Influence of somatic cell count in heifers on lifetime milk yield and disease management

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Abstract

The aim was to assess the impact of milk somatic cell count (SCC) during the first lactation on the lifetime milk production of cows, and therefore estimate potential savings through heifer mastitis control. Cow level SCC over the first lactation was summarised as SCC between 5 and 30 days in milk (SCC1), and the geometric mean and variance of first lactation SCC. The impact of SCC1 on cumulative milk yield over different time periods was assessed for cows in Irish, English, and Welsh dairy herds. The impact of SCC1 and the geometric mean and variance of first lactation SCC on lifetime milk yield, and the association between SCC1 and disposal risk were assessed for cows in Irish dairy herds. Increase in SCC throughout the first lactation was associated with large reductions in the milk yield of cows, and increased disposal risk. Bayesian micro-simulation was used to demonstrate the impact in different herd scenarios. This was extended to synthesise evidence on potential savings using previous research, to estimate the economic impact of specific interventions to reduce the prevalence of cows with high SCC1. There was considerable variation between herds in the apparent impact of SCC1 on SCC throughout the first lactation, indicating the importance of a herd specific approach to control. 'Cost effectiveness' of interventions to reduce the prevalence of cows with high SCC1, was found to be highly dependent on the willingness of decision makers to pay for control measures. Increase in herd size was associated with increase in cow SCC, highlighting a need for improved management of mastitis when expansion is planned. An important component of this should be through monitoring and control of mastitis in heifers, especially those in spring-calving Irish dairy herds.

Publications

Chapter 2

Archer, S. C., F. Mc Coy, W. Wapenaar, and M. J. Green. 2013. Association of season and herd size with somatic cell count for cows in Irish, English, and Welsh dairy herds. Vet. J. 196:515-521.

Chapter 3

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Chapter 4

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Chapter 5

Archer, S. C., F. Mc Coy, W. Wapenaar, and M. J. Green. 2013. Association between somatic cell count early in the first lactation and the longevity of Irish dairy cows. J. Dairy Sci. 96:2939-2950.

Chapter 6

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Chapter 7

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"All models are wrong, but some are useful" Box, George E. P.

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Abbreviations

AFC	Age at first calving
BCI	Bayesian credible interval
CNS	Coagulase-negative *Staphylococci spp*.
DIM	Days in milk
FLMY	First lactation milk yield
LiMY	Lifetime milk yield
IMI	Intramammary infection
IQR	Interquartile range
kg	Kilogram(s)
ln	Natural logarithm of
mL	Millilitre
MCMC	Markov chain Monte Carlo
p	Probability
ppp	*Pre-* and *peri-partum*
SCC	Milk somatic cell count in thousands (unless stated)
SCC1	Milk somatic cell count in thousands (unless stated) measured at 5 to 30 days in milk during the first lactation
TDY	Test day milk yield

Chapter 1: Introduction

1.1 Importance of mastitis in dairy cows

Mastitis is one of the most costly endemic diseases of dairy cows (Kossaibati and Esslemont, 1997). Treatment costs, production losses, and reduced sale value of high somatic cell count (SCC) milk are well known consequences of the disease (Halasa et al., 2007). The European Commission Milk Hygiene Directive (92/46) requires that bulk milk for human consumption has a 3 month rolling geometric mean SCC not exceeding 400,000 cells/mL, which is also effectively the international export standard (More, 2009). In some countries, dairies pay a premium for milk with lower SCC (Bradley, 2002) to maximise the shelf life of pasteurised milk (Santos et al., 2003), and cheese yields (Barbano et al., 1991). In addition to the adverse effect of high SCC on milk quality, food safety is adversely affected through increased risk of antibiotic residues and bacterial contamination from infected quarters (van Schaik et al., 2002). Antibiotics are widely used in the treatment and management of mastitis, and drug residues in milk are of public health concern, because resistant strains of bacteria could enter the food chain (White and McDermott, 2001). The negative environmental impact of mastitis has rarely been studied, but if mastitis rates are high, through discarded milk, lower productivity, and increased culling risk, larger herds are required for the same milk output, with relatively more manure, methane, and ammonia produced (Garnsworthy, 2004). The lower efficiency of herds with high mastitis rates is reported to increase potential for global warming, eutrophication, and acidification of the environment per litre of milk (Hospido and Sonesson,

2005). Importantly, mastitis impairs cow welfare (Kemp et al., 2008), and this has potentially serious consequences for the public perception of dairy farming.

1.1.1 Pathogens associated with mastitis

Pathogens associated with bovine mastitis are typically bacteria, and these penetrate the teat canal to cause intramammary infection (IMI) and hence mastitis. One way of classifying pathogens is on niche or host adaption, which in turn determines the epidemiology of infection. The major 'contagious' bacteria *Mycoplasma spp*., *Staphylococcus aureus*, *Streptococcus dysgalactiae*, and *Streptococcus agalactiae* are generally adapted to survive in the bovine udder, often causing persistent IMI which may be associated with increase in herd bulk milk SCC. Transmission occurs during milking, making IMI more common in older cows, or in late lactation due to increased exposure (Barkema et al., 2009). The major 'environmental' bacteria *Escherichia coli* and *Streptococcus uberis* are opportunistic invaders of the udder, can cause persistent IMI during the dry period (Bradley and Green, 2000), and disease in *peri-parturient* cows that have compromised immune defences (Suriyasathaporn et al., 1999). Seasonal variations in mastitis incidence are often consistent with IMI of environmental origin (Bradley and Green, 2005; Morse et al., 1988). These classifications are not absolute and may be strain dependent, since 'environmental' bacteria have potential for contagious spread (Bradley and Green, 2001; Zadoks et al., 2003), and environmental sources of *S. aureus* can lead to IMI in heifers (Roberson et al., 1998). Minor pathogens include Coagulase-Negative *Staphylococcus spp.* (CNS), and IMI can be of environmental or contagious origin depending on species (Piessens et al., 2011). CNS can access the udder of *pre-partum* heifers leading to persistent

IMI, and are commonly isolated early in the first lactation (De Vliegher et al., 2012). Some CNS, along with the common minor pathogen *Corynebacterium bovis* may protect against IMI with major pathogens (Lam et al., 1997).

1.1.2 Milk somatic cell count data

Somatic cells found in milk are predominantly leucocytes. During the innate immune response to IMI, macrophages and mammary epithelial cells produce chemokines that attract peripheral neutrophils to the mammary gland and milk SCC can increase more than 10 fold (Paape et al., 2003; Sordillo et al., 1997). Cow SCC data are collected routinely by dairy farmers participating in recording schemes in many developed dairy nations, and are used as a screening test for subclinical mastitis (Bradley and Green, 2005). The samples for each cow are a composite of milk from all quarters, collected from the milking equipment. Cow SCC data should be interpreted in light of the following limitations: Firstly, misidentification of samples from individual cows can occur. Secondly, SCC data are often from visibly 'normal' cows, and those with clinical mastitis are typically 'missing'. Thirdly, sensitivity and specificity of cow level SCC cut off values for putative IMI require careful consideration depending on the objectives of the test (Dohoo et al., 1981; McDermott et al., 1982). Cow level SCC $\geq$ 200,000 or $\geq$ 400,000 cells/mL can be used to indicate IMI in at least one quarter, and these thresholds had sensitivities of 89% and 60% respectively, and specificities of 75% and 87% respectively for IMI associated with major pathogens in 12 herds in the United States of America (McDermott et al., 1982). Elsewhere test characteristics depend on the herd pathogen distributions. This has been demonstrated for quarter level SCC thresholds; using SCC $\geq$ 200,000 cells/mL to indicate

prevalent IMI with both major and minor pathogens in a Canadian herd, sensitivity and specificity were estimated to be 73%, and 86% respectively (Dohoo and Leslie, 1991). If only IMI with major pathogens were considered sensitivity increased to 84% and specificity was unchanged (Dohoo and Leslie, 1991). Varying the threshold depending on the stage of lactation and parity of cows could increase sensitivity, but this would decrease specificity (Schepers et al., 1997). Bacterial culture has been used as the gold standard test for IMI, requiring aseptic samples of foremilk. The same samples have been used to evaluate SCC (Dohoo and Leslie, 1991; Schepers et al., 1997), but at quarter level the SCC of foremilk may differ to that of the alveolar milk (Sarikaya et al., 2006), which is routinely collected (at cow level) in milk recording schemes. Quarter level studies should be extrapolated to the cow level with caution, mainly because high SCC from infected quarters can be diluted by low SCC milk from uninfected quarters (Barkema et al., 1999a). Despite these limitations, cow level SCC data are widely available, and high values are commonly used as a proxy for IMI. Trends in cow SCC can be informative, and these data are routinely used to monitor udder health and aid management decisions on farms, as well as for research.

1.2 Mastitis in heifers

When evaluating mastitis in dairy herds, the heifer group (primiparous cows) warrants special attention. It is typically the largest parity group, and mastitis risk factors for heifers are likely to differ from those in multiparous cows, for instance when rearing takes place away from the main herd (De

Vliegher et al., 2012). With most rearing systems, heifers have yet to reach mature adult size by their first calving, and this may have a negative influence on resistance to disease through sub-ordinance in the herd (Proudfoot et al., 2012).

1.2.1 Importance of heifers

Expansion of dairy herds worldwide increases demand for heifers in excess of replacement needs. Through selective breeding, heifers can be genetically superior to older cows and are therefore a valuable asset. Average replacement cost has been estimated to be $32,000 /year for a 100 cow herd in the United States of America (Tozer and Heinrichs, 2001), and €45,000 /year for a typical 100 cow Dutch dairy herd (Mohd Nor et al., 2012). Furthermore, there is no return on investment until after the first calving when milk can be sold, and rearing costs are unlikely to be recovered until the second lactation. In a study of English dairy herds, around 30% of heifers born alive were culled prior to a second calving (Brickell and Wathes, 2011), and were likely not profitable, increasing the economic burden of heifer rearing at herd level.

1.2.2 Economic impact of mastitis in heifers

Mastitis has been highlighted as a common problem for primiparous cows early in their first lactation, which is economically important due to its impact on first lactation productivity (De Vliegher et al., 2012; Piepers et al., 2009). Losses are mainly accrued through decreased milk production, and discarded milk (Kossaibati and Esslemont, 1997). However, mastitis has also been associated with reduced longevity (Beaudeau et al., 1993; Seegers et al., 1998), and this has been estimated as the next biggest cost (Heikkilä et al.,

2012; Huijps et al., 2008). Reduced longevity will limit the opportunity to recover the initial rearing costs from heifers that succumb to mastitis. Further losses such as the cost of drugs, veterinary services, diagnostic costs, labour, decreased milk quality, capital investments, and impact on other diseases (Halasa et al., 2007), are typically less, but may be important for particular herds (Huijps et al., 2008). The deleterious effects of high SCC after the first calving has been demonstrated in terms of first lactation milk yield and culling risk (De Vliegher et al., 2005a; 2005b). However, the effect of high SCC early in the first lactation on milk yield and longevity beyond the first lactation has not yet been investigated and is a major focus of this thesis.

1.2.3 Epidemiology of heifer mastitis

Despite lack of exposure to milking equipment, *pre-partum* heifers can develop IMI from around 9 months of age (Trinidad et al., 1990). These infections are persistent, and may manifest early in the first lactation when the incidence rate of clinical mastitis in heifers is at its highest, typically exceeding that for multiparous cows (Barkema et al., 1998b). However, SCC in early lactation is commonly elevated as a normal physiological response to parturition; which is also particularly marked in heifers, can last for a variable length of time (Barkema et al., 1999a; Dohoo, 1993), and biases the use of SCC thresholds for diagnosis of subclinical mastitis in early lactation. Therefore, subclinical heifer mastitis prevalence has mainly been described based on IMI determined by bacterial culture, but methodology has varied. As a result, estimates range from 29% to 75% *pre-partum*, and 12% to 57% *post-partum* (De Vliegher et al., 2012). The predominant major pathogen in these studies was either *S. aureus*, *E. coli*, or *S. uberis*, but CNS were isolated most

frequently (De Vliegher et al., 2012). The epidemiology, and pathogenicity of CNS varies by species. For example *Staphylococcus chromogenes* and *Staphylococus epidermidis* appear adapted to survive in the udder, whereas *Staphylococcus haemolyticus* and *Staphylococcus simulans* appear to be of environmental origin (Piessens et al., 2011). Although cases of clinical mastitis associated with CNS are rare, IMI with *S. chromogenes*, and *S. simulans* can lead to an increase in SCC comparable to that of the major pathogen *S. aureus* (Supré et al., 2011). Importantly, the frequency of isolation of CNS species varies by herd (Piessens et al., 2011; Supré et al., 2011), indicating it may no longer be appropriate to consider these bacteria as a single group, and molecular identification may be required to develop species specific control measures in different herds (Zadoks and Watts, 2009). This may become important in herds with heifer mastitis problems, and where IMI associated with major pathogens are under control (Schukken et al., 2009). A suggested intervention level is when ≥ 15% of heifers have high SCC, or clinical mastitis in early lactation (De Vliegher et al., 2012).

