)	Uniform (2.178,2.1781)
	Model (upper 95% CI)	Uniform (2.330,2.3301)
Lame_prev9toever	Model	Uniform (1.078,1.208); normal (1.142,5.474)
	Model (lower 95% CI)	Uniform (1.078,1.0781)
	Model (upper 95% CI)	Uniform (1.208,1.2081)
Parity 4		
Lame_prev1to4	Model	Uniform (2.860,3.032); normal (2.945,4.800)
	Model (lower 95% CI)	Uniform (2.860,2.8601)
	Model (upper 95% CI)	Uniform (3.032,3.0321)
Lame_prev5to8	Model	Uniform (2.128,2.270); normal (2.198,5.280)
	Model (lower 95% CI)	Uniform (2.128,2.1281)
	Model (upper 95% CI)	Uniform (2.270,2.2701)
Lame_prev9toever	Model	Uniform (1.077,1.203); normal (1.139,5.548)

	Model (lower 95% CI)	Uniform (1.077,1.0771)
	Model (upper 95% CI)	Uniform (1.203,1.2031)
Parity 5		
Lame_prev1to4	Model	Uniform (2.905,3.084); normal (2.994,4.700)
	Model (lower 95% CI)	Uniform (2.905,2.9051)
	Model (upper 95% CI)	Uniform (3.084,3.0841)
Lame_prev5to8	Model	Uniform (2.149,2.295); normal (2.221,5.204)
	Model (lower 95% CI)	Uniform (2.149,2.1491)
	Model (upper 95% CI)	Uniform (2.295,2.2951)
Lame_prev9toever	Model	Uniform (1.077,1.206); normal (1.140,5.517)
	Model (lower 95% CI)	Uniform (1.077,1.0771)
	Model (upper 95% CI)	Uniform (1.206,1.2061)

¹CI = credible interval

Table 6.3 Herd scenarios explored for adopting different culling rates.

Herd size	Culling rate	Parity 1 No. cows	Parity 1 removal rate	Parity 2 No. cows	Parity 2 removal rate	Parity 3 No. cows	Parity 3 removal rate	Parity 4 No. cows	Parity 5 + No. cows	% herd $\leq 3^{\text{rd}}$ lactation	% herd $\geq 4^{\text{th}}$ lactation
100	20	20	15	17	20	13	10	12	38	50	50
100	25	25	15	21	20	17	10	15	21	63	37
100	30	30	15	26	20	22	10	19	3	77	23
100	35	35	15	30	20	26	10	9		91	9
100	40	40	15	34	20	26				100	

No. = Number

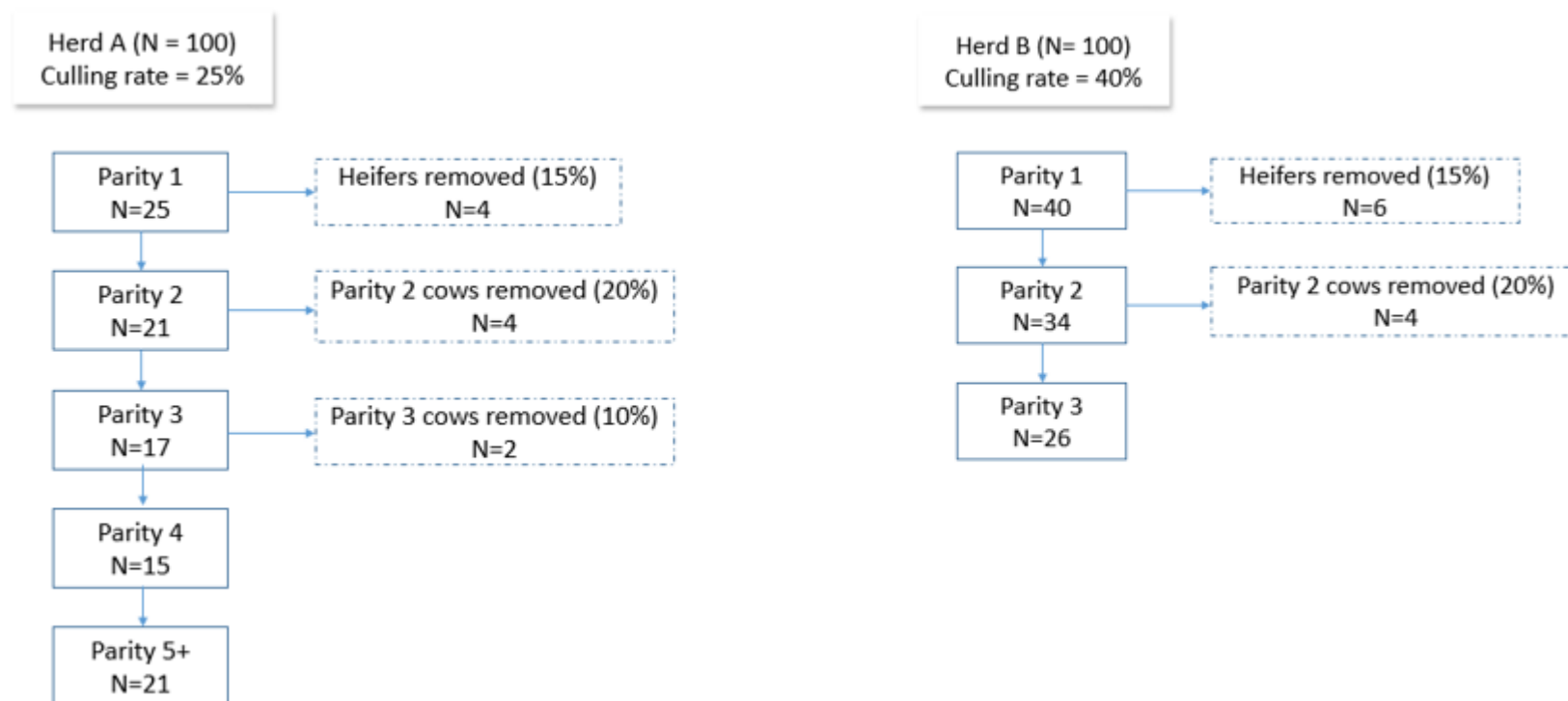


Figure 6.4 Flow chart showing example herds with culling rate of 25% and 40% to illustrate the culling rate scenarios described in Table 6.3. N = number of animals.

6.3 Results

6.3.1 Descriptive statistics

For the culling rate scenarios investigated the proportion of the herd in lactation ≤ 3 were 50, 63, 77, 90.5 and 100% for culling rates of 20, 25, 30, 35 and 40%, respectively reflecting a change in the age profile of the simulated herds (Table 6.3).

6.3.2 Model checking

The observed lameness prevalence in each of the 4 weeks of month 4 (as data was structured at the cow-week level) was between 19 and 22% (Table 6.4). The mean (95% credible interval) predicted prevalence from the simulation model when the culling rate was 25% (target for the herd from which observed data was obtained) was 17.2% (10 to 25%) and 17.6% (9 to 28%), when previous lameness risk distribution was uniform or normal, respectively and month 1 baseline risk was the mean estimate from the model with a uniform distribution (Table 6.5). Although the simulation using these distributions was under-predicting month 4 prevalence compared with the observed data the observed data did fall within the 95% credible intervals. In addition, it is expected that where prevalence was calculated from weekly locomotion scores this will be an overestimate compared with monthly scoring where some lame scores will be missed; therefore prevalence calculated from the observed data was expected to be higher than the simulated monthly prevalence. Overall, it was considered that the simulation model could be used to accurately predict prevalence and explore different herd scenarios.

Table 6.4 Prevalence of lameness events (locomotion score ≥ 3) for each week of the 4th month in lactation in 724 cows.

Week within month 4	Number of observations	Number of lameness events (LS ≥ 3)	Prevalence
1	1463	281	0.19
2	1456	300	0.21
3	1452	319	0.22
4	1441	284	0.20

6.3.3 Simulation results

The estimates of prevalence for the different culling rate scenarios and input parameter distributions are summarised in Table 6.5.

Culling rates and baseline month 1 risk

Values for month 1 baseline risk were lower when estimated from the logistic regression model compared with those calculated from the observed raw data. Where month 1 baseline risk was based on a narrow uniform distribution around the mean estimate from the model, predicted lameness prevalence in month 4 ranged from 14.6% with a 40% culling rate to 18.2% with a 20% culling rate compared with 36.2% with a 40% culling rate to 60.8% with a 20% culling rate when a narrow distribution around the observed risk was used. Different culling rates had a larger impact on lameness prevalence in simulated herds with a higher baseline lameness risk; for example, a change in culling rate from 40% to 20% resulted in lameness prevalence changing from 57.5% to 39.0% i.e. a 32.1% reduction when the higher baseline risk from observed data was used (with the uniform previous lameness distribution), whereas when the lower baseline lameness risk from model mean estimates was used in the equivalent scenario this resulted in lameness prevalence changing from 17.8% to 15.7% i.e. a 11.8% reduction. When a uniform distribution between the model mean estimate (i.e. lower baseline month 1 risk) and the calculated value from the observed data (i.e. higher baseline month 1 risk) was used mean lameness prevalence ranged from 25.0 to 41.07 (depending on the previous lameness distribution). When a uniform distribution for previous lameness was used, lameness prevalence changed from 38.2% to 27.1% with a culling rate of 40% versus 20% i.e. a 29.2% lower lameness prevalence.

Culling rates and previous lameness relative risk

The narrow uniform distributions using the lower and upper 95% credible intervals of estimated previous lameness RR gave lower and higher predicted mean lameness prevalence, respectively. As with baseline month 1 risk, where previous lameness RR values were higher, culling rates had a larger impact on lameness prevalence but this was only true where the baseline risk was lower (mean estimate from the model and uniform distribution between model model estimate and observed data); for example, when using the narrow uniform distribution around the upper 95% CI estimate (higher RR for previous lameness) the herd lameness prevalence changed

from 19.4% to 16.8% with a change in culling rate from 40% to 20% i.e. a 13.0% reduction in prevalence, whereas when using the lower 95% CI estimate (lower RR for previous lameness) herd lameness prevalence changed from 16.1% to 14.6% i.e. a 9.2% reduction in prevalence (with baseline month 1 risk as narrow uniform distribution around the mean estimate).

The mean lameness prevalence estimates were similar for both uniform and normal distributions of the 95% credible intervals of estimated previous lameness RR, however the 95% credible intervals of lameness prevalence estimates were larger with a normal distribution.

Table 6.5 Summary of predicted mean and 95% credible intervals for lameness prevalence for different herd scenarios including; a range of input parameter distributions for baseline month 1 risk and previous lameness relative risk (RR) as well as different culling rates.

Previous lameness RR distribution (model estimates) ¹	Culling rate	Baseline risk month 1 distributions								
		Narrow uniform (model mean estimate)			Narrow uniform (observed data)			Uniform (model mean, observed data)		
		<i>Parity 2: (0.0800,0.0801)</i> <i>Parity 3: (0.1050,0.1051)</i> <i>Parity 4: (0.1280,0.1281)</i> <i>Parity 5+: (0.1190,0.1191)</i>			<i>Parity 2: (0.1980,0.7981)</i> <i>Parity 3: (0.2160,0.2161)</i> <i>Parity 4: (0.3380,0.3381)</i> <i>Parity 5+: (0.2780,0.2781)</i>			<i>Parity 2: (0.080,0.198)</i> <i>Parity 3: (0.105,0.216)</i> <i>Parity 4: (0.128,0.338)</i> <i>Parity 5+: (0.119,0.278)</i>		
		Mean	Lower 95% CI	Upper 95% CI	Mean	Lower 95% CI	Upper 95% CI	Mean	Lower 95% CI	Upper 95% CI
Uniform (95% CI)	20	17.79	11.00	26.00	57.45	49.00	65.00	38.19	29.00	47.00
	25	17.22	10.00	25.00	52.53	44.00	61.00	35.48	27.00	45.00
	30	16.85	10.00	25.00	48.86	41.00	58.00	33.14	25.00	42.00
	40	15.69	9.00	23.00	39.02	30.00	47.00	27.05	19.00	35.00
Normal (95% CI)	20	18.17	9.00	30.00	53.00	31.00	70.00	36.49	20.00	52.00
	25	17.58	9.00	28.00	48.60	32.00	64.00	33.88	20.00	48.00
	30	17.34	9.00	27.00	45.80	30.00	61.00	32.11	19.00	47.00
	40	15.94	8.00	24.00	37.11	20.00	53.00	26.80	14.00	40.00
Narrow uniform (Lower 95% CI)	20	16.14	9.00	23.00	53.83	45.00	62.00	35.12	27.00	44.00
	25	15.72	9.00	23.00	49.46	41.00	58.00	32.77	25.00	42.00
	30	15.41	9.00	22.00	46.02	38.00	55.00	30.64	23.00	40.00
	40	15.06	8.00	22.00	36.17	28.00	44.00	24.97	17.00	33.00
Narrow uniform (Upper 95% CI)	20	19.43	12.00	27.00	60.8	52.00	69.00	41.07	32.00	50.00
	25	18.71	11.00	28.00	55.5	47.00	64.00	38.11	29.00	48.00
	30	18.26	11.00	26.00	51.52	44.00	60.00	35.52	27.00	45.00
	40	16.83	10.00	25.00	41.63	33.00	50.00	29.06	21.00	38.00

¹These distributions are shown in Table 6.2. CI = credible interval

6.4 Discussion

To date no studies have investigated the effect of herds adopting different culling rates on lameness prevalence. In this chapter, development of a simulation model has shown that across a range of herd scenarios lameness prevalence will vary with culling rates. The largest effect of different culling rates was seen when there was a higher background lameness risk in the herd i.e. herds which have a higher lameness risk at the beginning of each lactation.

6.4.1 Use of simulation models

Stochastic modelling has a long history of use in biology (Galton and Watson, 1875, Goel and Richter-Dyn, 1974), however it is only recently that these models have been used to investigate dairy herds by exploring for example; farm efficiencies (Kumbhakar et al., 1991), control strategies and/or economic consequences for infectious diseases (Sørensen et al., 1995, Kudahl et al., 2007), lameness (Ettema et al., 2010) and mastitis (Down et al., 2013). Stochastic models allow uncertainty to be incorporated into a model when assessing the impact that a factor has on the output. In this chapter the factor being assessed was culling rate, and the uncertainty around the baseline month 1 risk, previous lameness risk and changes in risk over time were introduced through their distributions. Hudson et al. (2015) highlighted that assumptions over selection of input parameter distribution is an aspect of stochastic models that can be criticised. The purpose of developing the stochastic model in the current chapter was to simulate hypothetical herds to explore the effect of different culling rates in a wide range of herd scenarios; ‘high’ and ‘low’ risk scenarios were therefore investigated using the upper and lower 95% credible interval estimates respectively as well as different distributions.