1.2.4 Control of mastitis in heifers

Heifer mastitis is predominantly an early lactation problem (Barkema et al., 1998b), therefore most control measures should focus on the *pre* and *peri-partum* (ppp) period (Green et al., 2007b; 2008). Control of mastitis originating while heifers are in milk (> 30 days) may be important for particular herds, and preventive measures have been described, as for multiparous cows (NMC, 2011). These measures can be targeted towards either contagious or environmental mastitis based on patterns in the occurrence of clinical and subclinical mastitis and culture results from cases (Bradley and Green, 2005).

The relative importance of high SCC early in the first lactation compared to > 30 days in milk (DIM) in the first lactation is investigated in this thesis in relation to first lactation and lifetime milk production.

1.2.4.1 Control of mastitis during the pre- and peri-partum period

Possible herd level management changes to control heifer mastitis can be suggested based on known risk factors (De Vliegher et al., 2012), but intervention studies on mastitis control for ppp heifers are lacking. Individual heifer treatments have the support of intervention studies but require the handling of heifers which increases labour costs and can be hazardous for the operator and animal. This could influence cost effectiveness, compared to herd management changes. Although data on management strategies to prevent heifer mastitis are sparse, in this thesis spending budgets for implementing possible interventions are explored so that the decision maker can at least be aware of the rational 'scope for investment' to reduce disease.

Individual heifer treatments

Heifers can have open teat canals from 80 days prior to calving (Krömker and Friedrich, 2009). Therefore either using teat sealants (McDougall et al., 2008; Parker et al., 2007b; 2008), or *pre-partum* teat disinfection (Lopez-Benavides et al., 2009) has been successful in New Zealand pasture-based systems. *Pre-partum* antibiotic therapy (Nickerson, 2009) has also been assessed, but the impact is herd dependent (Borm et al., 2006), and is only appropriate for herds with heifer mastitis problems associated with a high prevalence of major pathogens to ensure the prudent use of antibiotics (De Vliegher et al., 2012). Mastitis vaccines are in development, but cost

effectiveness has yet to be demonstrated in field trials (McDougall et al., 2009). Individual animal treatments are unlikely to be successful in isolation without consideration of improvements to herd level management.

Herd level management changes

An overall aim is for heifers to be managed to minimise dystocia and *peri-partum* disease to also reduce mastitis risk (Svensson et al., 2006). Stress should be minimised during the transition period but how this is achieved may vary between systems (Barkema et al., 1999b; Parker et al., 2007a; Santman-Berends et al., 2012), emphasising the importance of a herd specific approach to control. For a holistic approach to mastitis control for ppp heifers, herd specific changes may be required to improve environmental hygiene, reduce contagious spread of pathogens, and improve host resistance (De Vliegher et al., 2012); the literature related to these risk factors is summarised as follows.

Environmental mastitis control

Hygienic calving areas (De Vliegher et al., 2004b; Piepers et al., 2011), and udders (Compton et al., 2007a) should be maintained. For heifers at pasture, overgrazing and poaching should be avoided (Green et al., 2007b; 2008) with access tracks maintained (Lopez-Benavides et al., 2007). For heifers that are housed, avoiding deep bedding (Elbers et al., 1998), but maintaining cleanliness of cubicles (Schukken et al., 1990), with frequent scraping of collecting yards and passages (Peeler et al., 2000) should be considered.

Contagious mastitis control

Control of contagious disease in cows (Neave et al., 1966) would be expected to reduce the risk of spread to heifers during milking (Piepers et al., 2011). Cross suckling can lead to IMI directly or indirectly through damage to the suckled teats, and should be avoided during rearing (McDougall et al., 2009). Flies can also act as vectors for mastitis pathogens (Nickerson et al., 1995; Piepers et al., 2011). Resistance of fly populations to common insecticides has been reported, therefore control rather than elimination of flies by chemical means should be emphasised, and avoidance strategies used where possible (Broce, 2006; McDougall et al., 2009). Operators should wear gloves when handling heifer teats (Huijps et al., 2010; Piessens et al., 2011).

Host resistance

Udder oedema is an important risk factor for heifer mastitis (Compton et al., 2007a), and has been associated with excessive energy and mineral intake *pre-partum*, excessive condition loss *post-partum*, increased milk yield, and increased age and size at calving, yet control measures remain unclear (McDougall et al., 2009). For instance, pre-calving milking reduced udder oedema and mastitis, but exacerbated negative energy balance (Daniels et al., 2007; Santos et al., 2004). This may conflict with attempts to minimise body condition loss in early lactation through improved nutritional management, since reduced prevalence of cows with subclinical ketosis is likely to benefit udder health (Compton et al., 2007a). Deficiency of minerals (copper, selenium and zinc), and vitamins (A and E) has been associated with mastitis. Heifers fed home grown forage may be deficient if not supplemented depending on

farm soil status (Heinrichs et al., 2009). The need for supplementation should be assessed carefully as in the absence of deficiency this can have adverse effects on udder health (Bouwstra et al., 2010). Increased concentrate feeding from 11 to 16 months of age was associated with increased SCC early in the first lactation but the reason for this remains unclear (Svensson et al., 2006).

1.2.4.2 Control of contagious mastitis during the first lactation

Methods for the control of contagious mastitis are well known (Neave et al., 1966), although often not satisfactorily applied (Barkema et al., 2009). If contagious mastitis is suspected in lactating heifers, whole herd control measures should be reviewed to reduce the risk of further transmission. Ensuring optimal milking machine operation may be a particular problem for heifers if their teats are smaller or shorter than those of older cows (Baxter et al., 1992; Rasmussen et al., 1998).

1.2.4.3 Control of environmental mastitis during the first lactation

Where contagious mastitis control has been successful, environmental mastitis can become a problem (Bradley, 2002). Control measures focussing on risk factors related to environmental hygiene, and increasing host resistance are important (Barkema et al., 1999b; Green et al., 2007b; 2008). Cubicle housing designed for mature cows may not be suitable for heifers, and this could lead to problems with comfort and hygiene. Grouping heifers separately may therefore have advantages in terms of udder health.

1.3 Study populations used in this research

1.3.1 Background

Consumption of dairy products is increasing worldwide, largely driven by increasing demand from developing countries (OECD-FAO, 2012). European Union milk quotas are to be removed by 2015, providing an opportunity for farmers to increase production and benefit from global trade. The downside of this is exposure to competition and hence potentially volatile world market prices. In a competitive market, maximising the value of milk through minimising SCC is crucial (More, 2009).

1.3.2 Dairy farming in Ireland, England and Wales

There are approximately 19,000 and 11,000 dairy farms in Ireland, and England and Wales respectively (DairyCo, 2012). The data analysed in this thesis are from subsets of these farms that have participated in milk recording schemes. Despite fewer herds, annual milk production in England and Wales is approximately double the 5 billion litre annual production of Ireland (DairyCo, 2012; ICBF, 2011). Population density in Ireland is approximately 4 times lower (67 people /km^2) than in England and Wales (Anon, 2012), meaning relatively more land is available for farming. Compared to other agricultural land use, dairying generates most income (Hennessy et al., 2011). Low-input, spring-calving, pasture-based systems predominate in Ireland, and 85% of milk is exported (More, 2009). In contrast, domestic markets for milk are of major importance for English and Welsh dairy herds; 50% of raw milk is pasteurised for liquid consumption, and 4% of raw milk is exported (DairyCo, 2012). In England and Wales, seasonal variation in milk price therefore favours autumn-

calving. Production systems with higher input costs (largely through feed), that are dependent on scale in terms of milk yield and herd size are therefore required to operate efficiently (Bailey et al., 1997). Trends for fewer, larger herds are evident in both countries (DairyCo, 2012; ICBF, 2011). However, in Ireland the decline in producer numbers is around 10 times less than the 4% annual decline seen in England and Wales (DairyCo, 2012). In general to increase efficiency, optimal management of higher stocking rates is required for Irish pasture-based systems, and optimal management of higher yielding cows is required for English and Welsh confined systems. Both systems can benefit from economies of scale.

1.3.3 Importance of heifer mastitis in Ireland

The Irish dairy industry is well placed to increase production (Lips and Relder, 2005; O' Donnell et al., 2008), and aims for a 50% increase in milk output by 2020 (DAFM, 2011). One way this could be achieved is through increasing herd sizes and stocking rates, to exploit the competitive advantage of low-cost, pasture-based production systems (Läpple et al., 2012). Expansion depends on an increased supply of heifers, and maximising the longevity of cows; the former trend is evident in Irish herds (ICBF, 2011). Due to the predominance of spring-calving systems in Ireland, ppp heifers are typically housed. Herd expansion may lead to overstocking and increase environmental mastitis risk in heifers, unless investment in improved facilities and management can be justified. Therefore the impact of high SCC in heifers on lifetime milk yield (LiMY) and longevity is of huge significance, and is a major focus of this thesis. The importance of heifer mastitis, including the prevalence of heifers with high SCC, has not been reported for Irish herds. This

information is essential to estimate the economic impact of heifer mastitis and monitor herd performance. For interest, comparisons are made to data from English and Welsh herds where there is also a dearth of information in this area.

1.4 Statistical methods used in the thesis

A particular strength of this research is the use of large national milk recording datasets, based on multiple production records from cows in almost 10,000 dairy herds, to make robust statistical inferences of relevance to herds in Ireland, England, and Wales. However, these datasets have a multilevel hierarchical structure (herd, cow, parity, recording), implying clustering at each level and hence units are not independent; an assumption required for classical statistical tests (Petrie and Watson, 2004). As the data are observational, relationships of interest have the potential to be obscured by the confounding influence of other variables, or modified by intervening variables (Dohoo et al., 2009). The aims of statistical analyses in this thesis were to summarise relationships of relevance to the research questions, while accounting for lack of independence between observations, and confounding influences. Multilevel models with random effects for each level were therefore used for analyses (Goldstein, 2003; Rasbash et al., 2009), but compromise was required between presenting too complex models that represent the data well, or simpler models that are easier to explain to stakeholders but still useful (Gelman et al., 1996). The assessment of model fit, and usefulness was therefore an important aspect of the analyses.

1.4.1 Limitations of observational studies

The data used for this thesis are entirely observational. There are important limitations to consider for interpretation of results relating to the design of observational studies. Unlike in a randomised controlled trial, the exposure of interest in observational studies has not been randomly allocated between groups, and direct comparisons therefore cannot be made. This is due to the confounding influence of other variables related to the exposure and outcome of interest, meaning that exposed individuals may also differ in some other way to those that are unexposed (Dohoo et al., 2009). For example, the relationship between mastitis and milk yield can be confounded by cow parity, stage of lactation, and season. In other circumstances the relationship between the exposure and outcome of interest can be altered by another variable. In an additive model, an interaction is deemed to be present if the combined impact of 2 variables on the exposure is not equal to its sum. An example of an interaction would be if the impact of mastitis on milk yield was different in cows of different breed. Confounding can lead to biased results, and therefore appropriate adjustment is important. Interactions may be biologically important and these should be reported. Multivariate statistical models are required to adjust for multiple confounding variables and interactions, without loss of power. As a result of confounding and lack of control over exposures, observational studies do not provide strong evidence of a causal relationship between an exposure and the outcome of interest, and only ‘associations’ should be claimed.