6.4.2 Culling rates and reasons for culling

A range of culling rates were investigated based on culling rates relevant to the UK, which are also relevant to the average culling rates reported in the literature worldwide (Bell et al., 2010b, De Vries et al., 2010, CDIC, 2016, Nor et al., 2014, Maher et al., 2008). The literature also reports a wide variation in culling rates between individual farms, which the ranges explored encompassed. For example, Nor et al. (2014) reported an overall average culling rate of 29.6% across 1903 Dutch dairy herds over the years 2007 to 2010 with a range of between 12.4 and 63%. More extreme

scenarios to those investigated in this chapter are also likely to also be relevant. It should also be noted that the scenarios investigated, whilst they may not be accurate to reality on farm (for example it is unlikely to have a herd with now cows in parity > 3), they do provide an good representation of a range of situations that are possible (in herds with very high culling rates the number of cows in parity > 3 will be rare). Culling rate is defined as the percentage of cows removed from a herd because of sale, slaughter, salvage or death (Fetrow et al., 2006). Difficulties with both recording reasons for culling as well as mis-interpretation of terminology have been highlighted as problems with research regarding culling rates (Nor et al., 2014, Fetrow et al., 2006). This is an aspect that was not explored using the simulation model, as there is little information regarding the impacts on the distribution of input parameters.

6.4.3 Effect of culling rates and input parameter distribution on lameness prevalence

The simulation model propagated risk through each month i.e. the probability of being lame in the previous month was taken into account when calculating the risk for the current month. This meant that if the risk in month 1 was higher (i.e. herds with a greater background risk of lameness at the start of each lactation) in one scenario compared to another, the herd lameness prevalence would be higher. Uniform distributions for month 1 risk were selected with ranges around i) the mean estimate from the logistic regression model and ii) the risk calculated from the observed data. Uniform distributions were chosen for this parameter so that it could take any value throughout the whole range with an equal probability, as it was not known where other herds would sit on this distribution. Investigating different ranges of baseline month 1 risk enabled the impact of culling rates on herds with different baseline risks for each lactation to be investigated. The risk ranges investigated were considered plausible as they were either mean estimates from a model which fitted the observed data well or were from the observed herd data itself. Changing the baseline risks had considerable impact on the mean predicted lameness prevalence. Culling rates had a larger impact on mean lameness prevalence when there was a higher month 1 lameness risk. The aim of the simulation was to explore the impact of culling rates on lameness prevalence in other herds. As it is not known what the background risks would be in other herds, a uniform distribution between the mean estimated risk and the observed risk was also explored. With this distribution, mean herd prevalence ranged from 25.0% to 41.1%

and the impact of changing the culling rate from 40% to 20% resulted in a relative reduction in lameness prevalence of 26.6% to 29.2%. These findings demonstrate that in herds with a higher risk of lameness at the start of each lactation, different culling rates will have an impact on the herd lameness prevalence; this may be important when benchmarking or comparing herds with different culling rates.

The risk associated with previous lameness was similarly propagated through the simulation model. It was therefore expected that as the relative risk for previous lameness increased this would increase the mean lameness prevalence considerably i.e. the increase would not be linear. This was consistent with the simulation results. The distributions around the lower and upper 95% credible interval estimates allowed a lower and higher previous lameness RR distribution to be investigated. As with the higher and lower risk for baseline month 1, culling rates were found to have a greater impact on mean lameness prevalence when the previous lameness RR was higher, although this was only true for the scenarios with a lower baseline month 1 risk. A uniform or normal distribution based on the 95% credible intervals from the logistic regression model were investigated to explore how changes in culling rate may impact on lameness prevalence in wider dairy herds. As would be expected the mean estimates for these distributions were similar, with wider 95% credible intervals when the normal distribution was used. However, as we have no information regarding where on the distribution other herds may sit, a uniform distribution would appear to be more appropriate in this regard.

When the baseline month 1 risk was a uniform distribution between the mean estimated risk and observed risk and the previous lameness RR was a uniform distribution between the 95% credible intervals, for a 25% culling rate mean lameness prevalence was 35.43%. This is consistent with reported averages for the UK where culling rates are 22 to 25% (Whitaker et al., 2000, Bell et al., 2010b) and average lameness prevalence was 36.8% in 2010 (Barker et al., 2010). This would suggest that results from the simulation model are applicable to wider herds and can provide accurate information regarding the impact of culling rates on lameness prevalence. In this herd scenario lameness prevalence reduced from 38.2% to 27.1% with a change in culling rate from 40% to 20%; a relative reduction in lameness prevalence of 29.2%.

Findings suggest that where herds have a higher culling rates for example US herds compared to UK or Irish herds, lameness prevalence may differ as a function of the longevity of the cows in the herd and the duration of time over which they will be

exposed to lameness risk. Targets for lower lameness prevalence should be more easily achievable in herds with higher culling rates. On the other hand, in herds with lower culling rates lameness prevalence is likely to be higher and increased focus on lameness prevention and control may be necessary to maintain the health, welfare and productivity of these herds.

Correlations between the parameters previous lameness risk and baseline month 1 risk for each parity were not explored, as there was no clear reason or rationale from data in earlier chapters that these parameters were correlated. There was therefore no basis to simulate these correlations.

6.5 Conclusion

Findings from this chapter show that by adopting different herd culling rates this will have an impact on lameness prevalence; and that this will differ depending on the risk of lameness at the start of each lactation. Herds adopting a culling rate of 40% versus 20% were found to have a relative reduction in lameness prevalence of 29.2%. It is also possible that different reasons for culling may impact on these figures and further work is needed to explore this aspect. In herds with lower culling rates where lameness prevalence is likely to be higher compared to herds with a higher culling rate, increased focus on lameness prevention and control may be necessary to maintain the productivity and welfare of the herd.

CHAPTER 7: General Discussion and Conclusions

7.1 Introduction

The aim of this thesis was to evaluate risk factors for lameness, in particular BCS and early life events. A review of the literature in Chapter 1 highlighted that the aetiopathogenesis of CHLD is still poorly understood. These were the primary lesions of interest for hypotheses explored within this thesis. Lameness in dairy cattle is a complex disease due to its multifactorial nature and in addition, the majority of lameness research is conducted under field conditions which can further add to these complexities. The increased availability of statistical software to fit multivariable models that account for correlated data means that appropriate statistical modelling can be a core component of lameness studies. Throughout this thesis, complex multilevel statistical modelling and simulation methods, combined with a rich longitudinal dataset, have aided in the identification of important key findings that advance our knowledge and understanding surrounding the aetiopathogenesis of lameness, and in particular CHDLs. This progress has been important for contributing to identifying alternative pathways and hypotheses, such as those associated with metabolic changes around the time of calving, from that of the laminitis theory which has previously dominated the lameness literature. Furthermore, the development of novel approaches to estimating PAF for lameness risk factors using simulation is an important step forward towards identifying risk factors that will have the greatest impact on controlling lameness in dairy herds. Findings from this thesis demonstrated that low BCS was associated with an increased risk of repeated lifetime lameness event, as well as first lifetime lameness events in second lactation or greater cows. The estimated PAF for 0.5 loss in BCS across one herd showed that this risk factor may contribute towards 6 – 11% of the total lameness events in the herd (depending on lameness severity). By comparison, all previous lameness events (regardless of when they occurred) were found to contribute towards up to 80% of total herd lameness and previous lameness events that occur at least 16 weeks previously contributed towards 9 – 21% of total lameness. In these herds repeated bouts of lameness made a very large contribution to the total number of lameness events. The impact of lameness in heifers, as the future of the herds was also explored, with novel findings identifying that mild CHDLs resulted in a reduced risk of future lameness and culling, indicating that some mild insult may confer some resilience through adaptive changes in the hoof if lesions occur at a time when there is the ability

to cope with this insult. Finally, the effect of herd age profile on lameness, through adopting different culling rates was explored. Culling rates were found to have a greater impact on lameness prevalence in herds with a higher background lameness risk in month one of each lactation; herds with lower culling rates may need to pay particular attention to preventing and controlling lameness in order to maintain the health, welfare and productivity of the herd.

Whilst demonstrating significant associations and identifying risk factors in observational and cross-sectional studies is an important aspect of investigating the pathogenesis of lameness and CHDLs, using intervention studies to substantiate current hypotheses as well as quantifying the impacts of risk factors at the population level is vital if progress in terms of managing and controlling disease is to be made. As lameness remains one of the greatest challenges currently facing the dairy industry a greater understanding of risk factors and how to manage them is paramount.

7.2 The use of statistical modelling to evaluate risk factors for lameness

The construction of statistical models was central to Chapters 3 to 6 in order to investigate and evaluate risk factors for lameness. Statistical models are used to explain the relationships between measured variables (or risk factors) and measured outcomes, whilst also ideally encompassing assumptions about the random processes that influence these outcomes. No model is perfect, but to evaluate how well a model investigating lameness describes the data and therefore how reliable reported findings are there were a number of areas to consider when conducting this research, as well as when appraising the current literature in Chapter 1.

The data obtained for this research was observational data recorded over an 8 year time period. Within the totality of peer-reviewed lameness literature, observational and cross-sectional studies are the most commonly reported study design. Whilst variables that are found to be significantly associated with a lameness outcome may be causally linked, results from these studies must be interpreted with caution with regards to causality. Dohoo et al. (2003) outlines a set of criteria for demonstrating causality including for example, time sequence, plausibility and experimental evidence. BCS, BW (and other explanatory variables) were investigated in Chapter 3 by lagging the data to explore temporal associations with lameness. Although it is not possible from this study to state that the relationship was causal, a number of criteria such as time

sequence and plausibility were fulfilled adding to the strength of evidence. Further work using intervention studies is required to demonstrate that minimising the loss of BCS does indeed reduce the risk of lameness and to rule out other possible causal pathways. Causality is also relevant to the work of Chapter 6 where the contribution that the risk factors BCS and previous lameness made towards total lameness events within a herd was investigated. Estimating PAF is based on the relationship between the risk factor and the outcome being causal; whilst further evidence is required to substantiate causality for the risk factors investigated, the evidence available suggests that this assumption is entirely reasonable. Furthermore, the development of simulation methods to estimate PAF for lameness risk factors using longitudinal data was also an important output from this chapter that can be applied to other risk factors. This could potentially help to identify interventions which could have the greatest impact on reducing lameness in dairy herds.

The rich longitudinal dataset used throughout this thesis and described in Chapter 2 enabled detailed investigation of risk factors, for example the fine thresholds of BCS and their association with mild or severe lameness in Chapter 3. Whilst sample size can be a common limitation for multivariable analysis, the volume of data available here meant that complex multivariable models could be constructed. This was important for identifying relationships between explanatory variables and lameness that were independent of other potentially confounding factors e.g. BCS independent of BW. Across the lameness literature reporting of sample sizes and sampling procedures is very commonly under-reported and was identified as a limitation particularly in the literature investigating laminitis (Chapter 1). Sample size calculations for correlated data, such as that commonly encountered when investigating lameness, are not straightforward and bespoke software is often required (for example, GLIMMPSE (Kreidler et al., 2013)) (Liu and Liang, 1997, Guo et al., 2013). In Chapter 3, subsets of the data were used to investigate the association between low BCS and first lifetime lameness events. In creating smaller subsets of data, sample sizes were much smaller and it is possible that study power was not sufficient to identify significant associations that may have been present. The results from these models should therefore be interpreted with this in consideration.

It was important to recognise the hierarchical nature of the data throughout this thesis. Data gathered from studies conducted under field conditions commonly have a hierarchical (clustered) structure, for example repeated measures of lameness (e.g.

lameness score or treatment events (level 1)) within a cow (level 2) within a farm (level 3). Similarities exist between the units at each level such that outcomes may be correlated. By recognising this structure as well as avoiding biased estimates (Rabash et al., 2009, Woodhouse and Goldstein, 1988), complex temporal associations between risk factors and lameness can be explored. Until more recently when software, such as MLWin (Rabash et al., 2009), became more widely available it has been a common limitation in the lameness literature that hierarchical nature of data has not been recognised. This is true particularly for the laminitis research and contributed towards the publication of a large body of work on one hypothesis that is supported by weak evidence. In Chapter 6, simulation methods for estimating the PAF for BCS and previous lameness enabled both the clustered nature of the data to be accounted for as well as the complex changes in risk factors over time. This approach, which has not previously been used to investigate PAF for lameness risk factors, allowed the importance of previous lameness as a risk factor to be highlighted which may be central to future work in understanding how to effectively control lameness in dairy herds.

Reporting of model assumptions and evaluation of model fit is another aspect of statistical modelling that is under-reported in the lameness literature. Chapter 5 was a good example of why this is important; although it was expected that mixed effects models would provide the best model fit for models estimating the relative risk for BCS and previous lameness events with the outcome lameness event (Models 1a and 1b), the process of assessing model fit demonstrated that removing random effects achieved the best model fit. Statistical models that have a poor fit to the data can lead to erroneous conclusions being drawn and therefore this process was repeated for all models constructed throughout this thesis.

Very relevant to the primary hypotheses being investigated in the current thesis were the temporal associations between risk factors and outcomes. This is also particularly pertinent issue in the lameness literature due to the cyclical nature of risk factors. Even if the direction of causality can be attributed, this can be complex; for example lying times impact on lameness, which in itself impacts on lying times, therefore complete historical data and modelling to control for effects is required in longitudinal studies in order to understand temporal associations. Chapter 2 explored these relationships by constructing models to investigate the association between BCS and first lifetime lameness events. This enabled the confounding effect of a previous lameness event to be avoided. Sufficient historical data is rarely available in lameness

studies to allow this analysis which is a limitation within the lameness literature. Future research should also consider this aspect at the study design stage.

Within each of Chapters 3 to 6 the generalisability of study findings has been discussed in relation to the data having been obtained from one UK dairy herd. Extrapolation of results from one study to a larger population is an important aspect of interpreting study findings and relies on detailed descriptions being provided such that the reader can make inferences about how to extrapolate findings to wider populations (Polit and Beck, 2010). As research investigating CHDLs is still relatively limited (Potterton et al., 2012) and commonly carried out on data from a relatively small number of farms and/or animals this is particularly relevant in the lameness field. In Chapter 5, estimates of PAF were reported for two separate herds, which allowed a comparison between herds with different environments and therefore may give an indication of how PAF may vary between herds in the wider population. The associations identified in Chapter 3 and 4, whilst unlikely to be quantitatively generalisable, are likely to be relevant qualitatively to other herds with similar management systems.

Chapter 1 (section 1.5.1) reviews the literature and evidence surrounding the laminitis theory. Use of the terminology ‘subclinical laminitis’ to describe the presence and diagnosis of CHDLs has a long history stretching back to the 1970’s. With that description comes an implied nutritional aetiology based on the occurrence of rumen acidosis. However, it has been largely due to limitations in study design and statistical modelling that much of the original laminitis research contributed to a lack of strong, but highly prevalent evidence in the literature; consequently the volume of potentially weak findings became the received norm of understanding in the field. The literature investigating the aetiopathogenesis of CHDLs is limited and especially when the statistical methods and reporting are critically appraised. This current thesis therefore adds to the research that has utilised appropriate statistical modelling to investigate the aetiopathogenesis of lameness and CHDLs and is therefore important in helping to move the lameness field forward.