1.4.2 Model outcomes and fit

The model outcomes in this thesis are either continuous (lifetime milk yield; chapters 3, 4, and 6), or discrete (disposal in a 50 day interval; chapter 5). Continuous outcomes can be modelled directly with linear models, and these are deemed to fit the data if residuals at all levels are distributed normally, with a mean of 0 (Goldstein, 2003; Rasbash et al., 2009). Discrete outcomes require a transformation to be modelled on a linear scale, for example the logit function in logistic regression, but the residuals are constrained as the outcome can only be 0 or 1. For a model fitted value of μ, residuals can only take the values $(1-\mu)$ or $(0-\mu)$, therefore assessment of model fit based on graphical inspection of these residuals is often inadequate (Green et al., 2009). A rational approach to model assessment is therefore to demonstrate that the model can predict biologically useful aspects of the observed data, and in particular that predictions related to the research question are reliable (Gelman et al., 1996), and this principle has been applied throughout the thesis. Model fit was assessed by comparing predictions to the observed data that was used for model development. To assess model usefulness, predictions were compared to observed data which was relevant to the research question but not used for model development. In order to assess whether the results could be generalised to other herds, cross validation was used, that is a comparison was made between predicted and observed data for herds that were not used for the estimation of model parameters.

1.4.3 Classical approach

In classical (frequentist) analyses as used in chapter 2, the probabilities obtained refer to the chance of obtaining similar results in repeated trials, and this is used to make decisions around accepting hypotheses. Likelihood functions (L) describe the probability of obtaining the data as a function of unknown parameters, given the hypothesis. With multiple unknowns in a linear model, the likelihood function is multi-dimensional (Myung, 2003) and computer algorithms are required for estimation. As likelihood values can be very small, the transformation to deviance (-2 x ln[L]) is used (Dohoo et al., 2009). The deviance should be minimised (to maximise likelihood), but a compromise may be required between model complexity and fit (Spiegelhalter et al., 2002). Importantly, probabilities from classical analyses do not refer to the model parameters directly, but to the likelihood of obtaining the same results if the trial was repeated. Classical probabilities therefore cannot be applied in a predictive sense for future trials, or to inform decisions (Berry and Stangl, 1996; Bolstad, 2007). With the Bayesian paradigm, the converse is true, and this methodology is applied in chapters 3 to 7.

1.4.4 Bayesian approach

In Bayesian analyses, prior knowledge is combined with the data obtained in a particular study to generate probability distributions for parameters (Spiegelhalter et al., 2004). These are termed posterior distributions, which represent the updated state of knowledge, and can be interpreted as the distribution of probability for particular outcomes, and hence prior knowledge for future trials (Berry and Stangl, 1996; Bolstad, 2007). This

is inherently useful for decision makers, as it gives information on how likely different outcomes are, based on a synthesis of all available evidence (Parmigiani, 2002). In this research there was no prior knowledge of parameters, and this was represented as flat prior distributions, with all values over a large range being equally likely. With vague prior distributions and a lot of data, the data has the major influence in the estimation of posterior distributions for parameters (Green et al., 2004), giving similar results to a frequentist analyses for linear models.

1.4.5 Markov chain Monte Carlo

For logistic models, likelihood methods can lead to bias in parameter estimates (Browne and Draper, 2006). Alternative algorithms are available, but these may lead to problems with convergence (Rasbash et al., 2009). One method to avoid this is Markov chain Monte Carlo (MCMC), which necessitates working in a Bayesian framework, and this was of particular importance in this research such that posterior distributions could be used directly for prediction. Therefore parameters for the logistic model of cow disposal (chapter 5) were estimated by MCMC using Gibbs sampling (Gilks et al., 1996). In this procedure, starting values for Markov chains are specified and each new value is generated by an algorithm that samples from a proposed conditional distribution given the current value. After a number of iterations to 'burn in', a Markov chain converges to a stationary distribution. Initial 'burn in' iterations are discarded leaving a probability distribution for the parameter of interest. Determining when a Markov chain has converged is controversial, and may require running several parallel chains or a very long chain (Gilks et al., 1996). Following convergence, parameter estimates at each iteration can be

used for onward prediction and simulation. Therefore, MCMC was also used for linear models in this thesis (chapters 3, 4, and 6).

1.4.6 Micro-simulation

The meaning of model parameters may not be intuitive. This can occur if herd level interpretation of a cow level model is required, or the parameters are on a non-linear scale. In these circumstances, micro-simulation can be used to demonstrate the impact of results in a context relevant to interpretation as a further aid to decision making (Parmigiani, 2002). The trajectory of individuals is modelled as if a carefully controlled trial were conducted, varying only the exposure of interest. This approach is useful when such a trial would be impossible or very expensive. Micro-simulation can involve either a 1-step or a 2-step procedure. The 2-step procedure is also described as probabilistic sensitivity analysis and involves summarising parameter distributions, often by assuming they are parametric, and act independently. Parameter distributions for a 2-step analysis can be obtained from a separate Bayesian analysis, previous research, or elicited from experts (O' Hagan et al., 2006). A 1-step micro-simulation procedure runs in parallel with a Bayesian analysis of the underlying data; following ‘burn in’ each parameter estimate is propagated forwards and used for prediction (Spiegelhalter et al., 2004). The 1-step procedure therefore does not make distributional assumptions about parameters, and any relationship between parameters is also maintained (Chessa et al., 1999). One-step micro-simulation is used in chapters 3 to 6 of this thesis to show the impact of model results in a useful context to aid interpretation. Chapter 7 extends the simulations from chapters 3 and 5 to

estimate rational budgets for specific herd level interventions to control heifer mastitis.

1.5 Aims of the thesis

1.5.1 Summary

The overall aim was to describe the prevalence of heifer mastitis based on SCC and assess the impact of SCC during the first lactation on the lifetime milk production of cows. Potential savings from increased milk sales through heifer mastitis control could then be estimated to give approximate budgets for the development of cost effective management interventions.

1.5.2 Descriptive data (chapter 2)

Chapter 2 presents descriptive data from the Irish, English, and Welsh herds used throughout the thesis, in particular the prevalence of heifers with high SCC through lactation. Having collated and assessed the available data two further questions arose: Firstly, to compare seasonal variation in cow SCC for Irish, English, and Welsh dairy herds. Secondly, to assess the association between herd size and cow SCC to evaluate the potential impact of trends for increased herd size on udder health.

1.5.3 Somatic cell count early in the first lactation and the lifetime milk yield of cows in Irish dairy herds (chapter 3)

The aims of chapter 3 were to assess the associations between SCC at 5 to 30 days in milk during parity 1 (SCC1) and lifetime milk yield, and also to first lactation milk yield for cows in Irish dairy herds.

1.5.4 Somatic cell count early in the first lactation and the cumulative milk yield of cows in English and Welsh dairy herds (chapter 4)

The aim of chapter 4 was to assess the association between SCC1 and cumulative milk yield over 2 years for cows in English and Welsh dairy herds.

1.5.5 Somatic cell count early in the first lactation and longevity of cows in Irish dairy herds (chapter 5)

The aim of chapter 5 was to assess the association between SCC1 and survival over a 5 year period for cows in Irish dairy herds.

1.5.6 Somatic cell count during the first lactation and the lifetime milk yield of cows in Irish dairy herds (chapter 6)

The aims of chapter 6 were firstly to compare associations between the exposures; SCC1 and SCC throughout the entire first lactation, on cumulative milk yield over both the first lactation and the subsequent lifetime of cows in Irish dairy herds. The second aim was to assess the association between SCC1 and SCC throughout the entire first lactation of cows in Irish dairy herds.

1.5.7 Bayesian evaluation of budgets for endemic disease control; an example using management changes to reduce somatic cell count early in the first lactation of cows in Irish dairy herds (chapter 7)

The aim of chapter 7 was to use 1-step Bayesian micro-simulation to synthesise evidence from previous research with the outcomes from chapters 3 and 5, to determine budgets for specific management interventions to control mastitis early in the first lactation of cows in Irish dairy herds under different circumstances.

Chapter 2: Association of season and herd size with somatic cell count for cows in Irish, English, and Welsh dairy herds

2.1 Introduction

For individual dairy producers, treatment costs, production losses and reduced sale value of high somatic cell count (SCC) milk are well known consequences of mastitis (Halasa et al., 2007). In the dairy processing industry, increased SCC is associated with both shortened shelf life of pasteurised milk (Santos et al., 2003), and reduced cheese yields (Barbano et al., 1991). Seasonal increase in bulk milk SCC (BMSCC) supplied to dairies has been reported from Ireland (Berry et al., 2006), and England and Wales (Green et al., 2006b), reducing the ability of these countries to meet demand for high quality milk products.

In general BMSCC tends to be highest in spring and summer, in countries where calving patterns are non-seasonal, such as England and Wales (Green et al., 2006b), Canada (Olde Riekerink et al., 2007; Sargeant et al., 1998), and Holland (Barkema et al., 1998a; Lievaart et al., 2007); possibly related to the influence of higher temperature and humidity on intramammary infection (IMI) risk (Morse et al., 1988). In Ireland however, BMSCC is generally lowest during April, and highest in November (Berry et al., 2006), probably because spring-calving predominates. BMSCC in Ireland is therefore lowest when most milk is produced but this may not reflect udder health, because cow level SCC dynamics associated with IMI may be masked by dilution (Green et al., 2006a). A key time for the occurrence of new infections

in Irish dairy herds may therefore be overlooked if monitoring strategies use only BMSCC.

Increasing herd size is common throughout the developed dairy industry worldwide; producers hope to benefit from economies of scale accrued from lower investments per cow, lower variable costs per unit of production and increased labour efficiency (Bailey et al., 1997). Larger herds in the United States of America have been reported to have lower cow level average SCC compared to smaller herds (Oleggini et al., 2001), however, larger Dutch herds have been reported to have higher BMSCC (Barkema et al., 1998a). In general, Irish, English, and Welsh dairy herds are increasing in size (DairyCo, 2012; ICBF, 2011) and it is important for these industries to evaluate the effect on SCC.

In addition to describing the datasets used throughout the thesis, the specific aims of this chapter were twofold: Firstly, to investigate the association between time of year and cow SCC, particularly in Irish dairy herds after accounting for stage of lactation. Secondly, to evaluate the association between herd size and cow SCC in Irish, English, and Welsh dairy herds, to evaluate the possible impact of expansion on SCC.

2.2 Materials and methods

2.2.1 Data selection

Data from 2005 to 2009, comprising 11,619,287 records from 964,612 cows in 8,095 Irish herds were provided by Irish Cattle Breeding Federation (ICBF; County Cork, Ireland), and restricted to remove implausible values (Table 2.1). For each herd year, the mean number of cows present per test day was determined (a proxy for herd size); herds with a mean of ≤ 10 cows were excluded. The minimum proportion of cows present per test day in each herd year had a distribution with distinct modes at 0.05, and 0.65. It was deemed that there were likely to be differences between recordings with a low minimum proportion of the herd present at a test date, compared to the majority (possibly associated with purchased cows), and 0.7% of herd test day recordings were excluded in which < 10% of the mean annual number of cows were present. For inclusion ≥ 4 herd test day recordings per year were required; 5% of herd years not meeting this criterion were excluded, leaving herd years with a median of 8 test days (interquartile range (IQR) 7 to 13). The cleaned dataset (Ire_dat) contained 10,181,545 recordings from 1,938,359 lactations in 860,563 cows, in 7,551 herds.

A second dataset was available for English and Welsh (UK) herds from 2004 to 2006, provided by National Milk Records (Chippenham, UK). Selection criteria for this dataset have been described in detail (Madouasse, 2009). Briefly, herd years with at least 10 test dates based on ≥ 20 cows were included, and those with factored data (daily milk yield and milk constituents extrapolated from a single sampling point) were removed. At least 80% of

cows were of Holstein or Friesian breeds. The data were limited (Table 2.1), and the final dataset (UK_dat) contained 6,772,182 records from 953,242 lactations in 474,669 cows in 2,128 herds.

Table 2.1. Selection criteria for the Irish, and English and Welsh datasets

Irish dataset			
Variable	Range before selection	Range after selection	Recordings removed (%)
Days in milk	-503 to 3,548	5 to 304	10
Parity	1 to 87	< 15	0.2
Test day milk yield (kg)	0.2 to 92.6	> 1 and < 71	0.003
Calving interval[1] (days)	-1,046 to 2,265	≥ 300	0.4
English and Welsh dataset			
Variable			
Days in milk	1 to 1,794	5 to 304	17
Parity	1 to 19	< 15	0.001
Test day milk yield (kg)	0.2 to 99.8	> 1 and < 71	0.003
Calving interval (days)	36 to 1,647	≥ 300	0.3

[1] For cows with more than 1 recorded calving date from subsequent parities.

2.2.2 Descriptive statistics

Not all variables were normally distributed, therefore median and interquartile range (IQR) were evaluated and reported for each. The number of cows (parity 1 and > 1) calving in each calendar month were determined. Herd level geometric means of test day SCC were calculated for cows by lactation month (1 to 10), and parity (1 and > 1, because lactation curve shape differed mostly between these groups). Herd level proportions of primiparous cows with SCC ≥ 200,000 cells/mL, and ≥ 400,000 cells/mL by lactation month were determined for comparison.

Random samples of 497 Irish, and 200 UK herds were selected from Ire_dat, and UK_dat respectively using R (R-Development-Core-Team. 2010),

and the corresponding records extracted. Sample sizes were selected to give the largest sub-datasets of Irish (Ire_dat^{SUB1}) and UK (UK_dat^{SUB1}) herds, with similar numbers of lines in each that could be handled with the available computing power. Ire_dat^{SUB1} contained 633,751 records from 122,707 lactations in 56,899 cows, and UK_dat^{SUB1} contained 635,346 records from 88,798 lactations in 43,943 cows. Actual BMSCC was not available for the herds of interest; therefore BMSCC over the study period was estimated from individual cow records using Ire_dat^{SUB1} and UK_dat^{SUB1}. For each calendar month j, in each herd k, BMSCC was approximated by the arithmetic mean of the yield corrected SCC from test day records i as;

$$BMSCC_{jk} \approx \sum (SCC_{ijk} \text{ (cells/mL)} \times TDY_{ijk} \text{ (mL)}) / \sum TDY_{ijk} \text{ (mL)},$$

where; $\sum$ = sum of, and TDY = test day milk yield.

Estimated BMSCC was compared graphically with the cow level data, both before and after adjustment for the confounding influence of stage of lactation, and milk yield in the following models.

2.2.3 Model development

Models including random effects, in addition to fixed effects, were used to account for a lack of independence due to clustering in the data. Models were constructed using Ire_dat^{SUB1} and UK_dat^{SUB1} separately; ln SCC at the test day level for individual cows was the outcome variable used, to ensure normality of residuals. The models took the form;

$$y_{ijkl} = \alpha + \mathbf{X_{ijkl}}\boldsymbol{\beta_1} + \mathbf{X_{jkl}}\boldsymbol{\beta_2} + \mathbf{X_{kl}}\boldsymbol{\beta_3} + \mathbf{X_l}\boldsymbol{\beta_4} + \mathbf{f_l} + v_{kl} + u_{jkl} + e_{ijkl}$$

$$\mathbf{f_l} \sim \text{MVN}(0, \textstyle\sum_{\mathbf{f}})$$

$v_{kl} \sim N(0, \sigma^2_v)$

$u_{jkl} \sim N(0, \sigma^2_u)$

$e_{ijkl} \sim N(0, \sigma^2_e)$

where y_{ijkl} = ln SCC at test day i, in parity j, for cow k, in herd l, α = intercept value, $\mathbf{X_{ijkl}}$ = matrix of test day variables, $\boldsymbol{\beta_1}$ = vector of coefficients for $\mathbf{X_{ijkl}}$, $\mathbf{X_{jkl}}$ = matrix of parity variables, $\boldsymbol{\beta_2}$ = vector of coefficients for $\mathbf{X_{jkl}}$, $\mathbf{X_{kl}}$ = matrix of cow variables, $\boldsymbol{\beta_3}$ = vector of coefficients for $\mathbf{X_{kl}}$, $\mathbf{X_l}$ = matrix of herd variables (including polynomials of herd size), $\boldsymbol{\beta_4}$ = vector of coefficients for $\mathbf{X_l}$, $\mathbf{f_l}$ = matrix of random effects to account for herd level variation in α, and fixed effect coefficients for calendar month (multivariate normal distribution with mean = 0 and covariance matrix $\sum_{\mathbf{f}}$), v_{kl} = random effect to account for variation between cows (normal distribution with mean 0 and variance σ^2_v), u_{jkl} = random effect to account for variation between parities (normal distribution with mean 0 and variance σ^2_u), and e_{ijkl} = residual level 1 error (normal distribution with mean 0 and variance σ^2_e). Model parameters were estimated by the iterative generalised least squares procedure (Goldstein, 2003), using MLwiN 2.22 (Rasbash et al., 2009).

Categorical variables were constructed for year, calendar month, and parity (1 to 5+). To account for dilution of SCC with increased test day milk yield (TDY) on a linear scale, and reduced TDY with increased SCC due to IMI on an exponential scale (Green et al., 2006a), ln TDY, and ln ln TDY were included, as the outcome of the models was ln SCC. Stage of lactation was included as days in milk (DIM) + $e^{-0.065 \text{ X DIM}}$ (Silvestre et al., 2006). Biologically plausible interactions, and herd level variation in fixed effects

were assessed. Variables remained in the model if the mean value of coefficients was > twice the standard error ($p \leq 0.05$), and their inclusion resulted in a decrease in the deviance. Intra-class correlation coefficients (ICC) for the unexplained variance at each level of the model were calculated (Dohoo et al., 2009).

2.2.4 Assessment of model fit

To assess model fit, distributions of standardised residuals at the herd, cow, parity, and recording level were examined for normality. Further checking used within model predictions; fixed effects were applied to each line of Ire_dat^{SUB1} and UK_dat^{SUB1} to predict ln SCC. Predictions were compared graphically to observed data, and correlation was assessed (r^2; (Petrie and Watson, 2004)). Equations for regression lines between observed and predicted values were estimated.

To further assess model fit and usefulness, cross validation was carried out in two further random samples of 493 different Irish, and 200 different UK herds taken from Ire_dat and UK_dat respectively. The second Irish sub-dataset (Ire_dat^{SUB2}) contained 678,950 records from 125,493 lactations in 56,902 cows, and the second UK sub-dataset (UK_dat^{SUB2}) contained 613,072 records from 86,036 lactations in 42,539 cows. Fixed effects from the respective model were used to predict ln SCC for every line of Ire_dat^{SUB2}, and UK_dat^{SUB2} using Microsoft Excel (2007). Comparisons with the observed data were repeated. Shrinkage of r^2 on cross validation (Dohoo et al., 2009) was assessed to determine if the models could be generalised to other herds, not involved in parameter estimation.

2.3 Results

2.3.1 Descriptive statistics

Summaries of TDY, test day fat proportion (TDF), test day protein proportion (TDP), SCC, and herd size are presented in Table 2.2. In Ire_dat, 25, 50 and 25% of recordings were from cows in parities 1, 2 to 4, and ≥ 5 respectively. In UK_dat, 22, 53, and 25% of recordings were from cows in parities 1, 2 to 4, and ≥ 5 respectively. Calving patterns also differed (Figure 2.1); 59 and 56% of parity 1 and parity 2+ cows' calving dates were from January to March in Ire_dat. In UK_dat, 64 and 58% of parity 1 and parity 2+ cows' calving dates were from July to December.

Table 2.2. Descriptive results for the selected Irish dataset (Ire_dat), and the selected English and Welsh dataset (UK_dat)

Irish dataset:

Variable	Lower quartile	Median	Upper quartile
Test day milk yield	17 kg	22 kg	28 kg
Test day fat proportion	0.034	0.038	0.043
Test day protein proportion	0.032	0.034	0.036
Test day somatic cell count	55,000 cells/mL	110,000 cells/mL	243,000 cells/mL
Mean herd size (cows)	46	71	81

English and Welsh dataset:

Variable			
Test day milk yield	21 kg	27 kg	33 kg
Test day fat proportion	0.034	0.039	0.043
Test day protein proportion	0.030	0.032	0.034
Test day somatic cell count	37,000 cells/mL	74,000 cells/mL	173,000 cells/mL
Mean herd size (cows)	101	139	189

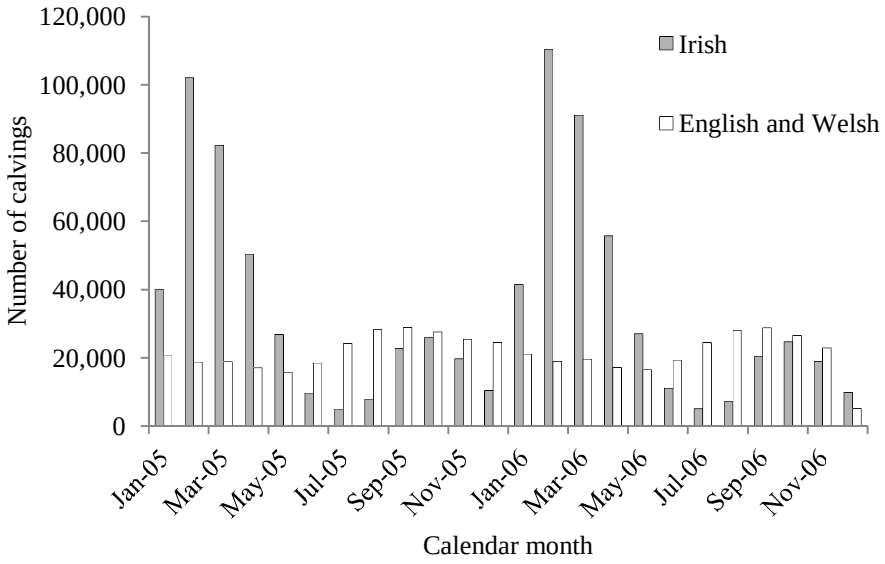

Figure 2.1. Number of cows calving per month during 2005 and 2006 for 7,551 Irish and 2,128 English and Welsh dairy herds

2.3.1.1 Herd level descriptive statistics

The median herds' geometric means of cow SCC for primiparous and multiparous cows by month of lactation are shown in Table 2.3, with the full distributions in Figure 2.2. For comparison herd level proportions of primiparous cows with SCC ≥ 200,000 cells/mL and ≥ 400,000 cells/mL by month of lactation for the median herd are shown in Table 2.4, with the full distributions in Figure 2.3.

Table 2.3. Geometric mean cow level somatic cell count (cells/mL) by month of lactation for the median herd from 7,551 Irish and 2,128 English and Welsh dairy herds

Month of lactation	Irish dataset		English and Welsh dataset	
	Parity 1	Parity > 1	Parity 1	Parity > 1
1	104,000	101,000	75,000	75,000
2	75,000	93,000	50,000	60,000
3	77,000	106,000	50,000	67,000
4	83,000	121,000	54,000	76,000
5	89,000	137,000	57,000	86,000
6	96,000	154,000	59,000	96,000
7	102,000	173,000	61,000	107,000
8	112,000	196,000	65,000	121,000
9	122,000	224,000	69,000	137,000
10	127,000	245,000	74,000	158,000

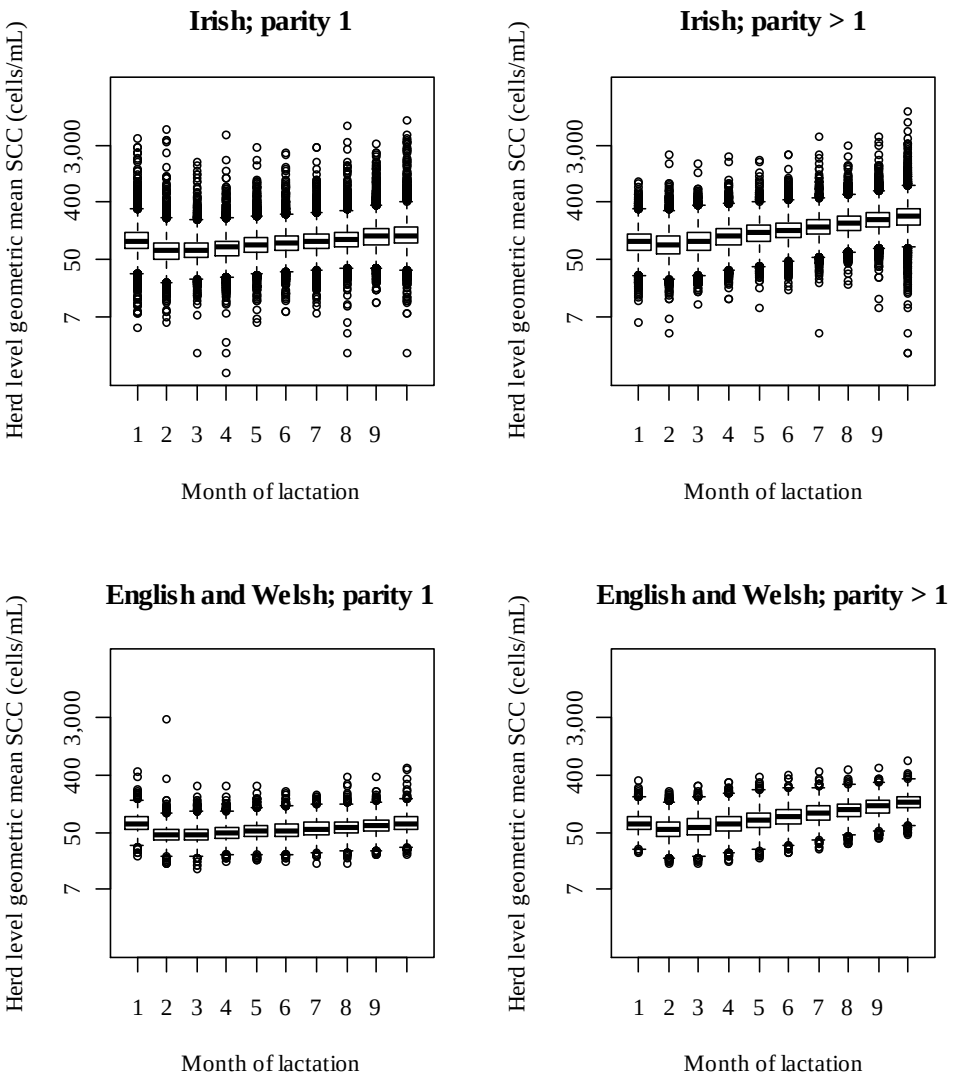

Figure 2.2. Distributions[2] of herd level geometric mean test day somatic cell count (SCC), for primiparous and multiparous cows, by month of lactation for 7,551 Irish and 2,128 English and Welsh dairy herds

[2] For each month; the median herd is the horizontal black line, the surrounding boxes contain data for 50% of herds, the attached whiskers extend to 1.5 times the interquartile range (95% of the data), and outliers are marked by circles.