7.3 The role of body condition score as a risk factor for lameness

The primary aim of Chapter 3 was to investigate the associations between fine thresholds of BCS (including BCS loss post-calving) and mild and severe lameness. Previous work had identified significant associations between low BCS and lameness,

however detailed analyses including how these associations differ for heifers and older cows had not been conducted. With a reduction in BCS, it is hypothesised that the digital cushion, a structure sitting beneath the distal phalanx that contains depots of adipose tissue and functions to dissipate concussive forces, becomes thinner (Bicalho et al., 2009). An understanding of the temporal associations between BCS, milk yield, digital cushion thickness and CHDLs is important as lameness itself can also directly impact on these risk factors (potentially through alternations to feeding behaviour ((Thorup et al., 2016, Norring et al., 2014)). It was therefore important that the analyses conducted in Chapter 3 explored the temporal associations between BCS and first lifetime lameness events as well as repeated lameness events. Positive associations were identified for first lifetime events in cows in their second lactation and greater as well as for repeated first lifetime lameness events; demonstrating that cows with low BCS in the previous 3-weeks were more likely to become lame. This association was correlated up to 16 weeks previously. Although these findings are important in providing further evidence to demonstrate that BCS has a role in the aetiopathogenesis of lameness and CHDLs, this does not quantify the impact that this risk factor has on the total herd lameness, which was the aim of Chapter 5. Compared to previous lameness events as a risk factor, a loss in BCS contributed towards a smaller proportion (4 to 11%) of the total number of lameness events. Although it is possible that in herds with less repeat lameness cases BCS could relatively be a more important risk factor. These findings highlight the relative importance of BCS as a risk factor for CHDLs; prioritising resources towards risk factors that make the greatest contribution towards the total herd lameness may have greater impact on controlling lameness. BCS should not be ignored as a risk factor and minimising loss post-calving as well as maintaining a target of 2.5 or greater should remain as recommendations.

Until recently the hypothesised mechanism by which BCS impacts on CHDLs were thought to be primarily due to loss in digital cushion thickness. However, Newsome et al. (2017b) reported findings from a prospective cohort study of 158 cows showing that whilst thin sole soft tissue thickness (SST; corium and digital cushion) predisposed cows to CHDL later in lactation, thinning of SST did not influence the likelihood of lesion occurrence and an additional effect of loss of back fat thickness was found. Other factors may contribute to a thin digital cushion including changes to the suspensory apparatus associated with calving or laminar pathology as well as previous lameness (Newsome et al., 2016, Knott et al., 2007, Tarlton et al., 2002). Further work

is required to understand whether controlling body condition loss does indeed reduce lameness caused by CHDL as well as the underlying biological mechanism. Another hypothesis that was discussed by Newsome et al. (2017b) was the role of insulin resistance, as a common causal pathway to BCS loss and development of CHDL. In order to meet the demands associated with the onset of lactation, homeorhetic adaptations occur. Findings suggest that inflammatory signals may contribute to homeorhetic adaptations to lactation, including a decrease in insulin sensitivity (Farney et al., 2013). In addition there are many factors that can contribute to inflammation post-partum including infection (for example mastitis or uterine infections), heat stress and lipopolysachharide (LPS) absorption due to changes in diet and/or changes in feeding behaviour (Bradford et al., 2015). An increased insulin resistance in transition cows may exacerbate lipid mobilization and impair metabolic health (causing suppression of DMI) (Zachut et al., 2013) and can therefore contribute to loss in BCS. High producing dairy cows will be genetically predisposed to be more insulin resistant (Chagas et al., 2009) which could also explain the association between high milk production and the increased risk of lameness. Further works is required to understand whether metabolic (or subacute) inflammation does have a direct role in the development of CHDLs and by what mechanisms.

7.4 Lameness history and the role of previous lameness as a risk factor

A positive association between CHDLs and future lameness risk has been demonstrated and suggests that underlying pathology, or other unidentified risk, is carried over from one case to the next, with the consequence of increasing the likelihood of future lameness events (Randall et al., 2016, Hirst et al., 2002b). Findings from Chapter 4 showed that severe CHDLs in heifers post-calving were associated with an increased risk of lameness across all future lactations for the duration of time cows spent within the herd. This demonstrated not only the potential lifetime impacts of hoof lesions in heifers, but also that claw health in heifers will have long-term effects. It has been demonstrated that the development of new bone on the distal phalanx is positively associated with CHDL during life, supporting the hypothesis that long term anatomical changes may occur in response to CHDL and contribute to future lameness (Newsome et al., 2016).

Chapter 5 explored the role of previous lameness events by estimating PAF and therefore quantifying the contribution that this risk factor makes towards the total

number of lameness events in a herd. Although only 2 herds were investigated, the PAF for previous lameness events was found to be similar between the herds, despite differences in lameness identification (weekly locomotion score versus treatment of clinical cases), environment and management. Whilst the external validity of this study may be limited due to the small number of herd investigated, these results give some indication that the main findings may be generalisable more widely in a qualitative way. In these herds repeated bouts of lameness made a very large contribution to the total number of lameness events. The reasons for and methods of preventing repeated lameness events requires further research, however it appears plausible that an escalation of lameness events may occur following the first lifetime event that then accumulates over a lifetime. It is also possible that there are other environmental and/or animal-based factors which influence whether a cow goes on to have repeat lameness events after the first lifetime lameness event. Further work is needed to understand how previous lameness history contributes to total herd lameness and what interventions will have the greatest impact in controlling herd level lameness.

Controlling lameness in heifers, as the future of the dairy herd, is potentially very important as highlighted in Chapter 5 and reported elsewhere. Findings from Chapter 3 suggest it is possible that some degree of mild insult may be beneficial to long-term claw health. Bergsten et al. (2015) investigated the effect of flooring surface during the rearing period and post-calving on claw health in heifers and identified a similar association between mild lesions in heifers that had been reared on concrete having the lowest prevalence of CHDL when they were housed on straw post-calving. It appears that management of heifers can impact on future claw health that lasts a lifetime. Further work is required to investigate the optimum housing and management conditions to reduce lameness in heifers as well as throughout their lifetime; this may be different for different systems (e.g. grazed versus continuously housed herds).

As the exposure to previous lameness increases with the duration of time a cow spends in the herd, Chapter 6 aimed to investigate the effect of previous lameness in herds with different culling rates to identify the impact on herd lameness prevalence. By altering culling rates, the age profile of herds is manipulated and therefore the time over which cows will be exposed to previous lameness risk changes. This study demonstrated that lameness prevalence will differ between herds with different culling rates and this has the greatest impact on herds where there is a higher background lameness risk. Herds with lower culling rates are likely to have higher lameness

prevalence due to the higher number of older cows and therefore managing lameness in these herds becomes even more important to maintain health, welfare and productivity.

7.5 Future research

A number of key areas for future research have been identified throughout this discussion. Currently, a major lack of intervention studies (RCTs) persists throughout the lameness literature and future work must address this in order to move the current evidence forward, which is primarily based on observational studies. Studies which recognise the cyclical nature of many of the risk factors for lameness is also key for consideration in future research; the importance of previous lameness as a risk factor highlights this in particular. Estimating PAF is another important aspect that should be explored more often in lameness research in order to identify which risk factors have most impact on lameness, thereby informing where resources to reduce these risk factors should be allocated and prioritised (both at the farm level and at a wider industry level). Findings from the current thesis suggest that previous lameness has an overwhelming impact on future lameness; supporting the idea that once a cow becomes lame this will escalate over her lifetime or that there is an interaction with environmental/animal-based factors that predisposes some cows to repeat lameness. Understanding why repeat cases occur and how to prevent this could have considerable impact on lameness level in dairy herds and future research should aim to investigate this. Controlling lameness in heifers including management during the rearing period may be one aspect of this, whilst environmental and other animal-based factors could also be important.

7.6 Conclusions

This thesis builds on the existing evidence identifying BCS as a risk factor for lameness by demonstrating that low BCS is associated with an increased risk of lameness in first lifetime lameness events in cows in their second lactation and greater. In addition, a novel approach to estimating PAF for lameness risk factors, BCS and previous lameness, using simulation demonstrated the importance of previous lameness as a risk factor. This has highlighted where further work is urgently needed for understanding which aspects are important for lameness history, for example, preventing the occurrence of repeat cases or even first cases. Heifer management and the rearing period may be important in terms of where this research should be focussed

towards preventing first lameness events. The research conducted throughout this thesis used a rich longitudinal dataset that enabled detailed investigation and evaluation of risk factors for lameness in dairy cows, such that findings have built on existing knowledge surrounding the aetiopathogenesis of lameness, and in particular CHDLs.

Reducing lameness in dairy herds is important to the dairy industry for a number of reasons including the negative impacts of the disease on economics as well as the health and welfare of dairy cows. The dairy industry is under increasing pressure globally to become more sustainable from environmental, economic and social perspectives, and delivering this in the face of fluctuations in milk prices and farm incomes. Lameness is a disease of high producing dairy cows and this further highlights the need to control this important and prevalent disease. Identifying the risk factors that will have most influence on reducing lameness in dairy herds is therefore important to aid in improving the health, welfare and sustainability of dairy herds worldwide.

References

- AHDB DAIRY. 2017a. *AHDB Dairy Healthy Feet Programme* [Online]. Available: <https://dairy.ahdb.org.uk/technical-services/healthy-feet-programme/> [Accessed 31.03.2017 2017].
- AHDB DAIRY. 2017b. *Market Information; Average Milk Yield* [Online]. Available: <https://dairy.ahdb.org.uk/market-information/farming-data/milk-yield/average-milk-yield/> [Accessed 10.04.2017 2017].
- ALAWNEH, J. I., LAVEN, R. A. & STEVENSON, M. A. 2011. The effect of lameness on the fertility of dairy cattle in a seasonally breeding pasture-based system. *Journal of Dairy Science*, 94, 5487-93.
- ALAWNEH, J. I., STEVENSON, M. A., WILLIAMSON, N. B., LOPEZ-VILLALOBOS, N. & OTLEY, T. 2014. The effects of liveweight loss and milk production on the risk of lameness in a seasonally calving, pasture fed dairy herd in New Zealand. *Preventive Veterinary Medicine*, 113, 72-79.
- ALSAAOD, M., SCHAEFER, A. L., BÜSCHER, W. & STEINER, A. 2015. The Role of Infrared Thermography as a Non-Invasive Tool for the Detection of Lameness in Cattle. *Sensors (Basel, Switzerland)*, 15, 14513-14525.
- AMORY, J. R., BARKER, Z. E., WRIGHT, J. L., MASON, S. A., BLOWEY, R. W. & GREEN, L. E. 2008. Associations between sole ulcer, white line disease and digital dermatitis and the milk yield of 1824 dairy cows on 30 dairy cow farms in England and Wales from February 2003–November 2004. *Preventive Veterinary Medicine*, 83, 381-391.
- ANDERSSON, L. & LIBERG, P. 1980. Blood serum and synovial fluid in bovine laminitis and arthritis, with particular reference to the protein composition. *Acta Veterinaria Scandinavica*, 21, 567-577.
- ARCHER, S., BELL, N. & HUXLEY, J. 2010a. Lameness in UK dairy cows: a review of the current status. *In Practice*, 32, 492-504.
- ARCHER, S. C., GREEN, M. J. & HUXLEY, J. N. 2010b. Association between milk yield and serial locomotion score assessments in UK dairy cows. *Journal of Dairy Science*, 93, 4045-53.
- ARCHER, S. C., MC COY, F., WAPENAAR, W. & GREEN, M. J. 2013. Association between somatic cell count after first parturition and cumulative milk yield in dairy cows. *Veterinary Record*, 173, 316.
- ASSUREWEL. 2017. Available: <http://www.assurewel.org/> [Accessed last accessed 26.5.2017].
- BACH, A. 2011. Associations between several aspects of heifer development and dairy cow survivability to second lactation. *J. Dairy Sci.*, 94, 1052-1057.
- BARKEMA, H. W., WESTRIK, J. D., KEULEN, K. A. S. V., SCHUKKEN, Y. H. & 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, Z. E., AMORY, J. R., WRIGHT, J. L., BLOWEY, R. W. & GREEN, L. E. 2007. Management factors associated with impaired locomotion in dairy cows in England and Wales. *Journal of Dairy Science*, 90, 3270-7.
- BARKER, Z. E., AMORY, J. R., WRIGHT, J. L., MASON, S. A., BLOWEY, R. W. & GREEN, L. E. 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, 1971-8.
- BARKER, Z. E., LEACH, K. A., WHAY, H. R., BELL, N. J. & MAIN, D. C. J. 2010. Assessment of lameness prevalence and associated risk factors in dairy herds in England and Wales. *Journal of Dairy Science*, 93, 932-941.
- BAZELEY, K. & PINSENT, P. J. N. 1984. Preliminary observations on a series of outbreaks of acute laminitis in dairy cattle. *Veterinary Record*, 115, 619 - 622.