Table 2.4. Proportions of primiparous cows with high somatic cell count at different thresholds by month of lactation for the median herd from 7,551 Irish and 2,128 English and Welsh dairy herds

Month of lactation	Irish dataset		English and Welsh dataset	
	≥ 200,000 cells/mL	≥ 400,000 cells/mL	≥ 200,000 cells/mL	≥ 400,000 cells/mL
1	0.21	0.11	0.16	0.09
2	0.13	0.06	0.10	0.05
3	0.13	0.06	0.09	0.04
4	0.14	0.06	0.10	0.04
5	0.16	0.06	0.10	0.04
6	0.18	0.06	0.10	0.04
7	0.19	0.06	0.11	0.04
8	0.21	0.07	0.11	0.04
9	0.24	0.07	0.12	0.04
10	0.26	0.07	0.15	0.05

2.3.1.2 Estimated herd bulk milk somatic cell count

Distributions of calculated herd level BMSCC by calendar month, based on sub-datasets; Ire_dat^{SUB1} and UK_dat^{SUB1} are shown in Figure 2.4. For the Irish herds, geometric mean BMSCC was lowest in April (223,000 cells/mL), and highest in November and December (314,000 cells/mL). For the UK herds, geometric mean BMSCC was lowest in January (176,000 cells/mL) and highest in August (205,000 cells/mL).

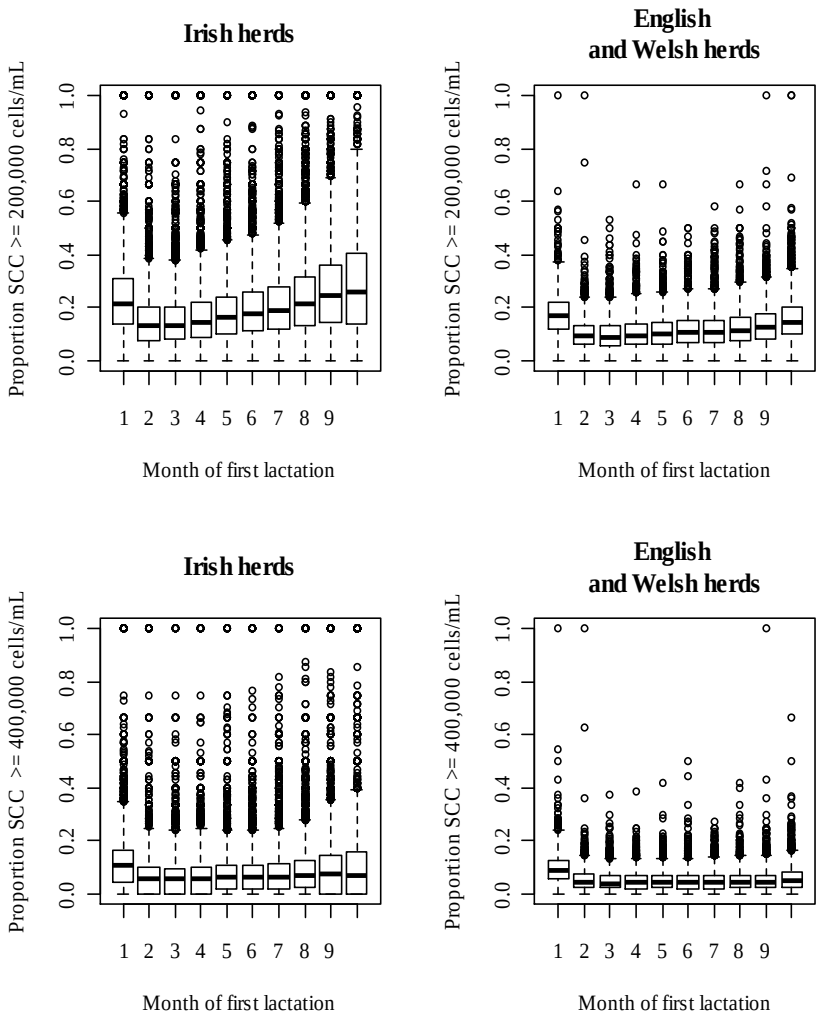

Figure 2.3. Distributions[3] of herd level proportions of primiparous cows with test day somatic cell count (SCC) ≥ 200,000 cells/mL, and ≥ 400,000 cells/mL by month of lactation for 7,551 Irish and 2,128 English and Welsh dairy herds

[3] For each month; the median herd is the horizontal black line, the surrounding boxes contain data for 50% of herds, the attached whiskers extend to 1.5 times the interquartile range (95% of the data), and outliers are marked by circles.

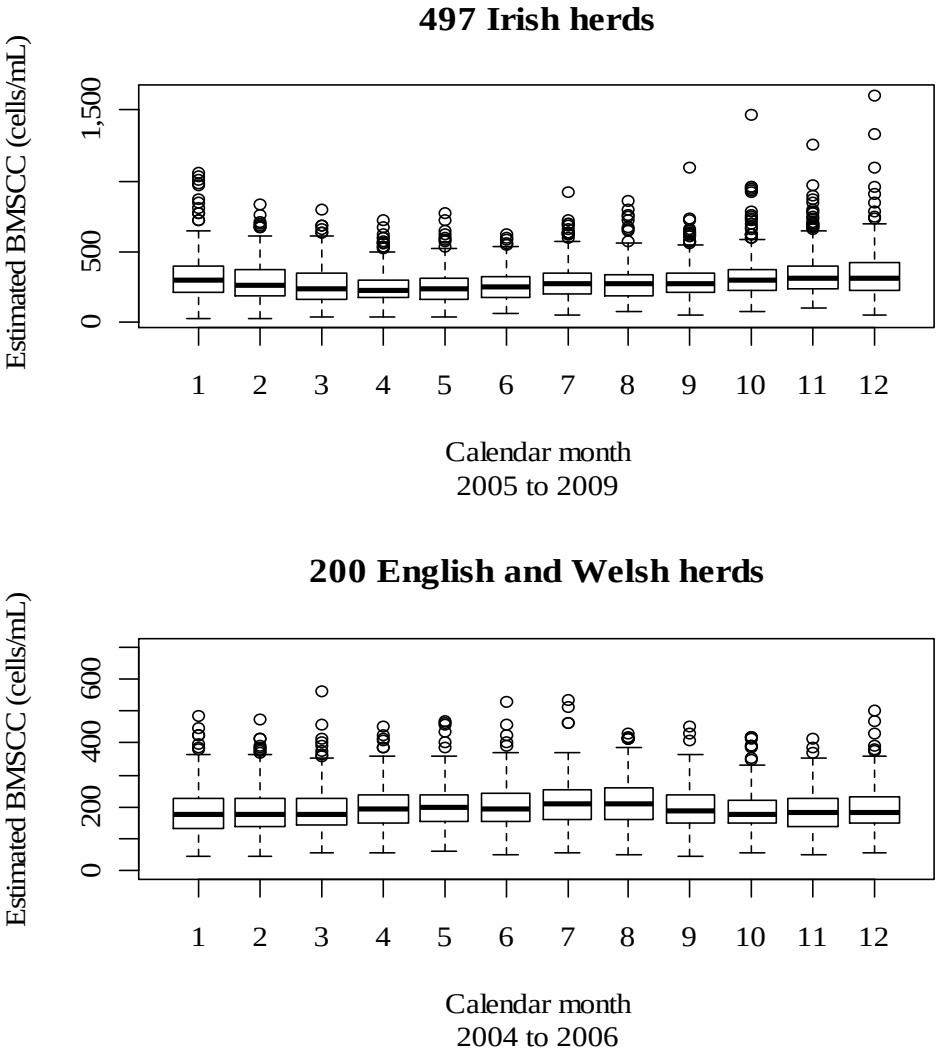

Figure 2.4. Herd level distributions of bulk milk somatic (BMSCC)[4] by calendar month[5] for 497 Irish and 200 English and Welsh dairy herds

[4] Estimated from test day milk yield and somatic cell count data.
[5] Where 1 = January, 2 = February, 3 = March, 4 = April, 5 = May, 6 = June, 7 = July, 8 = August, 9 = September, 10 = October, 11 = November, and 12 = December.

2.3.2 Model results

2.3.2.1 Association between season and somatic cell count

Table 2.5 shows the fixed effect coefficients in the final models for ln SCC, developed from Ire_dat^{SUB1} and UK_dat^{SUB1}. Having accounted for stage of lactation and TDY, October was associated with lowest ln SCC in both models and was set as the reference. Calendar month interacted with stage of lactation and parity. For baseline cows (parity 2, mean TDY, TDF, and TDP, and in herds of mean size) in Irish herds (Figure 2.5), geometric mean SCC was highest from February to August, independent of stage of lactation; for cows that were 100 DIM, geometric mean SCC peaked at 111,000 cells/mL (95% confidence interval (CI); 92,000 to 133,000) during May, and was 61,000 cells/mL (95% CI; 56,000 to 66,000) in October. For baseline cows in UK herds (Figure 2.5), geometric mean SCC was highest from January to June; for cows that were 100 DIM, geometric mean SCC was highest during February and June, at 84,000 cells/mL (95% CI; 71,000 to 100,000), and was 66,000 cells/mL (95% CI; 60,000 to 72,000) in October. Random effects and ICC from the models (Table 2.6), show additional herd level variance in ln SCC from February to August; this was larger for the Irish than the UK herds. As a result, less total variance in ln SCC in the null model (Table 2.7) was explained by the fixed effects in the Irish model from February to August (11 to 13%), compared to September to January (16%). For the UK model, 11 to 13% of the total variance in ln SCC in the null model was explained by the fixed effects all year round.