- BEAUDEAU, F., HENKEN, A., FOURICHON, C., FRANKENA, K. & SEEGER, H. 1993. Associations between health disorders and culling of dairy cows: a review. *Livestock Production Science*, 35, 213-236.
- BELL, M. J., WALL, E., RUSSELL, G., ROBERTS, D. J. & SIMM, G. 2010a. Risk factors for culling in Holstein-Friesian dairy cows. *Vet Rec*, 167, 238-40.
- BELL, M. J., WALL, E., RUSSELL, G., ROBERTS, D. J. & SIMM, G. 2010b. Risk factors for culling in Holstein-Friesian dairy cows. *Veterinary Record*, 167, 238-240.
- BELL, N. & HUXLEY, J. 2009. *Locomotion, lameness and mobility in dairy cows*, London, BMJ Publishing Group.
- BELL, N. J., BELL, M. J., KNOWLES, T. G., WHAY, H. R., MAIN, D. J. & WEBSTER, A. J. F. 2009. The development, implementation and testing of a lameness control programme based on HACCP principles and designed for heifers on dairy farms. *Veterinary Journal*, 180, 178-188.
- BENICHO, J. 2001. A review of adjusted estimators of attributable risk. *Statistical Methods in Medical Research*, 10, 195-216.
- BERGSTEN, C. 1994. Haemorrhages of the sole horn of dairy cows as a retrospective indicator of laminitis: an epidemiological study. *Acta Vet Scand*, 35, 55-66.
- BERGSTEN, C. 2001. Effects of conformation and management system on hoof and leg diseases and lameness in dairy cows. *Veterinary Clinics of North America: Food Animal Practice*, 17, 1-23.
- BERGSTEN, C. 2003. Causes, risk factors, and prevention of laminitis and related claw lesions. *Acta Veterinaria Scandinavica, Supplementum*, 98, 157-166.
- BERGSTEN, C. & FRANK, B. 1996. Sole haemorrhages in tied primiparous cows as an indicator of periparturient laminitis: effects of diet, flooring and season. *Acta Veterinaria Scandinavica*, 37, 383-394.
- BERGSTEN, C., TELEZHENKO, E. & VENTORP, M. 2015. Influence of soft or hard floors before and after first calving on dairy heifer locomotion, claw and leg health. *Animals*, 5, 662-686.
- BERRY, S. L., READ, D. H., FAMULA, T. R., MONGINI, A. & DOPFER, D. 2012. Long-term observations on the dynamics of bovine digital dermatitis lesions on a California dairy after topical treatment with lincomycin HCl. (Special Issue: Bovine lameness.). *Veterinary Journal*, 193, 654-658.
- BICALHO, R. C., CHEONG, S. H., CRAMER, G. & GUARD, C. L. 2007a. Association between a visual and an automated locomotion score in lactating Holstein cows. *Journal of Dairy Science*, 90, 3294-300.
- BICALHO, R. C., MACHADO, V. S. & CAIXETA, L. S. 2009. Lameness in dairy cattle: A debilitating disease or a disease of debilitated cattle? A cross-sectional study of lameness prevalence and thickness of the digital cushion. *Journal of Dairy Science*, 92, 3175-84.
- BICALHO, R. C. & OIKONOMOU, G. 2013. Control and prevention of lameness associated with claw lesions in dairy cows. *Livestock Science*, 156, 96 - 105.
- BICALHO, R. C., VOKEY, F., ERB, H. N. & GUARD, C. L. 2007b. Visual Locomotion Scoring in the First Seventy Days in Milk: Impact on Pregnancy and Survival. *Journal of Dairy Science*, 90, 4586-4591.
- BICALHO, R. C., WARNICK, L. D. & GUARD, C. L. 2008. Strategies to analyze milk losses caused by diseases with potential incidence throughout the lactation: a lameness example. *Journal of Dairy Science*, 91, 2653-61.
- BOOTH, C. J., WARNICK, L. D., GROHN, Y. T., MAIZON, D. O., GUARD, C. L. & JANSSEN, D. 2004. Effect of lameness on culling in dairy cows. *J. Dairy Sci.*, 87, 4115-4122.
- BRADFORD, B. J., YUAN, K., FARNEY, J. K., MAMEDOVA, L. K. & CARPENTER, A. J. 2015. Invited review: Inflammation during the transition to lactation: New adventures with an old flame. *J Dairy Sci*, 98, 6631-50.

- BRAVO-URETA, B. E. & RIEGER, L. 1991. Dairy farm efficiency measurement using stochastic frontiers and neoclassical duality. *American Journal of Agricultural Economics*, 73, 421-428.
- BRICKELL, J. S., BOURNE, N., MCGOWAN, M. M. & WATHES, D. C. 2009. Effect of growth and development during the rearing period on the subsequent fertility of nulliparous Holstein-Friesian heifers. *Theriogenology*, 72, 408-416.
- BRICKELL, J. S. & WATHES, D. C. 2011. A descriptive study of the survival of Holstein-Friesian heifers through to third calving on English dairy farms. *Journal of Dairy Science*, 94, 1831-1838.
- BROWNE, W. J. & DRAPER, D. 2006. A comparison of Bayesian and likelihood-based methods for fitting multilevel models. *Bayesian Analysis*, 1, 473 - 514.
- BRUIJNIS, M. R. N., HOGEVEEN, H. & STASSEN, E. N. 2010. Assessing economic consequences of foot disorders in dairy cattle using a dynamic stochastic simulation model. *Journal of Dairy Science*, 93, 2419-2432.
- BUROW, E., THOMSEN, P. T., ROUSING, T. & SORENSEN, J. T. 2014. Track way distance and cover as risk factors for lameness in Danish dairy cows. *Prev Vet Med*, 113, 625-8.
- CAPION, N., THAMSBORG, S. M. & ENEVOLDSEN, C. 2008. Conformation of hind legs and lameness in Danish Holstein heifers. *Journal of Dairy Science*, 91, 2089-2097.
- CAPION, N., THAMSBORG, S. M. & ENEVOLDSEN, C. 2009. Prevalence and severity of foot lesions in Danish Holstein heifers through first lactation. *Veterinary Journal*, 182, 50-58.
- CDIC. 2016. *Canadian Dairy Information Centre* [Online]. Available: <http://www.dairyinfo.gc.ca/index> [Accessed 17.5.2017].
- CHAGAS, L. M., LUCY, M. C., BACK, P. J., BLACHE, D., LEE, J. M., GORE, P. J., SHEAHAN, A. J. & ROCHE, J. R. 2009. Insulin resistance in divergent strains of Holstein-Friesian dairy cows offered fresh pasture and increasing amounts of concentrate in early lactation. *J Dairy Sci*, 92, 216-22.
- CHAPINAL, N., BARRIENTOS, A. K., KEYSERLINGK, M. A. G. V., GALO, E. & WEARY, D. M. 2013. Herd-level risk factors for lameness in freestall farms in the northeastern United States and California. *Journal of Dairy Science*, 96, 318-328.
- CHAPINAL, N., PASSILLE, A. M. D., PASTELL, M., HANNINEN, L., MUNKSGAARD, L. & RUSHEN, J. 2011. Measurement of acceleration while walking as an automated method for gait assessment in dairy cattle. *Journal of Dairy Science*, 94, 2895-2901.
- CHAPINAL, N., PASSILLE, A. M. D., WEARY, D. M., KEYSERLINGK, M. A. G. V. & RUSHEN, J. 2009. Using gait score, walking speed, and lying behavior to detect hoof lesions in dairy cows. *Journal of Dairy Science*, 92, 4365-4374.
- CHESTERTON, N. 1989. Environmental and behavioural factors affecting the prevalence of foot lameness in New Zealand dairy herds - a case-control study. *New Zealand Veterinary Journal*, 37, 135 - 142.
- CLARKSON, M. J., DOWNHAM, D. Y., FAULL, W. B., HUGHES, J. W., MANSON, F. J., MERRITT, J. B., MURRAY, R. D., RUSSELL, W. B., SUTHERST, J. E. & WARD, W. R. 1996. Incidence and prevalence of lameness in dairy cattle. *Veterinary Record*, 138, 563-7.
- COLLICK, D. W., WARD, W. R. & DOBSON, H. 1989. Associations between types of lameness and fertility. *Veterinary Record*, 125, 103-6.
- COOK, N. B. 2003. Prevalence of lameness among dairy cattle in Wisconsin as a function of housing type and stall surface. *Journal of American Veterinary Medical Association*, 223, 1324-8.
- COOK, N. B., HESS, J. P., FOY, M. R., BENNETT, T. B. & BROTZMAN, R. L. 2016. Management characteristics, lameness, and body injuries of dairy cattle housed in high-performance dairy herds in Wisconsin. *Journal of Dairy Science*, 99, 5879-91.
- COOK, N. B. & NORDLUND, K. V. 2009. The influence of the environment on dairy cow behavior, claw health and herd lameness dynamics. *Veterinary Journal*, 179, 360-369.

- COOK, N. B., NORDLUND, K. V. & OETZEL, G. R. 2004. Environmental influences on claw horn lesions associated with laminitis and subacute ruminal acidosis in dairy cows. *Journal of Dairy Science*, 87, E36 - E46.
- COOKE, J. S., CHENG, Z., BOURNE, N. E. & WATHES, D. C. 2013. Association between growth rates, age at first calving and subsequent fertility, milk production and survival in Holstein-Friesian heifers. *Open Journal of Animal Sciences*, 3, 1-12.
- CRAMER, G., LISSEMORE, K. D., GUARD, C. L., LESLIE, K. E. & KELTON, D. F. 2008. Herd- and cow-level prevalence of foot lesions in Ontario dairy cattle. *Journal of Dairy Science*, 91, 3888-3895.
- DANSCHER, A. M., ENEMARK, J. M., TELEZHENKO, E., CAPION, N., EKSTROM, C. T. & THOEFNER, M. B. 2009. Oligofructose overload induces lameness in cattle. *J Dairy Sci*, 92, 607-16.
- DANSCHER, A. M., TOELBOELL, T. H. & WATTLE, O. 2010. Biomechanics and histology of bovine claw suspensory tissue in early acute laminitis. *Journal of Dairy Science*, 93, 53-62.
- DE VRIES, A., OLSON, J. D. & PINEDO, P. J. 2010. Reproductive risk factors for culling and productive life in large dairy herds in the eastern United States between 2001 and 2006. *Journal of Dairy Science*, 93, 613-623.
- DEFRA 2003. Code of Recommendations for the Welfare of Livestock: Cattle. London: Defra Publications.
- DEMBELE, I., SPINKA, M., STEHULOVA, I., PANAMA, J. & FIRLA, P. 2006. Factors contributing to the incidence and prevalence of lameness on Czech dairy farms. *Czech Journal of Animal Science*, 51, 102-109.
- DIJKHUIZEN, A. A., STELWAGEN, J. & RENKEMA, J. A. 1986. A stochastic model for the simulation of management decisions in dairy herds, with special reference to production, reproduction, culling and income. *Preventive Veterinary Medicine*, 4, 273-289.
- DOHOO, I., MARTIN, W. & STRYHN, H. 2003. *Veterinary Epidemiologic Research*, Charlottetown, Canada, AVC Inc.
- DONOVAN, G. A., RISCO, C. A., TEMPLE, G. M., TRAN, T. Q. & VAN HORN, H. H. 2004. Influence of transition diets on occurrence of subclinical laminitis in Holstein dairy cows. *Journal of Dairy Science*, 87, 73-84.
- DOPFER, D., KOOPMANS, A., MEIJER, F. A., SZAKALL, I., SCHUKKEN, Y. H., KLEE, W., BOSMA, R. B., CORNELISSE, J. L., VAN ASTEN, A. J. A. M. & TER HUURNE, A. A. H. M. 1997. Histological and bacteriological evaluation of digital dermatitis in cattle, with special reference to spirochaetes and *Campylobacter faecalis*. *Veterinary Record*, 140, 620-623.
- DOWN, P. M., GREEN, M. J. & HUDSON, C. D. 2013. Rate of transmission: A major determinant of the cost of clinical mastitis. *Journal of Dairy Science*, 96, 6301-6314.
- EDDY, R. G. & SCOTT, C. P. 1980. Some observations on the incidence of lameness in dairy cattle in Somerset. *Veterinary Record*, 106, 140-144.
- ESPEJO, L. A. & ENDRES, M. I. 2007. Herd-level risk factors for lameness in high-producing Holstein cows housed in freestall barns. *Journal of Dairy Science*, 90, 306 - 314.
- ESSLEMONT, R. J. & KOSSAIBATI, M. A. 1996. Incidence of production diseases and other health problems in a group of dairy herds in England. *Veterinary Record*, 139, 486 - 490.
- ETTEMA, J., OSTERGAARD, S. & KRISTENSEN, A. R. 2010. Modelling the economic impact of three lameness causing diseases using herd and cow level evidence. *Preventive Veterinary Medicine*, 95, 64-73.
- ETTEMA, J. F. & SANTOS, J. E. P. 2004. Impact of age at calving on lactation, reproduction, health, and income in first-parity Holsteins on commercial farms. *J. Dairy Sci.*, 87, 2730-2742.
- FARNEY, J. F., MAMEDOVA, L. K., COETZEE, J. F., KUKANICH, B., SORDILLO, L. M., STOAKES, S. K., MINTON, J. E., HOLLIS, L. C. & BRADFORD, B. J. 2013. Anti-inflammatory salicylate

- treatment alters the metabolic adaptations to lactation in dairy cattle. *American Journal of Physiology - Regulatory, Integrative and Comparative Physiology*, 305, R110 - R117.
- FAULL, W. B., HUGHES, J. W., CLARKSON, M. J., DOWNHAM, D. Y., MANSON, F. J., MERRITT, J. B., MURRAY, R. D., RUSSELL, W. B., SUTHERST, J. E. & WARD, W. R. 1996. Epidemiology of lameness in dairy cattle: the influence of cubicles and indoor and outdoor walking surfaces. *Vet Rec*, 139, 130-6.
- FAWC 2005. Report on the Welfare Implications of Farm Assurance Schemes.
- FETROW, J., NORDLUND, K. V. & NORMAN, H. D. 2006. Culling: nomenclature, definitions and recommendations. *Journal of Dairy Science*, 89.
- FODITSCH, C., OIKONOMOU, G., MACHADO, V. S., BICALHO, M. L., GANDA, E. K., LIMA, S. F., ROSSI, R., RIBEIRO, B. L., KUSSLER, A. & BICALHO, R. C. 2016. Lameness prevalence and risk factors in large dairy farms in upstate New York. Model development for the prediction of claw horn disruption lesions. *PLoS One*, 11, e0146718.
- FRANCK, A., VERHEGGHE, B. & DE BELIE, N. 2008. The Effect of Concrete Floor Roughness on Bovine Claws Using Finite Element Analysis. *Journal of Dairy Science*, 91, 182-192.
- FREGONESI, J. A., VON KEYSERLINGK, M. A. & WEARY, D. M. 2009. Cow preference and usage of free stalls compared with an open pack area. *J Dairy Sci*, 92, 5497-502.
- GALEA, S., RIDDLE, M. & KAPLAN, G. A. 2010. Causal thinking and complex system approaches in epidemiology. *International Journal of Epidemiology*, 39, 97-106.
- GALINDO, F. & BROOM, D. M. 2000. The relationships between social behaviour of dairy cows and the occurrence of lameness in three herds. *Research in Veterinary Science*, 69, 75-9.
- GALINDO, F., BROOM, D. M. & JACKSON, P. G. 2000. A note on possible link between behaviour and the occurrence of lameness in dairy cows. *Appl. Anim. Behav. Sci.*, 67, 335-341.
- GALTON, F. & WATSON, H. W. 1875. On the probability of the extinction of families. *Journal of the Royal Anthropological Institute*, 4, 138 - 144.
- GARBARINO, E. J., HERNANDEZ, J., SHEARER, J. K., RISCO, C. & THATCHER, W. W. 2004. Effect of Lameness on Ovarian Activity in Postpartum Holstein Cows. *Journal of Dairy Science*, 87, 4123-4131.
- GARD, J. A., TAYLOR, D. R., WILHITE, D. R., RODNING, S. P., SCHNUELLE, M. L., BEYERS, R. J., EDMONSON, M. A., DEGRAVES, F. J. & VAN SANTEN, E. 2015. Effect of exercise and environmental terrain on development of the digital cushion and bony structures of the bovine foot. *American Journal of Veterinary Research*, 76, 246 - 252.
- GEARHART, M. A., CURTIS, C. R., ERB, H. N., SMITH, R. D., SNIFFEN, C. J., CHASE, L. E. & COOPER, M. D. 1990. Relationship of changes in condition score to cow health in holsteins. *Journal of Dairy Science*, 73, 3132-3140.
- GELMAN, A. 2000. Diagnostic checks for discrete data regression models using posterior predictive simulations. *Applied Statistics*, 49, 247-268.
- GELMAN, A., MENG, X. & STERN, H. 1996. Posterior predictive assessment of model fitness via realized discrepancies. *Statistica Sinica*, 6, 733-807.
- GELMAN, A. & SHIRLEY, K. 2011. *Handbook of Markov chain Monte Carlo*, Chapman and Hall.
- GITAU, T., MCDERMOTT, J. J. & MBIUKI, S. M. 1996. Prevalence, incidence, and risk factors for lameness in dairy cattle in small-scale farms in Kikuyu Division, Kenya. *Preventive Veterinary Medicine*, 28, 101-115.
- GOEL, N. S. & RICHTER-DYN, N. 1974. 1 - Introduction. *Stochastic Models in Biology*. Academic Press.
- GOLDSTEIN, H. 1995. *Multilevel Statistical Models*, London: Edward Arnold, New York: Halstead Press.
- GOLDSTEIN, H. 2003. *Multilevel Statistics Models*, London, UK, Arnold.

- GOMEZ, A., COOK, N. B., SOCHA, M. T. & DOPFER, D. 2015. First-lactation performance in cows affected by digital dermatitis during the rearing period. *Journal of Dairy Science*, 98, 4487-98.
- GREEN, L. E., HEDGES, V. J., SCHUKKEN, Y. H., BLOWEY, R. W. & PACKINGTON, A. J. 2002. The impact of clinical lameness on the milk yield of dairy cows. *Journal of Dairy Science*, 85, 2250-6.
- GREEN, L. E., HUXLEY, J. N., BANKS, C. & GREEN, M. J. 2014. Temporal associations between low body condition, lameness and milk yield in a UK dairy herd. *Preventive Veterinary Medicine*, 113, 63-71.
- GREEN, M. (ed.) 2012. *Dairy Herd Health*, Oxfordshire: CABI.
- GREEN, M. J., BURTON, P. R., GREEN, L. E., SCHUKKEN, Y. H., BRADLEY, A. J., PEELER, E. J. & MEDLEY, G. F. 2004. The use of Markov chain Monte Carlo for analysis of correlated binary data: patterns of somatic cells in milk and the risk of clinical mastitis in dairy cows. *Preventive Veterinary Medicine*, 64, 157-74.
- GREEN, M. J., MEDLEY, G. F. & BROWNE, W. J. 2009. Use of posterior predictive assessments to evaluate model fit in multilevel logistic regression. *Vet Res*, 40, 30.
- GREENOUGH, P. R. 1985. The sub clinical laminitis syndrome. *Bovine Practitioner*, 20, 144-149.
- GREENOUGH, P. R. & VERMUNT, J. J. 1991. Evaluation of subclinical laminitis in a dairy herd and observations on associated nutritional and management factors. *Veterinary Record*, 128, 11-17.
- GUARD, C. 2000. Investigating herds with lameness problems. *Proceedings of Hoof Health Conference, Duluth, Minnesota. Hoof Trimmers Association, Missoula, MT.*, 29-32.
- GUO, Y., LOGAN, H. L., GLUECK, D. H. & MULLER, K. E. 2013. Selecting a sample size for studies with repeated measures. *BMC Medical Research Methodology*, 13.
- HADLEY, G. L., WOLF, C. A. & HARSH, S. B. 2006. Dairy Cattle Culling Patterns, Explanations, and Implications. *Journal of Dairy Science*, 89, 2286-2296.
- HANLEY, J. A. 2001. A heuristic approach to the formulas for population attributable fraction. *Journal of Epidemiology and Community Health*, 55, 508-514.
- HASKELL, M. J., RENNIE, L. J., BOWELL, V. A., BELL, M. J. & LAWRENCE, A. B. 2006. Housing system, milk production, and zero-grazing effects on lameness and leg injury in dairy cows. *J Dairy Sci*, 89, 4259-66.
- HEDGES, J., BLOWEY, R. W., PACKINGTON, A. J., O'CALLAGHAN, C. J. & GREEN, L. E. 2001. A longitudinal field trial of the effect of biotin on lameness in dairy cows. *Journal of Dairy Science*, 84, 1969-1975.
- HERNANDEZ, J. A., GARBARINO, E. J., SHEARER, J. K., RISCO, C. A. & THATCHER, W. W. 2005. Comparison of the calving-to-conception interval in dairy cows with different degrees of lameness during the prebreeding postpartum period. *Journal of the American Veterinary Medical Association*, 227, 1284-91.
- HERRERO, M. & THORNTON, P. K. 2013. Livestock and global change: emerging issues for sustainable food systems. *Proc. Natl. Acad. Sci. USA*, 110, 20878-81.
- HINTERHOFER, C., FERGUSON, J. C., APPRICH, V., HAIDER, H. & STANEK, C. 2005. A finite element model of the bovine claw under static load for evaluation of different flooring conditions. *New Zealand Veterinary Journal*, 53, 165-170.
- HIRST, W. M., LE FEVRE, A. M., LOGUE, D. N., OFFER, J. E., CHAPLIN, S. J., MURRAY, R. D., WARD, W. R. & FRENCH, N. P. 2002a. A systematic compilation and classification of the literature on lameness in cattle. *Veterinary Journal*, 164, 7-19.
- HIRST, W. M., MURRAY, R. D., WARD, W. R. & FRENCH, N. P. 2002b. A mixed-effects time-to-event analysis of the relationship between first-lactation lameness and subsequent lameness in dairy cows in the UK. *Preventive Veterinary Medicine*, 54, 191-201.
- HOEDEMAEKER, M., PRANGE, D. & GUNDELACH, Y. 2009. Body condition change ante- and postpartum, health and reproductive performance in German Holstein cows. *Reproduction in Domestic Animals*, 44, 167-173.

- HOSMER, D. W. & LEMESHOW, S. 1989. *Applied Logistic Regression*, New York, Wiley.
- HUDSON, C. D., BRADLEY, A. J., BREEN, J. E. & GREEN, M. J. 2015. Dairy herd mastitis and reproduction: Using simulation to aid interpretation of results from discrete time survival analysis. *Veterinary Journal*, 204, 47-53.
- HUDSON, C. D., HUXLEY, J. N. & GREEN, M. J. 2014. Using simulation to interpret a discrete time survival model in a complex biological system: fertility and lameness in dairy cows. *PLoS One*, 9, e103426.
- HULTGREN, J. & BERGSTEN, C. 2001. Effects of a rubber-slatted flooring system on cleanliness and foot health in tied dairy cows. *Preventive Veterinary Medicine*, 52, 75-89.
- HUXLEY, J. N. 2012. Lameness in cattle: An ongoing concern. *The Veterinary Journal*, 193, 610-611.
- HUXLEY, J. N., BURKE, J., RODERICK, S., MAIN, D. C. J. & WHAY, H. R. 2004. Animal welfare assessment benchmarking as a tool for health and welfare planning in organic dairy herds. *Veterinary Record*, 155, 237-239.
- ICAR 2015. International Committee for Animal Recording (ICAR) Claw Health Atlas. Rome, Italy: ICAR.
- ITO, K., VON KEYSERLINGK, M. A., LEBLANC, S. J. & WEARY, D. M. 2010. Lying behavior as an indicator of lameness in dairy cows. *Journal of Dairy Science*, 93, 3553-60.
- JUAREZ, S. T., ROBINSON, P. H., DEPETERS, E. J. & PRICE, E. O. 2003. Impact of lameness on behavior and productivity of lactating Holstein cows. *Applied Animal Behaviour Science*, 83, 1-14.
- KATZ, L. M. & BAILEY, S. R. 2012. A review of recent advances and current hypotheses on the pathogenesis of acute laminitis. *Equine Vet J*, 44, 752-61.
- KENDALL, M. G. 1955. Rank Correlation Methods. New York: Hafner Publishing Co.
- KEYSERLINGK, M. A. G. V., BARRIENTOS, A., ITO, K., GALO, E. & WEARY, D. M. 2012. Benchmarking cow comfort on North American freestall dairies: lameness, leg injuries, lying time, facility design, and management for high-producing Holstein dairy cows. *Journal of Dairy Science*, 95, 7399-7408.
- KEYSERLINGK, M. A. G. V., RUSHEN, J., PASSILLE, A. M. D. & WEARY, D. M. 2009. The welfare of dairy cattle - key concepts and the role of science. *Journal of Dairy Science*, 92, 4101-4111.
- KNOTT, L., TARLTON, J. F., CRAFT, H. & WEBSTER, A. J. F. 2007. Effects of housing, parturition and diet change on the biochemistry and biomechanics of the support structures of the hoof of dairy heifers. *Veterinary Journal*, 174, 277-287.
- KOFLER, J. 1999. Clinical study of toe ulcer and necrosis of the apex of the distal phalanx in 53 cattle. *Veterinary Journal*, 157, 139-147.
- KREIDLER, S. M., MULLER, K., GRUNWALD, G. K., RINGHAM, B. M., COKER-DUKOWITZ, Z. T., SAKHADEO, U. R., BARON, A. E. & GLUECK, D. H. 2013. GLIMPSE: Online power computation for linear models with and without a baseline covariate. *Journal of Statistical Software*, 54.
- KUDAHL, A. B., ØSTERGAARD, S., SØRENSEN, J. T. & NIELSEN, S. S. 2007. A stochastic model simulating paratuberculosis in a dairy herd. *Preventive Veterinary Medicine*, 78, 97-117.
- KUMBHAKAR, S. C., GHOSH, S. & MCGUCKIN, J. T. 1991. A generalized production frontier approach for estimating determinants of inefficiency in U.S. dairy farms. *Journal of Business & Economic Statistics*, 9, 279-286.
- LATREMOLIERE, A. & WOOLF, C. J. 2009. Central sensitization: a generator of pain hypersensitivity by central neural plasticity. *Journal of Pain*, 10, 895-926.
- LAVEN, R. 2006. Nutrition and lameness in dairy cattle - a different perspective? *UK Vet: Livestock*, 11, 1 - 3.

- LAVEN, R. A., LAWRENCE, K. E., WESTON, J. F., DOWSON, K. R. & STAFFORD, K. J. 2008. Assessment of the duration of the pain response associated with lameness in dairy cows and the influence of treatment. *New Zealand Veterinary Journal*, 56, 210 - 217.
- LEACH, K. A., OFFER, J. E., SVOBODA, I. & LOGUE, D. N. 2005. Effects of type of forage fed to dairy heifers: associations between claw characteristics, clinical lameness, environment and behaviour. *Veterinary Journal*, 169, 427-36.
- LIM, P. Y., HUXLEY, J. N., WILLSHIRE, J. A., GREEN, M. J., OTHMAN, A. R. & KALER, J. 2014. Unravelling the association between lameness and body condition score in dairy cattle using a multistate modelling approach. *Preventive Veterinary Medicine*, 118, 370-377.
- LISCHER, C. J., KOLLER, U., GEYER, H., MULLING, C., SCHULZE, J. & OSSENT, P. 2002a. Effect of therapeutic dietary biotin on the healing of uncomplicated sole ulcers in dairy cattle - a double blinded controlled study. *Veterinary Journal*, 163, 51-60.
- LISCHER, C. J., KOLLER, U., GEYER, H., OSSENT, P. & AUER, J. A. 2001. Relation between radiologic findings at the third phalanx and the healing process of sole ulcers in dairy cows. *Tierärztliche Praxis*, 29, 122-127.
- LISCHER, C. J., OSSENT, P., RABER, M. & GEYER, H. 2002b. Suspensory structures and supporting tissues of the third phalanx of cows and their relevance to the development of typical sole ulcers (Rusterholz ulcers). *Veterinary Record*, 151, 694-698.
- LIU, G. & LIANG, K.-Y. 1997. Sample size calculations for studies with correlated observations. *Biometrics*, 53, 937 - 947.
- LIVESEY, C. T. & FLEMING, F. L. 1984. Nutritional influences on laminitis, sole ulcer and bruised sole in Friesian cows. *Veterinary Record*, 114, 510-512.
- LIVESEY, C. T., HARRINGTON, T., JOHNSTON, A. M., MAY, S. A. & METCALF, J. A. 1998. The effect of diet and housing on the development of sole haemorrhages, white line haemorrhages and heel erosions in Holstein heifers. *Animal Science*, 67, 9-16.
- LIVESEY, C. T., METCALF, J. A. & LAVEN, R. A. 2003. Effect of concentrate composition and cubicle bedding on the development of hoof haemorrhages in Holstein heifers after calving. *Veterinary Record*, 152, 735-739.
- LOGUE, D. N., OFFER, J. E. & MCGOVERN, R. D. 2004. *The bovine digital cushion - how crucial is it to contusions on the bearing surface of the claw of the cow?*, London, Bailliere Tindall Ltd.
- LUBBE, K., WIELSCH, B., BOSSE, I., BAHRAMSOLTANI, M. & MULLING, C. Claw Horn Disruption: An In Vitro Study of Early Pathomechanisms. In: FIEDLER, A. A. S., K., ed. 19th International Symposium and 11th Conference Lameness in Ruminants, 7th to 9th September 2017 Munich, Germany.
- LUCEY, S., ROWLANDS, G. J. & RUSSELL, A. M. 1986. The association between lameness and fertility in dairy cows. *Veterinary Record*, 118, 628-631.
- LUNN, D., SPIEGELHALTER, D., THOMAS, A. & BEST, N. 2009. The BUGS project: Evolution, critique and future directions (with discussion). *Statistics in Medicine*, 28, 3049-3082.
- MACHADO, V. S., CAIXETA, L. S., MCART, J. A. A. & BICALHO, R. C. 2010. The effect of claw horn disruption lesions and body condition score at dry-off on survivability, reproductive performance, and milk production in the subsequent lactation. *Journal of Dairy Science*, 93, 4071-4078.
- MACLEAN, C. W. 1971. The long-term effects of laminitis in dairy cows. *Veterinary Record*, 89, 34-37.
- MAERTENS, W., VANGEYTE, J., BAERT, J., JANTUAN, A., MERTENS, K. C., CAMPENEERE, S. D., PLUK, A., OPSOMER, G., WEYENBERG, S. V. & NUFFEL, A. V. 2011. Development of a real time cow gait tracking and analysing tool to assess lameness using a pressure sensitive walkway: the GAITWISE system. *Biosystems Engineering*, 110, 29-39.
- MAHER, P., GOOD, M. & MORE, S. 2008. Trends in cow numbers and culling rate in the Irish cattle population, 2003 to 2006. *Irish Veterinary Journal*, 61, 455.