Table 2.5. Final models of repeated ln[6] SCC[7] ('000 cells/mL) within cow parity, from 497 and 200 randomly selected herds from Ireland and England and Wales respectively; fixed effects

	Irish model		English and Welsh model	
Fixed effects (baseline)	Mean	Standard error[8]	Mean	Standard error
Intercept	4.146	0.040	4.119	0.036
Year (2005)				
2004	NA[9]	NA	-0.055	0.004
2006	0.004	0.006	0.027	0.004
2007	-0.038	0.006	NA	NA
2008	-0.105	0.007	NA	NA
2009	-0.020	0.007	NA	NA
ln TDY[10] (mean)[11]	-0.965	0.026	-1.396	0.034
ln ln TDY (mean)[14]	0.762	0.068	1.650	0.096
ln TDF[12] (mean)	0.444	0.008	0.351	0.008
ln TDP[13] (mean)	1.124	0.017	1.477	0.019
DIM[14] (5)	-0.0004	0.00001	0.0007	0.0001
$e^{(-0.065 \times DIM)}$ (5 DIM)	0.055	0.060	0.188	0.041
Month of recording (October)				
January	0.308	0.047	0.282	0.032
February	0.481	0.042	0.312	0.034
March	0.483	0.040	0.237	0.034
April	0.495	0.041	0.242	0.036
May	0.524	0.043	0.149	0.037
June	0.549	0.049	0.235	0.036
July	0.494	0.059	0.168	0.033
August	0.463	0.068	0.125	0.031
September	0.122	0.056	0.058	0.029
November	0.126	0.049	0.173	0.030
December	0.422	0.054	0.200	0.032
Parity (2)				
1	0.320	0.020	0.220	0.021
3	0.082	0.022	0.150	0.022
4	0.266	0.024	0.307	0.024
5+	0.514	0.020	0.533	0.021

[6] Natural logarithm.
[7] Milk somatic cell count.
[8] Coefficients are significant at the 5% level if the mean effect > twice the standard error.
[9] Not applicable.
[10] Test day milk yield (kg).
[11] Baseline = mean value in respective dataset.
[12] Test day fat proportion.
[13] Test day protein proportion.
[14] Days in milk.

Table 2.5 continued

	Irish model		English and Welsh model	
Fixed effects (baseline)	Mean	Standard error	Mean	Standard error
Size (mean)				
$(Size)^1$	0.00007	0.0001	0.0005	0.0002
$(Size)^2$	0.000003	0.000001	0.000004	0.000001
$(Size)^3$	-0.000000007	0.000000004	NA	NA
Month of recording and DIM (October, 5 DIM)				
January	-0.0002	0.0001	-0.0007	0.00008
February	0.0002	0.0001	-0.0007	0.00008
March	0.0008	0.0001	-0.0005	0.00008
April	0.0011	0.0001	-0.0003	0.00008
May	0.0008	0.0001	-0.000004	0.00008
June	0.0004	0.0001	0.00003	0.00008
July	0.00002	0.0001	-0.0001	0.00008
August	-0.0003	0.0001	-0.00005	0.00007
September	-0.0001	0.0001	0.0001	0.00007
November	0.0002	0.0001	-0.0003	0.00007
December	-0.00002	0.0001	-0.0006	0.00007
Month of recording and $e^{DIM \times -0.065}$ (October, 5 DIM)				
January	0.290	0.079	0.226	0.053
February	0.574	0.070	0.219	0.054
March	0.709	0.065	0.129	0.054
April	0.760	0.064	0.087	0.056
May	0.756	0.068	-0.051	0.058
June	0.671	0.076	0.081	0.057
July	0.514	0.090	-0.062	0.054
August	0.485	0.102	-0.071	0.052
September	0.076	0.089	0.007	0.049
November	0.229	0.081	0.219	0.049
December	0.507	0.090	0.149	0.053

Table 2.5 continued

Fixed effects (baseline)	Irish model Mean	Irish model Standard error	English and Welsh model Mean	English and Welsh model Standard error
Parity and DIM (Parity 2, 5 DIM)				
1	-0.0011	0.00004	-0.0011	0.00004
3	0.0003	0.00005	0.0004	0.00005
4	0.0002	0.00005	0.0003	0.00005
5+	-0.0002	0.00004	0.0002	0.00004
Parity and $e^{DIM \times -0.065}$ (Parity 2, 5 DIM)				
1	0.525	0.031	0.400	0.032
3	-0.147	0.034	-0.079	0.034
4	-0.198	0.037	-0.185	0.037
5+	-0.355	0.031	-0.201	0.031
Deviance	1,646,471		1,647,317	

Table 2.6. Final models of repeated ln[15] SCC[16] ('000 cells/mL) within cow parity, from 497 and 200 randomly selected herds from Ireland and England and Wales respectively; random effects

	Level	Variance	Standard error	ICC[17]
Irish model	Herd	$\sum_{f1}$	$\sum_{f1}$	0.08
	Cow	0.256	0.003	0.21
	Parity	0.296	0.002	0.24
	Recording	0.570	0.001	0.47
English and Welsh model	Herd	$\sum_{f2}$	$\sum_{f2}$	0.08
	Cow	0.289	0.004	0.22
	Parity	0.351	0.003	0.26
	Recording	0.592	0.001	0.44

[15] Natural logarithm.
[16] Milk somatic cell count.
[17] Intra-class correlation coefficient = proportion of unexplained variance at each level from September to January.

Table 2.6 continued

$\sum_{f1}$ = Herd level (co)variance matrix for the Irish model (standard error)								
Intercept	0.095							
	(0.0066)							
February	-0.020	0.082						
	(0.0056)	(0.0080)						
March	-0.015	0.046	0.072					
	(0.0046)	(0.0056)	(0.0060)					
April	-0.014	0.050	0.054	0.096				
	(0.0041)	(0.0061)	(0.0054)	(0.0071)				
May	-0.015	0.038	0.041	0.054	0.066			
	(0.0041)	(0.0050)	(0.0044)	(0.0050)	(0.0050)			
June	-0.0004	0.028	0.037	0.049	0.040	0.054		
	(0.0037)	(0.0045)	(0.0040)	(0.0045)	(0.0038)	(0.0041)		
July	0.0031	0.017	0.024	0.033	0.031	0.030	0.044	
	(0.0034)	(0.0041)	(0.0035)	(0.0039)	(0.0034)	(0.0031)	(0.0035)	
August	0.0027	0.017	0.018	0.032	0.029	0.029	0.022	0.042
	(0.0034)	(0.0040)	(0.0033)	(0.0038)	(0.0032)	(0.0030)	(0.0027)	(0.0034)

Table 2.6 continued

$\sum_{f2}$ = Herd level (co)variance matrix for the English and Welsh model (standard error)								
Intercept	0.11 (0.011)							
February	0.00039	0.013						
	(0.0030)	(0.0016)						
March	-0.0015	0.0073	0.013					
	(0.0031)	(0.0013)	(0.0016)					
April	-0.0084	0.0048	0.0083	0.021				
	(0.0038)	(0.0014)	(0.0016)	(0.0025)				
May	-0.0094	0.0018	0.0048	0.0077	0.017			
	(0.0035)	(0.0013)	(0.0014)	(0.0017)	(0.0021)			
June	-0.0089	0.00069	0.0061	0.0092	0.013	0.02		
	(0.0037)	(0.0014)	(0.0014)	(0.0018)	(0.0018)	(0.0024)		
July	-0.012	0.00011	0.0038	0.0064	0.0092	0.014	0.022	
	(0.0039)	(0.0014)	(0.0015)	(0.0018)	(0.0018)	(0.0020)	(0.0025)	
August	-0.0067	-0.0032	0.00085	0.0052	0.0068	0.012	0.012	0.019
	(0.0036)	(0.0014)	(0.0014)	(0.0017)	(0.0016)	(0.0019)	(0.0019)	(0.0022)

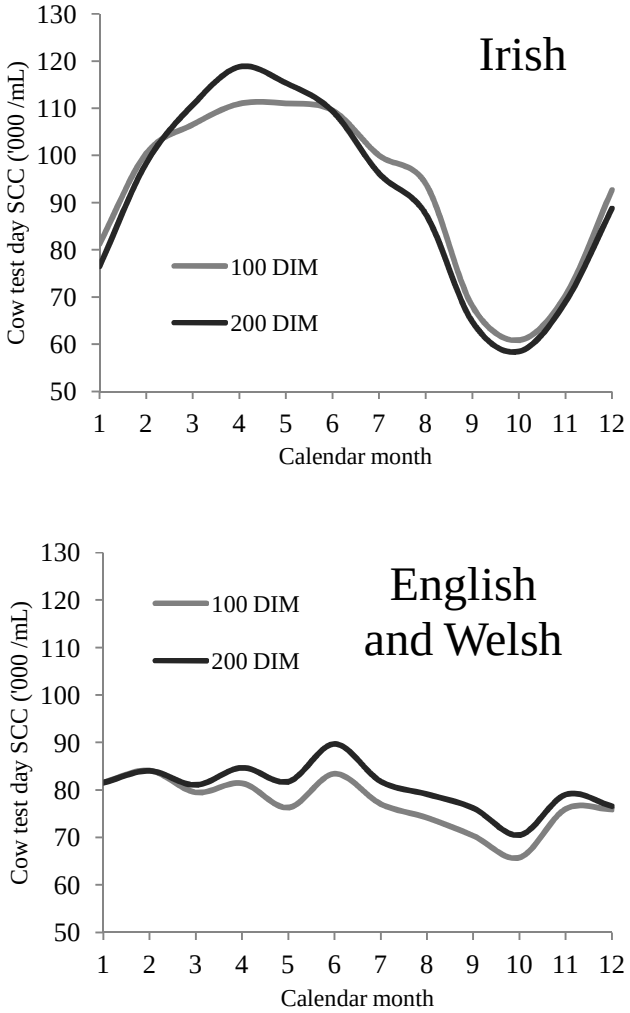

Figure 2.5. Model predictions for the impact of calendar month[18] on cow level geometric mean test day somatic cell count (SCC)[19] ('000 cells /mL) for cows at 100 and 200 days in milk (DIM) in Irish, English, and Welsh dairy herds

[18] Where 1 = January, 2 = February, 3 = March, 4 = April, 5 = May, 6 = June, 7 = July, 8 = August, 9 = September, 10 = October, 11 = November, and 12 = December.

[19] Refers to parity 2 cows in 2005 with mean test day milk yield (Irish herds; 21 kg, English and Welsh herds; 27 kg), and fat (3.8%) and protein proportions (Irish herds; 3.4%, English and Welsh herds; 3.2%), in herds of mean size for Ireland (96 cows), and England and Wales (196 cows).

Table 2.7. Random effects from the null models of repeated ln[20] SCC[21] ('000 cells/mL) within cow parity, from 497 and 200 randomly selected herds from Ireland and England and Wales respectively

Null model	Level	Variance	Standard error	ICC[22]
Irish (deviance = 1,813,845)	Herd	0.107	0.007	0.074
	Cow	0.295	0.003	0.204
	Parity	0.313	0.002	0.217
	Recording	0.730	0.001	0.505
	Totals	1.445		1.000
English and Welsh (deviance = 1,771,367)	Herd	0.108	0.011	0.070
	Cow	0.321	0.004	0.208
	Parity	0.384	0.003	0.248
	Recording	0.732	0.001	0.474
	Totals	1.545		1.000

[20] Natural logarithm.
[21] Milk somatic cell count.
[22] Intra-class correlation coefficient; proportion of the variance at each level.

2.3.2.2 Association between herd size and somatic cell count

Following adjustment for confounding influences, there was a non-linear relationship between herd size and test day SCC, included in the final Irish and UK models as 3rd and 2nd degree polynomials respectively (Figure 2.6). For herd sizes of up to 130 cows, test day SCC for baseline cows (parity 2, 5 DIM, recorded in October with mean TDY, TDF, and TDP) in Irish herds remained at 63,000 cells/mL (95% CI; 59,000 to 68,000). Further increase in herd size was associated with non-linear increase in test day SCC; reaching 68,000 cells/mL (95% CI; 59,000 to 89,000) with a herd size of 300 cows. In UK herds, test day SCC decreased for baseline cows in herds of up to 130 cows; reaching 60,000 cells/mL (95% CI; 57,000 to 65,000), and this was maintained in herd sizes up to 180 cows. For larger herds, test day SCC increased with increasing size at a higher rate than for the Irish herds; also reaching 68,000 cells/mL (95% CI; 59,000 to 77,000) with a herd size of 300 cows. For the

Irish herds, there was more uncertainty in these estimates that increased with increasing herd size from 130 cows, due to relatively few larger herds compared to the UK dataset. For the UK herds, uncertainty in the estimates, increased with increasing herd size, particularly for > 230 cows.

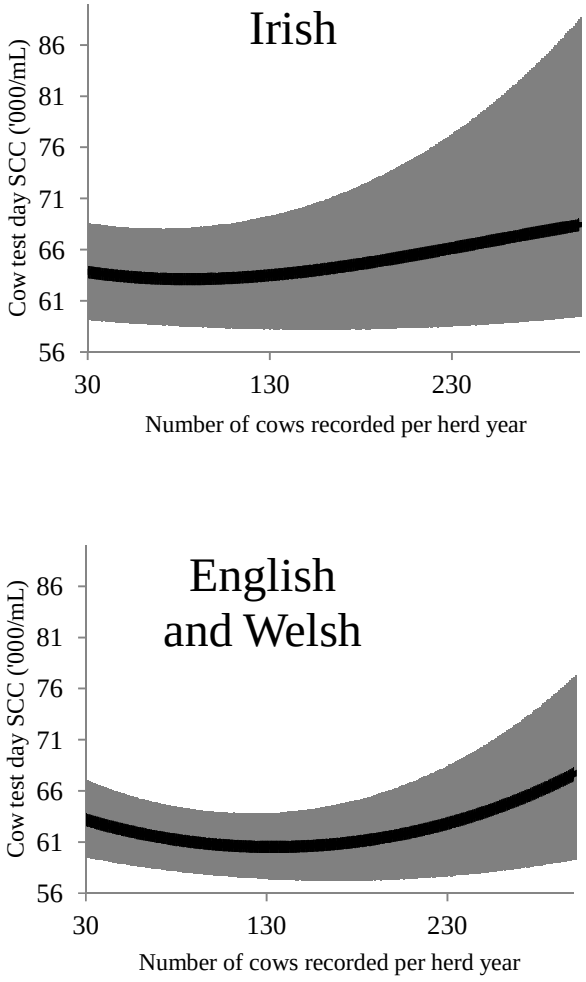

Figure 2.6. Model predictions for the impact of herd size on cow level geometric mean test day somatic cell count (SCC)[23] ('000 cells /mL) with 95% confidence interval for Irish, English and Welsh dairy herds

[23] Refers to parity 2 cows in October 2005 that are 5 days in milk, and have mean test day milk yield (Irish herds; 21 kg, English and Welsh herds; 27 kg), and fat (3.8%) and protein proportions (Irish herds; 3.4%, English and Welsh herds; 3.2%), truncated at 400 cows per herd.

2.3.3 Model fit

For Ire_dat^{SUB1} and UK_dat^{SUB1}, standardised residuals were distributed approximately normally at all levels, suggesting good model fit. For the Irish and UK within model predictions, lines of best fit between predicted and observed ln SCC had intercepts of 0.6 and 0.9 respectively, both with slopes of 0.8 (r^2 = 0.14 (Irish) and 0.12 (UK)). For Ire_dat^{SUB2} and UK_dat^{SUB2}, lines of best fit between predicted and observed ln SCC had intercepts of 0.6 and 1.2, and slopes of 0.9 and 0.7 (r^2 = 0.14 (Irish) and 0.12 (UK)), indicating zero shrinkage on cross validation, suggesting that the model results can be generalised to herds not involved in parameter estimation (Dohoo et al., 2009). However, the models were not good at predicting extremes of SCC in either sample datasets, resulting in low r^2 values.

2.4 Discussion

2.4.1 Association between season and somatic cell count

The association between calendar month and cow SCC was of particular interest in the Irish dataset. When confounding by stage of lactation and TDY were removed, the underlying values of cow SCC were highest, and most variable from February to August, despite BMSCC being at its lowest at this time in Irish herds. Although the number of years studied was limited, seasonal patterns in SCC dynamics for both datasets were consistent with previous observations (Green et al., 2006b; Lievaart et al., 2007; Olde Riekerink et al., 2007), with underlying cow SCC being increased, and more variable during spring and summer. In addition to an association with high SCC, infection

status is reported to be the most important factor influencing SCC variance (Schepers et al., 1997). Having adjusted for other confounding factors, unexplained variation in SCC is therefore most likely to be attributable to increased new IMI rate (resulting in low r^2 values). Inclusion of herd level random coefficients between February and August, demonstrated additional unexplained variation in cow SCC that was herd specific, and this suggests that there is important between herd variation in the rates of new IMI and cures during these months. Monitoring new IMI rate using SCC thresholds is recommended, so control measures can be applied and adapted as necessary (Bradley and Green, 2005). It thus appears important to characterise differences in rates of new IMI between Irish herds so achievable targets, based on individual cow SCC can be used to improve udder health management.

2.4.2 Association between herd size and somatic cell count

In general, increase in herd size was associated with increased cow SCC, although the rate of increase differed between the Irish, and English and Welsh herds studied. This suggests more attention is required to optimise udder health management when herds increase cow numbers. These findings contrast with the previously observed lower average SCC with increasing herd size in a dataset with a higher frequency of larger herds (Oleggini et al., 2001), but are consistent with Dutch experience (Barkema et al., 1998a). For typical ranges of Irish, English, and Welsh herd sizes, these results suggest that expansion may be associated more with penalties, and loss of efficiency, than economic advantage in terms of SCC. The size of this effect on geometric mean cow SCC was small, and uncertainty increased with herd size, however the 95% CI

indicate that for Irish herds, increased herd size was more likely associated with higher, than lower cow SCC (Figure 2.6). Risk of transmission of udder pathogens during milking may increase with herd size, as more susceptible quarters could be exposed. Poor management of higher pasture stocking rates in larger herds could contribute to increased risk of *Streptococcus uberis* IMI (Lopez-Benavides et al., 2009). Capital investments in improved facilities requires a critical herd size such that the fixed cost per cow is acceptable; many Irish, English, and Welsh herds may not have reached this point. More labour units are required by larger herds, although the number of labour units per cow is less, emphasising the importance of farm staff developing expertise in cow management.

2.5 Conclusion

The Irish herds contained predominantly spring-calving cows, typically with lower milk yield and higher SCC, compared to cows in the year-round calving English and Welsh herds. After correcting for stage of lactation and milk yield, SCC for cows in Irish, English and Welsh dairy herds was higher and more variable in spring and summer, than autumn and winter. For Irish dairy herds, monitoring individual cows is particularly important in spring and summer, despite low BMSCC and farmers should not be complacent about udder health at this time. Increasing herd size was associated with a non-linear increase in cow SCC in these countries, highlighting an important area that may influence cost effective dairy herd expansion.

Chapter 3: Association between somatic cell count early in the first lactation and the lifetime milk yield of cows in Irish dairy herds

3.1 Introduction

Mastitis in primiparous cows early in their first lactation has been highlighted as a common problem that is economically important through its impact on their future productivity (De Vliegher et al., 2012; Piepers et al., 2009), which limits their ability to achieve genetic potential for milk yield. Increased milk somatic cell count early in the first lactation (SCCel) has been associated with decreased milk yield throughout the entire first lactation (Coffey et al., 1986; De Vliegher et al., 2005a). This loss has been estimated in Belgian primiparous cows at 0.13 kg/d for every unit increase in ln transformed somatic cell count (SCC) measured between 5 and 14 days in milk (De Vliegher et al., 2005a). The relationship between SCCel and cumulative milk yield in subsequent lactations is less clear. Coffey et al. (1986) reported that for cows in Virginia (United States of America), mean first lactation milk yield (FLMY) decreased with increasing SCCel, and was 6,452 kg, 6,050 kg, and 5,696 kg for groups of cows with SCCel < 100,000 cells/mL, 100,000 to 400,000 cells/mL, and > 400,000 cells/mL respectively. However, over subsequent lactations beyond the first this trend did not continue, and mean lactation milk yields were 6,840 kg, 7,241 kg, and 7,163 kg respectively for the same groups (Coffey et al., 1986). This study did not control for clustering of cows in different herds, any potential confounding variables, or importantly, how long cows survived. The impact of SCCel on lifetime milk yield (LiMY)

has not otherwise been considered. This is an important omission, as it may not be until cows reach their second lactation, that sufficient milk is produced to break even on rearing costs, and the true cost of milk loss may extend further than the first lactation. For example, under Irish conditions the cost of rearing to the point of calving is approximately €1,451 /heifer (Kennedy et al., 2011). Therefore with an average margin over variable costs of €0.17 /kg (Hennessy et al., 2011), 8,535 kg of saleable milk is required to break even, which likely requires > 1 lactation. Furthermore, primiparous cows have yet to achieve mature adult weight and size. Hence, lactation milk yield and financial returns increase in subsequent lactations (Madouasse, 2009). Considering impact on LiMY is therefore important to evaluate the total cost of SCCel, and aid decision making around mastitis control measures for *pre-* and *peri-partum* (ppp) heifers.

The aims of this chapter were to assess the associations between SCC at 5 to 30 days in milk during parity 1 (SCC1) and lifetime milk yield, and also first lactation milk yield for cows in Irish dairy herds. A Bayesian approach was taken, and posterior predictions were used to evaluate the economic impact of the results on meaningful, intuitive scales, and for particular herd scenarios.

3.2 Materials and methods

3.2.1 Data selection

Herds were selected for the analysis using the criteria specified in chapter 2. To be eligible for inclusion, cows required a first SCC recording between 5 and 30 days in milk (DIM) during parity 1. There were 233,176 eligible cows in 7,423 herds (DAT); 893 of these cows had more than 1 record between 5 and 30 DIM during parity 1, and SCC at the first of these was taken as SCC1. A sub-dataset of production records from 25% of cows in DAT, with a record of SCC1 between January 2005 and March 2007, and with dates of birth available was then created. Cows with age at first calving (AFC) < 700 days were deemed at increased risk of culling because of dystocia (Berry and Cromie, 2009), and individual cows with AFC < 700 days (6% of the total population) were discarded to remove this effect. For the selected cows, cumulative milk yields for all lactations up to July 25, 2012 were determined based on a published method (Olori et al., 1999), and provided by ICBF. These were summed to give an estimate of 'lifetime milk yield' for each cow over follow up times from 5.3 to 7.5 years, based on the time from the first calving to the end of the study for each cow. The selected dataset included records from 53,652 cows in 5,922 herds. Random samples of 2,500 (samp_1), and 3,422 (samp_2) of these herds were selected using R (R-Development-Core-Team, 2010) and the data for all 22,023 and 31,629 eligible cows in samp_1 and samp_2 respectively were collated. The statistical models were fitted to samp_1, and samp_2 was used for cross validation; sample sizes were determined based on the computational constraints imposed by these

procedures. Median, and interquartile range (IQR) for LiMY, first lactation milk yield (FLMY), proportion of cows surviving lactations 1 and 4, and SCC1 were determined for each sample, stratified by sub-groups based on SCC1 (SCC1_gp; group 1; < 55,000 cells/mL, group 2; 55,000 to 149,000 cells/mL, group 3; 150,000 to 400,000 cells/mL, group 4; ≥ 400,000 cells/mL).

3.2.2 Statistical analysis

The outcomes used were lifetime or first lactation milk yield (LiMY or FLMY; y_{ij}), for the ith cow, in the jth herd. The models developed for samp_1, took the form;

$$y_{ij} = \alpha + \mathbf{X_{ij}}\,\boldsymbol{\beta_1} + \mathbf{X_j}\,\boldsymbol{\beta_2} + u_j + e_{ij},$$

$$u_j \sim \text{Normal}\,(0, \sigma^2_u),$$

$$e_{ij} \sim \text{Normal}\,(0, \sigma^2_e),$$

where α = intercept value, $\mathbf{X_{ij}}$ = matrix of exposure variables for each cow, $\boldsymbol{\beta_1}$ = vector of coefficients for $\mathbf{X_{ij}}$, $\mathbf{X_j}$ = matrix of exposure variables for each herd, $\boldsymbol{\beta_2}$ = vector of coefficients for $\mathbf{X_j}$, u_j = a random effect to account for residual variation between herds (assumed to be normally distributed with mean = 0, and variance = σ^2_u), and e_{ij} = residual level 1 error (assumed to be normally distributed with mean = 0, and variance = σ^2_e). SCC1 was the exposure of interest for each cow, and was included on a ln scale. To focus attention on the ppp period, for the control of heifer mastitis, only confounding variables deemed to be operating by 30 DIM during parity were selected. Therefore, polynomials for ln AFC, and DIM at the first recording were investigated for

inclusion. Due to the importance of seasonal production to Irish dairy herds (chapter 2), month, and year of first calving were included as categorical terms. Biologically plausible interactions, and herd level random slopes (herd x fixed effect interactions) were assessed. Initial values for all covariates were generated in MLwiN 2.22 (Rasbash et al., 2009), using the iterative generalised least squares procedure (Goldstein, 2003). To facilitate posterior predictions that incorporated all uncertainty in parameters, the models were developed in a Bayesian framework using WinBUGS 1.4.3 (Lunn et al., 2000). Parameters were estimated from 10,000 MCMC simulations, following a burn in of 1,000 simulations during which time chain convergence occurred (determined by inspection of 3 simultaneous chains to ensure a stationary distribution had been reached (Gilks et al., 1996)). Vague prior distributions were used for; $\sigma^{-2}_{u} \sim$ Gamma (0.001, 0.001), $\sigma^{-2}_{e} \sim$ Gamma (0.001, 0.001), and $\boldsymbol{\beta} \sim$ Normal (0, 10^{6}), to give the major influence to the data in the estimation of parameters (Green et al., 2004). Distributions of covariates, and interaction terms were inspected, these remained in the model based on biological plausibility, and if the 95% Bayesian credible interval (BCI) excluded 0. Sensitivity of the final model results to prior distributions for the herd level random effect variance (Spiegelhalter et al., 2004) was evaluated by repeating simulations using the prior; $\sigma^{2}_{u} \sim$ Uniform (10^{-7}, 10^{7}).