- MANSKE, T., HULTGREN, J. & BERGSTEN, C. 2002. Prevalence and interrelationships of hoof lesions and lameness in Swedish dairy cows. *Preventive Veterinary Medicine*, 54, 247-263.
- MANSON, F. J. & LEAVER, J. D. 1988. The influence of concentrate amount on locomotion and clinical lameness in dairy cattle. *Animal Production*, 47, 185-190.
- MAXWELL, O. J., HUDSON, C. D. & HUXLEY, J. N. 2015. Effect of early lactation foot trimming in lame and non-lame dairy heifers: a randomised controlled trial. *Veterinary Record*, 177, 100.
- MUGGLI, E., WEIDMANN, E., KIRCHER, P. & NUSS, K. 2016. Radiographic Measurement of Hindlimb Digit Length in Standing Heifers. *Anat Histol Embryol*, 45, 463-468.
- MULLING, C. Theories on the pathogenesis of white line disease - an anatomical perspective. Proceedings of the 12th Int Symp of Lameness in Ruminants, 9th - 13th January 2002 Orlando, Florida.
- MULLING, C. & GREENOUGH, P. R. Physiopathology of the Foot. XXIV World Buiatrics Congress, 2006 Nice, France.
- MURRAY, R. D., DOWNHAM, D. Y., CLARKSON, M. J., FAULL, W. B., HUGHES, J. W., MANSON, F. J., MERRITT, J. B., RUSSELL, W. B., SUTHERST, J. E. & WARD, W. R. 1996. Epidemiology of lameness in dairy cattle: description and analysis of foot lesions. *Veterinary Record*, 138, 586-591.
- NELSON, A. J. & CATTELL, M. B. 2001. Culling and laminitis: real herds, real cows, real deaths. *Bovine Practitioner*, 35, 42-45.
- NEWSOME, R., GREEN, M. J., BELL, N. J., BOLLARD, N. J., MASON, C. S., WHAY, H. R. & HUXLEY, J. N. 2017b. A prospective cohort study of digital cushion and corium thickness. Part 2: does thinning of the digital cushion and corium lead to lameness and claw horn disruption lesions? *Journal of Dairy Science*, 100, 4759 - 4771.
- NEWSOME, R., GREEN, M. J., BELL, N. J., CHAGUNDA, M. G. G., MASON, C. M., STURROCK, C. J., WHAY, H. R. & HUXLEY, J. N. 2016. Linking bone development on the caudal aspect of the distal phalanx with lameness during life. *Journal of Dairy Science*, 99, 4512-4525.
- NIELSEN, L. A. & HENRIKSSON, K. G. 2007. Pathophysiological mechanisms in chronic musculoskeletal pain (fibromyalgia): the role of central and peripheral sensitization and pain disinhibition. *Clinical Rheumatology*, 21, 465-80.
- NIEUWHOF, G. J., NORMAN, H. D. & DICKINSON, F. N. 1989. Phenotypic trends in herd life of dairy cows in the United States. *Journal of Dairy Science*, 72, 726 - 736.
- NILSSON, S. A. 1963. Clinical, morphological and experimental studies of laminitis in cattle. *Acta vet. scand.*, 4.
- NOCEK, J. E. 1997. Bovine acidosis: implications on laminitis. *Journal of Dairy Science*, 80, 1005-1028.
- NOR, N. M., STEENEVELD, W. & HOGEVEEN, H. 2014. The average culling rate of Dutch dairy herds over the years 2007 to 2010 and its association with herd reproduction, performance and health. *J Dairy Res*, 81, 1-8.
- NORDLUND, K., COOK, N. B. & OETZEL, G. R. 2004a. Investigation Strategies for Laminitis Problem Herds. *Journal of Dairy Science*, 87, E27 - E35.
- NORDLUND, K. V., COOK, N. B. & OETZEL, G. R. 2004b. Investigation strategies for laminitis problem herds. *Journal of Dairy Science*, 87, E27-E35.
- NORRING, M., HÄGGMAN, J., SIMOJOKI, H., TAMMINEN, P., WINCKLER, C. & PASTELL, M. 2014. Short communication: Lameness impairs feeding behavior of dairy cows. *Journal of Dairy Science*.
- NUSS, K. 2014. The role of biomechanical factors in the development of sole ulcer in dairy cattle *Cattle Lameness Conference*. Worcester, UK.

- OFFER, J. E., FISHER, G. E. J., KEMPSON, S. A. & LOGUE, D. N. 2001. The effect of feeding grass silage in early pregnancy on claw health during first lactation. *Veterinary Journal*, 161, 186-193.
- OFFER, J. E., LEACH, K. A., BROCKLEHURST, S. & LOGUE, D. N. 2003. Effect of forage type on claw horn lesion development in dairy heifers. *Veterinary Journal*, 165, 221-227.
- OFFER, J. E., MCNULTY, D. & LOGUE, D. N. 2000. Observations of lameness, hoof conformation and development of lesions in dairy cattle over four lactations. *Veterinary Record*, 147, 105-9.
- ONYIRO, O. M., OFFER, J. & BROTHERSTONE, S. 2008. Risk factors and milk yield losses associated with lameness in Holstein-Friesian dairy cattle. *Animal*, 2, 1230-7.
- OSSENT, P. & LISCHER, C. 1998. Bovine laminitis: the lesions and their pathogenesis. *In Practice*, 20.
- ØSTERGAARD, S., CHAGUNDA, M. G. G., FRIGGENS, N. C., BENNEDSGAARD, T. W. & KLAAS, I. C. 2005. A Stochastic Model Simulating Pathogen-Specific Mastitis Control in a Dairy Herd. *Journal of Dairy Science*, 88, 4243-4257.
- PALMER, M. & CONNELL, N. 2015. Digital Dermatitis in Dairy Cows: A Review of Risk Factors and Potential Sources of Between-Animal Variation in Susceptibility. *Animals*, 5, 0369.
- PASTELL, M., HANNINEN, L., PASSILLE, A. M. D. & RUSHEN, J. 2010. Measures of weight distribution of dairy cows to detect lameness and the presence of hoof lesions. *Journal of Dairy Science*, 93, 954-960.
- PEREZ-CABAL, M. A. & CHARFEDDINE, N. 2016. Short communication: association of foot and leg conformation and body weight with claw disorders in Spanish Holstein cows. *Journal of Dairy Science*, 99, 9104-9108.
- PETERSE, D. J. 1979. Nutrition as a possible factor in the pathogenesis of ulcers of the sole in cattle. *Tijdschrift voor Diergeneeskunde*, 104, 966-970.
- PHILIPOT, J. M., PLUVINAGE, P., CIMAROSTI, I., SULPICE, P. & BUGNARD, F. 1994. Risk factors of dairy cow lameness associated with housing conditions. *Veterinary Research*, 25, 244-248.
- PLUK, A., BAHR, C., LEROY, T., POURSAHERI, A., SONG, X., VRANKEN, E., MAERTENS, W., VAN NUFFEL, A. & BERCKMANS, D. 2010. Evaluation of Step Overlap as an Automatic Measure in Dairy Cow Locomotion. *Transactions of the ASABE*, 53, 1305.
- POLIT, D. F. & BECK, C. T. 2010. Generalization in quantitative and qualitative research: myths and strategies. *Int J Nurs Stud*, 47, 1451-8.
- POLLITT, C. C. 1996. Basement membrane pathology: a feature of acute equine laminitis. *Equine Veterinary Journal*, 28, 38-46.
- POLLITT, C. C. & DAVIES, C. T. 1998. Equine laminitis: its development coincides with increased sublamellar blood flow. *Equine Vet J Suppl*, 125-32.
- POTTERTON, S. L., BELL, N. J., WHAY, H. R., BERRY, E. A., ATKINSON, O. C., DEAN, R. S., MAIN, D. C. & HUXLEY, J. N. 2012. A descriptive review of the peer and non-peer reviewed literature on the treatment and prevention of foot lameness in cattle published between 2000 and 2011. *Veterinary Journal*, 193, 612-6.
- POURSAHERI, A., BAHR, C., PLUK, A., VAN NUFFEL, A. & BERCKMANS, D. 2010. Real-time automatic lameness detection based on back posture extraction in dairy cattle: Shape analysis of cow with image processing techniques. *Computers and Electronics in Agriculture*, 74, 110-119.
- PRENTICE, D. E. & NEAL, P. A. 1972. Some observations on the incidence of lameness in dairy cattle in West Cheshire. *Veterinary Record*, 91, 1-6.
- PRITCHARD, T., COFFEY, M., MRODE, R. & WALL, E. 2013. Genetic parameters for production, health, fertility and longevity traits in dairy cows. *Animal*, 7, 34-46.
- PRYCE, J. E., NIELSEN, B. L., VEERKAMP, R. F. & SIMM, G. 1999. Genotype and feeding system effects and interactions for health and fertility traits in dairy cattle. *Livestock Production Science*, 57, 193-201.

- PRYCE, J. E., VEERKAMP, R. F., THOMPSON, R., HILL, W. G. & SIMM, G. 1997. Genetic aspects of common health disorders and measures of fertility in Holstein Friesian dairy cattle. *Animal Science*, 65, 353-360.
- R CORE TEAM 2016. R: A language and environment for statistical computing. R Foundation for Statistical Computing. Vienna, Austria.
- RABASH, J., CHARLTON, C., BROWNE, W. J., HEALY, M. & CAMERON, B. 2009. *MLwiN Version 2.1* Centre for Multilevel Modelling, University of Bristol.
- RABER, M., LISCHER, C. J., GEYER, H. & OSSENT, P. 2004. The bovine digital cushion - a descriptive anatomical study. *Veterinary Journal*, 167, 258-264.
- RABER, M., SCHEEDER, M. R. L., OSSENT, P., LISCHER, C. J. & GEYER, H. 2006. The content and composition of lipids in the digital cushion of the bovine claw with respect to age and location - a preliminary report. *The Veterinary Journal*, 172, 173-177.
- RANDALL, L. V., GREEN, M. J., CHAGUNDA, M. G., MASON, C., GREEN, L. E. & HUXLEY, J. N. 2016. Lameness in dairy heifers; impacts of hoof lesions present around first calving on future lameness, milk yield and culling risk. *Preventive Veterinary Medicine*, 133, 52-63.
- RANDALL, L. V., GREEN, M. J., CHAGUNDA, M. G. G., MASON, C., ARCHER, S. C., GREEN, L. E. & HUXLEY, J. N. 2015. Low body condition predisposes cattle to lameness: An 8-year study of one dairy herd. *Journal of Dairy Science*, 98, 3766-3777.
- READER, J. D., GREEN, M. J., KALER, J., MASON, S. A. & GREEN, L. E. 2011. Effect of mobility score on milk yield and activity in dairy cattle. *Journal of Dairy Science*, 94, 5045-5052.
- RIOJA-LANG, F. C., ROBERTS, D. J., HEALY, S. D., LAWRENCE, A. B. & HASKELL, M. J. 2009. Dairy cows trade-off feed quality with proximity to a dominant individual in Y-maze choice tests. *Applied Animal Behaviour Science*, 117, 159-164.
- ROBERTS, D. J. & MARCH, M. 2013. *Feeding Systems for Dairy Cows: Homegrown versus By-Product Feeds*, Nottingham, Nottingham University Press.
- ROCKHILL, B., NEWMAND, B. & WEINBERG, C. 1998. Use and misuse of population attributable fractions. *American Journal of Public Health*, 88, 15-19.
- ROMS. 2017. *Register of Mobility Scorers* [Online]. Available: <http://roms.org.uk/> [Accessed 19.06.2017].
- RUSSELL, A. M., ROWLANDS, G. J., SHAW, S. R. & WEAVER, A. D. 1982. Survey of lameness in British dairy cattle. *Veterinary Record*, 111, 155-60.
- SANDERS, A. H., SHEARER, J. K. & DE VRIES, A. 2009. Seasonal incidence of lameness and risk factors associated with thin soles, white line disease, ulcers, and sole punctures in dairy cattle. *Journal of Dairy Science*, 92, 3165-3174.
- SCHOPKE, K., WEIDLING, S., PIJL, R. & SWALVE, H. H. 2013. Relationships between bovine hoof disorders, body condition traits, and test-day yields. *Journal of Dairy Science*, 96, 679-689.
- SHEARER, J. K. 2010. Nutritional and animal welfare implications to lameness. In: EASTRIDGE, M. L. (ed.). Indiana: Purdue University Press.
- SHEARER, J. K., AMSTEL, S. R. V. & BRODERSEN, B. W. 2012. Clinical diagnosis of foot and leg lameness in cattle. (Special Issue: Diagnostic pathology.). *Veterinary Clinics of North America, Food Animal Practice*, 28, 535-556.
- SHERWIN, V. E., HUDSON, C. D., HENDERSON, A. & GREEN, M. J. 2016. The association between age at first calving and survival of first lactation heifers within dairy herds. *Animal*, 10, 1877-1882.
- SOGSTAD, Å. M., FJELDAAS, T. & OSTERAS, O. 2005. Lameness and claw lesions of the Norwegian Red dairy cattle housed in free stalls in relation to environment, parity and stage of lactation. *Acta Veterinaria Scandinavica*, 46, 203 - 217.
- SOGSTAD, Å. M., ØSTERÅS, O., FJELDAAS, T. & NAFSTAD, O. 2007. Bovine claw and limb disorders related to culling and carcass characteristics. *Livestock Science*, 106, 87-95.

- SOLANO, L., BARKEMA, H. W., PAJOR, E. A., MASON, S., LEBLANC, S. J., NASH, C. G., HALEY, D. B., PELLERIN, D., RUSHEN, J., DE PASSILLE, A. M., VASSEUR, E. & ORSEL, K. 2016. Associations between lying behavior and lameness in Canadian Holstein-Friesian cows housed in freestall barns. *Journal of Dairy Science*, 99, 2086-101.
- SOLANO, L., BARKEMA, H. W., PAJOR, E. A., MASON, S., LEBLANC, S. J., ZAFFINO HEYERHOFF, J. C., NASH, C. G., HALEY, D. B., VASSEUR, E., PELLERIN, D., RUSHEN, J., DE PASSILLE, A. M. & ORSEL, K. 2015. Prevalence of lameness and associated risk factors in Canadian Holstein-Friesian cows housed in freestall barns. *Journal of Dairy Science*, 98, 6978-91.
- SØRENSEN, J. T., ENEVOLDSEN, C. & HOUE, H. 1995. A stochastic model for simulation of the economic consequences of bovine virus diarrhoea virus infection in a dairy herd. *Preventive Veterinary Medicine*, 23, 215-227.
- SPRECHER, D. J., HOSTETLER, D. E. & KANEENE, J. B. 1997. A lameness scoring system that uses posture and gait to predict dairy cattle reproductive performance. *Theriogenology*, 47, 1179-87.
- STEENLAND, K. & ARMSTRONG, B. 2006. An overview of methods for calculating the burden of disease due to specific risk factors. *Epidemiology*, 17, 512-9.
- STONE, W. C. 2004. Nutritional Approaches to Minimize Subacute Ruminal Acidosis and Laminitis in Dairy Cattle. *Journal of Dairy Science*, 87, E13-E26.
- TADICH, N., FLOR, E. & GREEN, L. 2010. Associations between hoof lesions and locomotion score in 1098 unsound dairy cows. *Veterinary Journal*, 184, 60-5.
- TARLTON, J. F., HOLAH, D. E., EVANS, K. M., JONES, S., PEARSON, G. R. & WEBSTER, A. J. F. 2002. Biomechanical and histopathological changes in the support structures of bovine hooves around the time of first calving. *Veterinary Journal*, 163, 196-204.
- TELEZHENKO, E., BERGSTEN, C., MAGNUSSON, M. & NILSSON, C. 2009. Effect of different flooring systems on claw conformation of dairy cows. *J Dairy Sci*, 92, 2625-33.
- THOEFNER, M. B., POLLITT, C. C., EPS, A. W. V., MILINOVICH, G. J., TROTT, D. J., WATTLE, O. & ANDERSEN, P. H. 2004. Acute bovine laminitis: a new induction model using alimentary oligofructose overload. *Journal of Dairy Science*, 87, 2932-2940.
- THOMAS, H. J., MIGUEL-PACHECO, G. G., BOLLARD, N. J., ARCHER, S. C., BELL, N. J., MASON, C., MAXWELL, O. J., REMNANT, J. G., SLEEMAN, P., WHAY, H. R. & HUXLEY, J. N. 2015. Evaluation of treatments for claw horn lesions in dairy cows in a randomized controlled trial. *Journal of Dairy Science*, 98, 4477-86.
- THORUP, V. M., MUNKSGAARD, L., ROBERT, P. E., ERHARD, H. W., THOMSEN, P. T. & FRIGGENS, N. C. 2015. Lameness detection via leg-mounted accelerometers on dairy cows on four commercial farms. *Animal*, 9, 1704-12.
- THORUP, V. M., NIELSEN, B. L., ROBERT, P.-E., GIGER-REVERDIN, S., KONKA, J., MICHIE, C. & FRIGGENS, N. C. 2016. Lameness affects cow feeding but not rumination behavior as characterized from sensor data. *Frontiers in Veterinary Science*, 3.
- TISDALL, D. A., BROWN, W. J., GROENEVELT, M., BELL, N. J., BARRETT, D. C. & MAIN, D. C. J. The relationship between body condition score and mobility score in dairy cows on four commercial UK farms. In: WHAY, H. & HOCKENHULL, J., eds. 17th International Symposium and 9th International Conference on Lameness in Ruminants, 2013 Bristol, UK.
- TRANter, W. P. & MORRIS, R. S. 1991. A case study of lameness in three dairy herds. *New Zealand Veterinary Journal*, 39, 88-96.
- TSURUTA, S., MISZTAL, I. & LAWLOR, T. J. 2005. Changing Definition of Productive Life in US Holsteins: Effect on Genetic Correlations. *Journal of Dairy Science*, 88, 1156-1165.
- VAN DER TOL, P. P. J., METZ, J. H. M., NOORDHUIZEN-STASSEN, E. N., BACK, W., BRAAM, C. R. & WEIJS, W. A. 2002. The pressure distribution under the bovine claw during square standing on a flat substrate. *Journal of Dairy Science*, 85, 1476 - 1481.
- VAN EPS, A. W. & POLLITT, C. C. 2006. Equine laminitis induced with oligofructose. *Equine Vet J*, 38, 203-8.

- VAN HERTEM, T., MALTZ, E., ANTLER, A., ROMANINI, C. E. B., VIAZZI, S., BAHR, C., SCHLAGETER-TELLO, A., LOKHORST, C., BERCKMANS, D. & HALACHMI, I. 2013. Lameness detection based on multivariate continuous sensing of milk yield, rumination, and neck activity. *Journal of Dairy Science*, 96, 4286-4298.
- VAN NUFFEL, A., ZWERTVAEGHER, I., VAN WEYENBERG, S., PASTELL, M., THORUP, V. M., BAHR, C., SONCK, B. & SAEYS, W. 2015. Lameness Detection in Dairy Cows: Part 2. Use of Sensors to Automatically Register Changes in Locomotion or Behavior. *Animals : an Open Access Journal from MDPI*, 5, 861-885.
- VANEGAS, J., OVERTON, M., BERRY, S. L. & SISCHO, W. M. 2006. Effect of rubber flooring on claw health in lactating dairy cows housed in free-stall barns. *Journal of Dairy Science*, 89, 4251-4258.
- VIAZZI, S., BAHR, C., SCHLAGETER-TELLO, A., VAN HERTEM, T., ROMANINI, C. E., PLUK, A., HALACHMI, I., LOKHORST, C. & BERCKMANS, D. 2013. Analysis of individual classification of lameness using automatic measurement of back posture in dairy cattle. *J Dairy Sci*, 96, 257-66.
- VOKEY, F. J., GUARD, C. L., ERB, H. N. & GALTON, D. M. 2001. Effects of Alley and Stall Surfaces on Indices of Claw and Leg Health in Dairy Cattle Housed in a Free-Stall Barn. *Journal of Dairy Science*, 84, 2686-2699.
- WALKER, S. L., SMITH, R. F., ROUTLY, J. E., JONES, D. N., MORRIS, M. J. & DOBSON, H. 2008. Lameness, activity time-budgets, and estrus expression in dairy cattle. *Journal of Dairy Science*, 91, 4552-9.
- WARNICK, L. D., JANSSEN, D., GUARD, C. L. & GROHN, Y. T. 2001. The effect of lameness on milk production in dairy cows. *Journal of Dairy Science*, 84, 1988-1997.
- WEBSTER, A. J. 2001. Effects of housing and two forage diets on the development of claw horn lesions in dairy cows at first calving and in first lactation. *Veterinary Journal*, 162, 56-65.
- WEBSTER, A. J. 2002. Effects of housing practices on the development of foot lesions in dairy heifers in early lactation. *Veterinary Record*, 151, 9 - 12.
- WEIGEL, K. A., PALMER, R. W. & CARAVIELLO, D. Z. 2003. Investigation of Factors Affecting Voluntary and Involuntary Culling in Expanding Dairy Herds in Wisconsin using Survival Analysis. *Journal of Dairy Science*, 86, 1482-1486.
- WELLS, S. J., TRENT, A. M., MARSH, W. E., MCGOVERN, P. G. & ROBINSON, R. A. 1993. Individual cow risk factors for clinical lameness in lactating dairy cows. *Preventive Veterinary Medicine*, 17, 95-109.
- WHAY, H. 2002. Locomotion scoring and lameness detection in dairy cattle. *In Practice*, 24, 444-449.
- WHAY, H. R., MAIN, D. C. J., GREEN, L. E. & WEBSTER, A. J. F. 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, H. R. & SHEARER, J. K. 2017. The Impact of Lameness on Welfare of the Dairy Cow. *Vet Clin North Am Food Anim Pract*, 33, 153-164.
- WHAY, H. R., WATERMAN, A. E. & WEBSTER, A. J. F. 1997. Associations between locomotion, claw lesions and nociceptive threshold in dairy heifers during the peri-partum period. *Veterinary Journal*, 154, 155-161.
- WHITAKER, D. A., KELLY, J. M. & SMITH, E. J. 1983. Incidence of lameness in dairy cows. *Veterinary Record*, 113, 60-62.
- WHITAKER, D. A., KELLY, J. M. & SMITH, S. 2000. Disposal and disease rates in 340 British dairy herds. *Veterinary Record*, 146, 363-367.
- WHITAKER, D. A., MACRAE, A. I. & BURROUGH, E. 2004. Disposal and disease rates in British dairy herds between April 1998 and March 2002. *Veterinary Record*, 155, 43-47.
- WILKINSON, D. J. 2009. Stochastic modelling for quantitative description of heterogeneous biological systems. *Nature Reviews Genetics*, 10, 122-133.

- WILSON-WELDER, J., ALT, D. & NALLY, J. 2015. Digital Dermatitis in Cattle: Current Bacterial and Immunological Findings. *Animals*, 5, 0400.
- WINKLER, B. & MARGERISON, J. K. 2012. Mechanical properties of the bovine claw horn during lactation. *Journal of Dairy Science*, 95, 1714-1728.
- WOODHOUSE, G. & GOLDSTEIN, H. 1988. Educational Performance Indicators and LEA League Tables. *Oxford Review of Education*, 14.
- WOOLF, C. J. 2011. Central sensitization: implications for the diagnosis and treatment of pain. *Pain*, 152, S2-15.
- ZACHUT, M., HONIG, H., STRIEM, S., ZICK, Y., BOURA-HALFON, S. & MOALLEM, U. 2013. Periparturient dairy cows do not exhibit hepatic insulin resistance, yet adipose-specific insulin resistance occurs in cows prone to high weight loss. *Journal of Dairy Science*, 96, 5656-5669.

Appendix

Supplementary to Chapter 5; Figures

Supplementary to Figure 4.1. Table of cumulative frequencies for white line lesion scores within each examination point for 158 heifers calving during the time period August 2003 to March 2006 and lesion scored during the period September 2003 to January 2006 at the Scotland's Rural College (SRUC) Dairy Research and Innovation Centre. Examination points 1 to 3 represent; 1 = 0 to 2 months pre-calving, 2 = 0 to 2 months post-calving and 3 = 2 to 4 months post-calving.

Examination point 1	
Lesion score	Cumulative frequency
1	0.63
2	0.90
3	0.98
4	0.99
5	1.00
Examination point 2	
Lesion score	
1	0.25
2	0.63
3	0.86
4	0.95
5	0.99
6	0.99
7	1.00
Examination point 3	
Lesion score	
1	0.38
2	0.72
3	0.91
4	0.98
5	1.00

Supplementary to Figure 4.2. Table of cumulative frequencies for sole lesion scores within each examination point for 158 heifers calving during the time period August 2003 to March 2006 and lesion scored during the period September 2003 to January 2006 at the Scotland's Rural College (SRUC) Dairy Research and Innovation Centre. Examination points 1 to 3 represent; 1 = 0 to 2 months pre-calving, 2 = 0 to 2 months post-calving and 3 = 2 to 4 months post-calving.

Examination point 1	
Lesion score	Cumulative frequency
1	0.59
2	0.92
3	0.97
4	1.00

Examination point 2	
Lesion score	
1	0.18
2	0.70
3	0.86
4	0.93
5	0.96
6	1.00
Examination point 3	
Lesion score	
1	0.38
2	0.72
3	0.91
4	0.98
5	1.00

Supplementary to Figure 4.4. Table showing mean number of days in herd for each sole lesion category 2 to 4 months post-calving (lesion scores for categories; 1 = 0 to 1, 2 = 2, 3 = 3, 4 = 4 to 8) for 157 heifers calving during the time period August 2003 to March 2006 and lesion scored during the period September 2003 to January 2006 at the Scotland's Rural College (SRUC) Dairy Research and Innovation Centre.

Lesion score category	Mean days in herd
1	1631.88
2	1700.40
3	1344.22
4	1305.89

Supplementary to Figure 4.5. Table showing mean for number of days in herd for the presence or absence of digital dermatitis 2 to 4 months post-calving (lesion scores for categories; 0 = lesion absent, 1 = lesion present) for 157 heifers calving during the time period August 2003 to March 2006 and lesion scored during the period September 2003 to January 2006 at the Scotland's Rural College (SRUC) Dairy Research and Innovation Centre.

Lesion score category	Mean days in herd
1	1557.36
2	1216.6

Supplementary to Chapter 5; Example OpenBUGS code for Model 5.2

The example code given here is based on that developed for Model 5.2 to investigate the contribution that exposure to BCS < 2 in the previous 1 to 30 days makes towards the total number of lameness events in one herd using longitudinal data. The outcome was farmer identified lameness (all causes included DD, SU, SH and WLD) in a 30 day risk period

```

model
{
  for (k in 1:22) { beta[k] ~ dflat() }
  tau.u2 ~ dgamma(0.001,0.001)
  sigma2.u2 <- 1/tau.u2

  for(i in 1:N) {
    Lame_in_risk_period[i] ~ dbin(p[i],denom[i])

    logit(p[i]) <- beta[1] * cons[i]
    + beta[2] * TimeSincePrevLameCAT1_6_1[i]
    + beta[3] * TimeSincePrevLameCAT1_6_2[i]
    + beta[4] * TimeSincePrevLameCAT1_6_3[i]
    + beta[5] * TimeSincePrevLameCAT1_6_4[i]
    + beta[6] * TimeSincePrevLameCAT1_6_5[i]
    + beta[7] * Yield_lagged[i]
    + beta[8] * Yield[i]
    + beta[9] * DIM305[i]
    + beta[10] * AnimalParity_2[i]
    + beta[11] * AnimalParity_3[i]
    + beta[12] * AnimalParity_4[i]
    + beta[13] * AnimalParity_5[i]
    + beta[14] * AnimalParity_6[i]
    + beta[15] * year_in_study_1[i]
    + beta[16] * year_quarter_2[i]
    + beta[17] * year_quarter_3[i]
    + beta[18] * year_quarter_4[i]
    + beta[19] * BCS1_60_99[i]
    + beta[20] * BCS1_60_0[i]
    + beta[21] * BCS61_120_99[i]
    + beta[22] * BCS61_120_0[i]
    + u2[AnimalID[i]] * cons[i]

    # Intercept
    # Time since previous lameness 1 to 30 days
    # Time since previous lameness 31 to 60 days
    # Time since previous lameness 61 to 90 days
    # Time since previous lameness 91 to 120 days
    # Time since previous lameness > 120 days
    # Yield previous month
    # Current yield
    # Days in milk
    # Parity 2
    # Parity 3
    # Parity 4
    # Parity 5
    # Parity 6 +
    # Month in herd
    # April to June
    # July to September
    # October to December
    # BCS in last 0 to 2 months missing
    # BCS < 2 in last 0 to 2 months
    # BCS in last 2 to 4 months missing
    # BCS < 2 in last 2 to 4 months

    pred.lame[i] ~ dbin(p[i],denom[i])
    pred.lame_BCS_baseline[i] <- BCS1_60_0[i] * pred.lame[i]
    baseline scenario

    logit(pred.lame_BCS_scenario[i]) <- beta[1] * cons[i]
    + beta[2] * TimeSincePrevLameCAT1_6_1[i]
    + beta[3] * TimeSincePrevLameCAT1_6_2[i]
    + beta[4] * TimeSincePrevLameCAT1_6_3[i]
    + beta[5] * TimeSincePrevLameCAT1_6_4[i]
    + beta[6] * TimeSincePrevLameCAT1_6_5[i]
    + beta[7] * Yield_lagged[i]
    + beta[8] * Yield[i]
    + beta[9] * DIM305[i]
    + beta[10] * AnimalParity_2[i]
    + beta[11] * AnimalParity_3[i]
    + beta[12] * AnimalParity_4[i]
    + beta[13] * AnimalParity_5[i]
    + beta[14] * AnimalParity_6[i]
    + beta[15] * year_in_study_1[i]
    + beta[16] * year_quarter_2[i]
    + beta[17] * year_quarter_3[i]
    + beta[18] * year_quarter_4[i]
    + beta[19] * BCS1_60_99[i]
    + 0 * BCS1_60_0[i] # move to baseline (BCS <2 yes)
    + beta[21] * BCS61_120_99[i]
    + beta[22] * BCS61_120_0[i]
    + u2[AnimalID[i]] * cons[i]

    lame_BCS_bin_all[i] ~ dbin(pred.lame_BCS_scenario[i],denom[i])
  }
}

```

Estimates for beta [k] for the baseline scenario

Estimates for beta [k] for the scenario investigated (no exposure to BCS < 2 in the previous 0 to 2 months)

```

lame_BCS_scenario[i] <- lame_BCS_bin_all[i] * BCS1_60_0[i]      # prediction of lameness events for
scenario investigated

}

for (j in 1:n2) {                                              # Cow level random effect
u2[j] ~ dnorm(0,tau.u2)

}

# Calculation of PAF from predictions

sum_lame_BCS_baseline <- sum(pred.lame_BCS_baseline[])
sum_lame_BCS_scenario <- sum(lame_BCS_scenario[])

total_lame <- sum(pred.lame[])

PAF <- (sum_lame_BCS_baseline - sum_lame_BCS_scenario) / total_lame
}

```

Supplementary to Chapter 6; Example OpenBUGS code for Model 6.1

The example code given here is based on that developed for Model 6.1 to investigate the effect of different culling rates on mid-lactation (month 4) herd lameness prevalence. An example loop for heifers and cows in parity 2 for months 1 to 4 of each lactation have been shown in this example.

```

Model
{

#LOOP FOR HEIFERS

for(i in 1:numberheifers)
{

# Probability of being lame in month 1

Lp1[i]~dunif(0.125,0.126)                                # Baseline month 1 lameness risk
Lame1[i]~dbin( Lp1[i],1)                                # Lameness outcome month 1

# Probability of being lame in month 2

Lp2[i]<- Lp1[i] + delta1[i]
delta1[i]~dunif(-0.00449,-0.00448)                      # Background lameness risk in month 2
                                                         # Change in lameness risk from month 1 to 2

Lameprob2[i]<- (Lame1[i]*Lp2[i]*prev1to4_heifer) + ((1-Lame1[i])*Lp2[i])
Lame2[i]~dbin(Lameprob2[i],1)                            # Probability of being lame in month 2
                                                         # Lameness outcome month 2

# probability of being lame in month 3

Lp3[i]<- Lp2[i] + delta2[i]
delta2[i]~dunif(-0.00395,-0.00394)                      # Background lameness risk in month 3
                                                         # Change in lameness risk from month 2 to 3

Lame0_0[i]<-((step(0.05-(Lame1[i]+Lame2[i])))*Lp3[i])
Lame0_1[i]<-((step(0.05-Lame1[i])*step(Lame2[i]-0.05))*Lp3[i]*prev1to4_heifer)
Lame1_1[i]<-((step(Lame1[i]-0.05)*step(Lame2[i]-0.05))*Lp3[i]*prev5to8_heifer*prev1to4_heifer)
Lame1_0[i]<-((step(Lame1[i]-0.05)*step(0.05-Lame2[i]))*Lp3[i]*prev5to8_heifer)
                                                         } Matrix to predict lameness probability in
                                                         month 2 based on the possible combinations
                                                         of previous lameness outcomes

Lameprob3[i]<-Lame0_0[i]+Lame0_1[i]+Lame1_1[i]+Lame1_0[i]
Lame3[i]~dbin(Lameprob3[i],1)                            # Probability of being lame in month 3
                                                         # Lameness outcome month 3

# probability of being lame in month 4

Lp4[i]<- Lp3[i] + delta3[i]
delta3[i]~dunif(-0.00345,-0.00344)                      # Background lameness risk in month 4
                                                         # Change in lameness risk from month 3 to 4

Lame4_0_0_0[i]<-((step(0.05-Lame1[i]))*(step(0.05-
(Lame3[i]+Lame2[i])))*Lp4[i])
Lame4_1_0_0[i]<-((step(Lame1[i]-0.05))*(step(0.05-
(Lame3[i]+Lame2[i])))*Lp4[i]*prev9toever_heifer)
Lame4_0_0_1[i]<-((step(0.05-Lame1[i]))*(step(0.05-Lame2[i])*step(Lame3[i]-
0.05))*Lp4[i]*prev1to4_heifer)
Lame4_1_0_1[i]<-((step(Lame1[i]-0.05))*(step(0.05-Lame2[i])*step(Lame3[i]-
0.05))*Lp4[i]*prev1to4_heifer*prev9toever_heifer)
Lame4_0_1_1[i]<-((step(Lame1[i]-0.05))*(step(0.05-Lame2[i])*step(Lame3[i]-
0.05))*Lp4[i]*prev1to4_heifer)
Lame4_1_1_1[i]<-((step(Lame1[i]-0.05))*(step(Lame2[i]-0.05)*step(Lame3[i]-
0.05))*Lp4[i]*prev1to4_heifer*prev5to8_heifer*prev9toever_heifer)
Lame4_0_1_0[i]<-((step(0.05-Lame1[i]))*(step(Lame2[i]-0.05)*step(0.05-
Lame3[i]))*Lp4[i]*prev5to8_heifer)
Lame4_1_1_0[i]<-((step(Lame1[i]-0.05))*(step(Lame2[i]-0.05)*step(0.05-
Lame3[i]))*Lp4[i]*prev5to8_heifer*prev9toever_heifer)
                                                         } Matrix to predict lameness probability in
                                                         month 3 based on the possible combinations
                                                         of previous lameness outcomes

Lameprob4[i]<-
Lame4_0_0_0[i]+Lame4_1_0_0[i]+Lame4_0_0_1[i]+Lame4_1_0_1[i]+Lame4_0_1_1[i]+Lame4_1_1_1[i]+Lame4_0_1_0[i]+Lame4_1_1_0[i]
                                                         # Probability of being lame in month 4

Lame4[i]~dbin(Lameprob4[i],1)                            # Lameness outcome month 4

# Reiterate code above for months 5 to 9

```

Calculate the probability of being lame in lactation 1

```
sumlameheifer[i]<-
step((Lame1[i]+Lame2[i]+Lame3[i]+Lame4[i]+Lame5[i]+Lame6[i]+Lame7[i]+Lame8[i]+Lame9[i]) - 0.05)

numberlameheifers<-sum(sumlameheifer[])
probprevlameheifer<-numberlameheifers/numberheifers

}
```

LOOP FOR PARITY 2 COW

```
for(i in 1:numbercowsparity2)
```

```
{
```

probability of being lame in month 1

```
lameheifer[i]~dbern(probprevlameheifer)
Lpc2_1[i]~dunif(0.080,0.198)
```

Draw for being lame in the previous lactation
Baseline risk for month 1

```
Lamecowprob1[i] <- (lameheifer[i]*Lpc2_1[i]*prev9toever_cowparity2) + ((1-
lameheifer[i])*Lpc2_1[i])
```

Probability of being lame in month 1

```
Lamecow1[i]~dbin(Lamecowprob1[i],1)
```

Lame outcome month 1

probability of being lame in month 2

```
Lpc2_2[i]<-Lpc2_1[i] + deltac1[i]
deltac1[i] ~ dunif(-0.00441,-0.00440)
```

Background lameness risk in month 2
Change in lameness risk from month 1 to 2

```
Lamecow2_00[i] <- (step(0.05-(lameheifer[i]+Lamecow1[i]))*Lpc2_2[i])
Lamecow2_01[i] <- ((step(0.05-lameheifer[i])*step(Lamecow1[i]-
0.05))*Lpc2_2[i]*prev1to4_cowparity2)
Lamecow2_11[i] <- ((step(lameheifer[i]-0.05)*step(Lamecow1[i]-
0.05))*Lpc2_2[i]*prev1to4_cowparity2*prev9toever_cowparity2)
Lamecow2_10[i] <- ((step(lameheifer[i]-0.05)*step(0.05-
Lamecow1[i]))*Lpc2_2[i]*prev9toever_cowparity2)
```

Matrix to predict lameness probability in month 2 based on the possible combinations of previous lameness outcomes

```
Lamecowprob2[i]<-
Lamecow2_00[i]+Lamecow2_01[i]+Lamecow2_11[i]+Lamecow2_10[i]
```

Probability of being lame in month 2

```
Lamecow2[i]~dbin(Lamecowprob2[i],1)
```

Lame outcome month 2

probability of being lame in month 3

```
Lpc2_3[i]<-Lpc2_2[i] + deltac2[i]
deltac2[i] ~dunif(-0.00387,-0.00386)
```

Background lameness risk in month 3
Change in lameness risk from month 2 to 3

```
Lamecow3_000[i]<-((step(0.05-lameheifer[i]))*(step(0.05-
(Lamecow1[i]+Lamecow2[i])))*Lpc2_3[i])
Lamecow3_100[i]<-((step(lameheifer[i]-0.05))*(step(0.05-
(Lamecow1[i]+Lamecow2[i])))*Lpc2_3[i]*prev9toever_cowparity2)
Lamecow3_001[i]<-((step(0.05-lameheifer[i]))*(step(0.05-
Lamecow1[i])*step(Lamecow2[i]-0.05))*Lpc2_3[i]*prev1to4_cowparity2)
Lamecow3_101[i]<-((step(lameheifer[i]-0.05))*(step(0.05-
Lamecow1[i])*step(Lamecow2[i]-
0.05))*Lpc2_3[i]*prev1to4_cowparity2*prev9toever_cowparity2)
Lamecow3_011[i]<-((step(0.05-lameheifer[i]))*(step(Lamecow1[i]-
0.05)*step(Lamecow2[i]-
0.05))*Lpc2_3[i]*prev1to4_cowparity2*prev5to8_cowparity2)
Lamecow3_111[i]<-((step(lameheifer[i]-0.05))*(step(Lamecow1[i]-
0.05)*step(Lamecow2[i]-
0.05))*Lpc2_3[i]*prev1to4_cowparity2*prev5to8_cowparity2*prev9toever_cowpa
rity2)
Lamecow3_010[i]<-((step(0.05-lameheifer[i]))*(step(Lamecow1[i]-
0.05)*step(Lamecow2[i]-
0.05))*Lpc2_3[i]*prev1to4_cowparity2*prev5to8_cowparity2)
Lamecow3_110[i]<-((step(lameheifer[i]-0.05))*(step(Lamecow1[i]-
0.05)*step(0.05-
Lamecow2[i]))*Lpc2_3[i]*prev5to8_cowparity2*prev9toever_cowparity2)
```

Matrix to predict lameness probability in month 3 based on the possible combinations of previous lameness outcomes

```

Lamecowprob3[i]<-
Lamecow3_000[i]+Lamecow3_100[i]+Lamecow3_001[i]+Lamecow3_101[i]+La
mecow3_011[i]+Lamecow3_111[i]+Lamecow3_010[i]+Lamecow3_110[i]

Lamecow3[i]~dbin(Lamecowprob3[i],1)

# Probability of being lame in month 3

# Change in lameness risk from month 2 to 3
Lpc2_4[i]<-Lpc2_3[i] + deltac3[i]
deltac3[i] ~ dunif(-0.00339,-0.00338)

# Background lameness risk in month 3
# Change in lameness risk from month 2 to 3

Lamecow4_000[i]<-((step(0.05-(lameheifer[i]+Lamecow1[i])))*(step(0.05-
(Lamecow2[i]+Lamecow3[i])))*Lpc2_4[i])
Lamecow4_100[i]<-((step((lameheifer[i]+Lamecow1[i])-0.05))*(step(0.05-
(Lamecow2[i]+Lamecow3[i])))*Lpc2_4[i]*prev9toever_cowparity2)
Lamecow4_001[i]<-((step(0.05-(lameheifer[i]+Lamecow1[i])))*(step(0.05-
Lamecow2[i])*step(Lamecow3[i]-0.05))*Lpc2_4[i]*prev1to4_cowparity2)
Lamecow4_101[i]<-((step((lameheifer[i]+Lamecow1[i])-0.05))*(step(0.05-
Lamecow2[i])*step(Lamecow3[i]-
0.05))*Lpc2_4[i]*prev1to4_cowparity2*prev9toever_cowparity2)
Lamecow4_011[i]<-((step(0.05-
(lameheifer[i]+Lamecow1[i]))*(step(Lamecow2[i]-0.05)*step(Lamecow3[i]-
0.05))*Lpc2_4[i]*prev1to4_cowparity2*prev5to8_cowparity2)
Lamecow4_111[i]<-((step((lameheifer[i]+Lamecow1[i])-
0.05))*(step(Lamecow2[i]-0.05)*step(Lamecow3[i]-
0.05))*Lpc2_4[i]*prev1to4_cowparity2*prev5to8_cowparity2*prev9toever_cowpa
rity2)
Lamecow4_010[i]<-((step(0.05-
(lameheifer[i]+Lamecow1[i]))*(step(Lamecow2[i]-0.05)*step(0.05-
Lamecow3[i]))*Lpc2_4[i]*prev5to8_cowparity2)
Lamecow4_110[i]<-((step((lameheifer[i]+Lamecow1[i])-
0.05))*(step(Lamecow2[i]-0.05)*step(0.05-
Lamecow3[i]))*Lpc2_4[i]*prev5to8_cowparity2*prev9toever_cowparity2)

# Matrix to predict lameness probability in
# month 4 based on the possible combinations
# of previous lameness outcomes

Lamecowprob4[i]<-
Lamecow4_000[i]+Lamecow4_100[i]+Lamecow4_001[i]+Lamecow4_101[i]+La
mecow4_011[i]+Lamecow4_111[i]+Lamecow4_010[i]+Lamecow4_110[i]

Lamecow4[i]~dbin(Lamecowprob4[i],1)

# Probability of being lame in month 4

# Reiterate code above for months 5 to 9

# Reiterate code above for months 5 to 9

# REITERATE LOOP ABOVE FOR PARITY 3 TO 5+

}

prev1to4_heifer ~dunif(3.096,3.308)
prev5to8_heifer ~dunif(2.232,2.395)
prev9toever_heifer ~dunif(1.080,1.214)

prev1to4_cowparity2 ~dunif(3.103,3.316)
prev5to8_cowparity2 ~dunif(2.234,2.398)
prev9toever_cowparity2 ~dunif(1.080,1.214)

# Distributions for previous lameness (1 to 4
# weeks, 5 to 8 weeks and 9 or more
# previously). A number of different distributions
# were explored.

# Repeat for previous lameness risk for parity 3 to 5 +

# Calculate prevalence for month 4

sumlameheifers_mth4<-sum(Lame4[])
sumlamecow2_mth4<-sum(Lamecow4[])
sumlamecow3_mth4<-sum(LamecowP3_M4[])
sumlamecow4_mth4<-sum(LamecowP4_M4[])
sumlamecow5_mth4<-sum(LamecowP5_M4[])

prevalence_mth4<-sumlameheifers_mth4+sumlamecow2_mth4+s
umlamecow3_mth4+sumlamecow4_mth4 + sumlamecow5_mth4

}

```