3.2.3 Model checking

For both models, the posterior distribution of the mean residual from samp_1 was inspected to determine if the 95% BCI included 0, suggesting adequate model fit. The LiMY model was of primary interest, and further checking, and simulations used this model only. To further evaluate model fit

and usefulness (Gelman et al., 1996), fixed and random effects were used to predict cow life time milk yield ($y.pred_{ij}$) thus;

$$y.pred_{ij}^{1} \sim p(y.pred_{ij} \mid \boldsymbol{\beta}, samp\_1, u_j)$$

$$y.pred_{ij}^{xval} \sim p(y.pred_{ij}^{xval} \mid \boldsymbol{\beta}, samp\_2),$$

where *p* represents conditional probability distributions, $y.pred_{ij}^{1}$ and $y.pred_{ij}^{xval}$ are posterior predictions of LiMY for the ith cow in the jth herd in samp_1, and samp_2 respectively, $\boldsymbol{\beta}$ is the vector of model coefficient distributions, u_j is the random effect for the jth herd in samp_1. Predicted and observed mean LiMYs were calculated for cows categorised by SCC1_gp; these categories were not in the final models. Posterior predicted distributions of mean LiMY for cows in these groups were inspected to determine if the observed mean LiMYs were within the 95% BCI of the posterior predictions, indicating the extent of model usefulness for predictions based on SCC1, and if the results could potentially be generalised to other Irish dairy herds (Gelman et al., 1996).

3.2.4 Micro-simulation

To illustrate the impact of SCC1 on LiMY at herd level, and to demonstrate financial relevance, 'micro-simulation' was used (Spiegelhalter et al., 2004). This method simulates the trajectory of individual cows, to evaluate the expected outcomes for particular scenarios with all variability in model parameters, and dependence between variables included (Spiegelhalter et al., 2004). This allows the impact of SCC1 (the effect of interest) to be evaluated in the absence of confounding influences, as if a carefully controlled trial had been carried out. Therefore, the Bayesian model for LiMY was extended to

include a 1-step micro-simulation for 1,000 theoretical cows with different characteristics, in herds with ≥ 20%, and ≥ 10% initial prevalence of cows with SCC1 ≥ 400,000 cells/mL. For each cow, values for ln SCC1 were drawn from normal distributions (determined from the initial dataset; DAT) for herds in these prevalence groups (Table 3.1). In order to demonstrate the impact of achievable reductions in the prevalence of cows with SCC1 ≥ 400,000 cells/mL on LiMY at herd level, herds ≥ 20% initial prevalence of cows with SCC1 ≥ 400,000 cells/mL were deemed to reduce this to < 10%, or < 5%, and herds with ≥ 10% initial prevalence of cows with SCC1 ≥ 400,000 cells/mL, were deemed to reduce this to < 5% (Table 3.1). To provide a straight forward comparison between different herd scenarios, all simulated cows had a first calving date in February 2005. At each of 10,000 MCMC simulations (following a burn in of 1,000), final model coefficients were combined with data from the theoretical cows to generate predictions of LiMY for the ith cow in the jth herd ($y.pred_{ij}$);

$$y.pred_{ij} \sim p(y.pred_{ij} | \boldsymbol{\beta}, X^{sim}),$$

where $\boldsymbol{\beta}$ is a vector of model coefficient distributions, and X^{sim} is a matrix of data for simulated cows, including a simulated value for ln SCC1, based on the herd level prevalence of cows with SCC1 ≥ 400,000 cells/mL (Table 3.1), and indicator variables to denote a first calving in February 2005.

3.2.5 Change in revenue

At each iteration, mean LiMY for the simulated cows in each herd scenario was calculated. Differences in mean LiMY were multiplied by an estimated gross margin (milk price – variable costs of production), that was

drawn from a normal distribution with mean = 0.17 €/L, and standard deviation = 0.03 €/L for each cow (Hennessy et al., 2011), to give the difference in expected revenue associated with reductions in the prevalence of cows with SCC1 ≥ 400,000 cells/mL. Posterior distributions of revenue change /heifer calved into the herd were plotted as a cumulative frequency distribution to show the probability of different levels of financial return. An example of the model code is given in the appendix.

Table 3.1. Frequency of 7,423 Irish dairy herds categorised by prevalence of cows with SCC1[24] ≥ 400,000 cells/mL, and mean and (variance) for ln[25] SCC1 for 233,176 primiparous cows in these herds

	Herd level prevalence of cows with SCC1 ≥ 400,000 cells/mL			
	≥ 10%	≥ 20%	< 5%	< 10%
Percentage of herds	55%	19%	25%	45%
ln SCC1	4.79 (1.52)	5.11 (1.78)	4.26 (0.80)	4.39 (1.04)

[24] First test day milk somatic cell count record between 5 and 30 days in milk during parity 1.
[25] Natural logarithm of.

3.3 Results

3.3.1 Descriptive results

Summary measures were similar in samp_1 and samp_2 (Table 3.2). In samp_1, median LiMY (IQR) decreased from 23.8 tonnes (11.5 to 36.4) for cows with SCC1 < 55,000 cells/mL, to 18.9 tonnes (8.7 to 31.9) for cows with SCC1 > 400,000 cells/mL. Median FLMY (IQR) decreased from 5.5 tonnes (4.5 to 6.9) for cows with SCC1 < 55,000 cells/mL, to 5.2 tonnes (4.2 to 6.5) for cows with SCC1 > 400,000 cells/mL. There was a trend for decreased proportions of cows surviving beyond the first, and fourth lactation with increasing SCC1 (Table 3.2).

Table 3.2. Descriptive results for sub-groups of eligible Irish primiparous dairy cows[26] based on somatic cell count between 5 and 30 DIM (SCC1); medians and (interquartile range) of SCC1 and lifetime milk yield, and proportions of cows surviving beyond the first and fourth lactation

Group	Variable	samp_1	samp_2
SCC1 < 55,000 cells/mL	SCC1 ('000 cells/mL)	36 (26 to 45)	36 (26 to 45)
	Lifetime milk yield (tonne)	23.8 (11.5 to 36.4)	22.4 (11.1 to 35.4)
	First lactation milk yield (tonne)	5.5 (4.5 to 6.9)	5.6 (4.5 to 7.0)
	First lactation survival	0.81	0.80
	Fourth lactation survival	0.16	0.15
	Number of cows	6,481	8,807
SCC1 55,000 to 149,000 cells/mL	SCC1 ('000 cells/mL)	85 (68 to 108)	86 (69 to 109)
	Lifetime milk yield (tonne)	22.8 (11.1 to 35.2)	21.9 (10.7 to 35.1)
	First lactation milk yield (tonne)	5.3 (4.3 to 6.6)	5.4 (4.4 to 6.7)
	First lactation survival	0.81	0.80
	Fourth lactation survival	0.17	0.16
	Number of cows	9,027	13,011
SCC1 150,000 to 400,000 cells/mL	SCC1 ('000 cells/mL)	218 (176 to 286)	218 (176 to 286)
	Lifetime milk yield (tonne)	21.3 (10.2 to 34.0)	20.3 (10.0 to 33.9)
	First lactation milk yield (tonne)	5.4 (4.3 to 6.6)	5.4 (4.4 to 6.7)
	First lactation survival	0.80	0.78
	Fourth lactation survival	0.15	0.14
	Number of cows	3,841	5,812
SCC1 > 400,000 cells/mL	SCC1 ('000 cells/mL)	927 (570 to 1,725)	889 (571 to 1,704)
	Lifetime milk yield (tonne)	18.9 (8.7 to 31.9)	19.2 (8.9 to 33.0)
	First lactation milk yield (tonne)	5.2 (4.2 to 6.5)	5.4 (4.3 to 6.6)
	First lactation survival	0.76	0.75
	Fourth lactation survival	0.13	0.14
	Number of cows	2,674	3,999

[26] Includes eligible cows from 2,500 herds used for model development (samp_1) and 3,422 herds used for cross validation (samp_2).

3.3.2 Model results

Unit increase in ln SCC1 was associated with a median decrease in LiMY of 864 kg (95% BCI 706 to 1,024), and in FLMY of 105 kg (95% BCI 77 to 133; Table 3.3). The final models adjust for month, and year of first calving. Cows that first calved in June 2007, with mean ln SCC1 were the baseline for comparison; although LiMY did not differ between the relatively

few heifers that calved from April to August (95% BCI includes 0). Heifers calving in January had the highest LiMY, and produced a median of 5,550 kg (95% BCI 4,055 to 7,027) more milk than those calving in June. The next highest month of first calving was October, and these heifers produced a median of 4,695 kg (95% BCI 2,944 to 6,449) more milk than those calving in June. In contrast FLMY was highest for heifers calving from August to December (Table 3.3). LiMY and FLMY did not differ by year of first calving (95% BCI includes 0). However, there was a trend for decrease in LiMY, and increase in FLMY with year of first calving. Decrease in AFC, from 27 to 24 months was associated with a median increase in LiMY of 691 kg (95% CI 832 to 547). AFC was not associated with FLMY (95% BCI included 0).

Table 3.3. Bayesian credible intervals from 10,000 simulations of the final models; outcomes cow level lifetime and first lactation milk yields (kg)

		Model 3.1; lifetime milk yield (kg)			Model 3.2; first lactation milk yield (kg)		
Exposure (baseline)		Lower 2.5%	Median	Upper 97.5%	Lower 2.5%	Median	Upper 97.5%
Intercept		-4,545	10,890	26,260	241	2,954	5,642
ln[27] SCC1[28] (4.65)		-1,024	-864	-760	-133	-105	-77
Month of first calving (June)	January	4,055	5,550	7,027	370	640	900
	February	2,978	4,396	5,786	-13	237	479
	March	1,523	2,936	4,353	-334	-83	167
	April	-81	1,373	2,807	-568	-308	-55
	May	-815	801	2,397	-664	-380	-99
	July	-2,493	-19	2,400	446	876	1,307
	August	-1,096	1,306	3,972	930	1,358	1,780
	September	1,315	3,147	4,948	946	1,270	1,586
	October	2,944	4,695	6,449	1,141	1,449	1,752
	November	1,973	3,827	5,750	904	1,226	1,551
	December	1,317	3,477	5,721	887	1,263	1,642
Year of first calving (2007)	2004	-3,865	11,790	27,520	-856	1,872	4,578
	2005	-5,605	9,646	25,010	-186	2,487	5,180
	2006	-8,010	7,305	22,660	-100	2,587	5,278
ln AFC[29] (6.71)		-8,320	-6,906	-5,470	NA[30]	NA	NA
Random effect standard deviation:		Lower 2.5%	Median	Upper 97.5%	Lower 2.5%	Median	Upper 97.5%
Cow level (x 10^6)		12,763	12,888	13,019	2,215	2,237	2,260
Herd level		6,752	7,053	7,372	1,226	1,318	1,374

[27] Natural logarithm of.
[28] First test day SCC record at 5 to 30 DIM during parity 1.
[29] Age at first calving (days).
[30] Not applicable.

3.3.3 Model checking

The posterior distribution of the mean residuals for samp_1 were normal with medians 6.0 kg (95% CI -270 to 277), and 0.3 kg (95% CI -48 to 50), with the outcomes LiMY, and FLMY respectively. Therefore, the final models fitted the data on which they were developed. Predictions of LiMY for cows in samp_1 aggregated by SCC1 group also indicated good fit and hence

this model was adequate for predictions in these herds (Figure 3.1). The final model for LiMY also appeared generalisable to other Irish dairy herds as the observed mean LiMY for cows in samp_2 aggregated by SCC1 group was within the 95% BCI of posterior predictions (Figure 3.1). There was ≤ 0.4% difference in the median, and 95% BCI limits of the ln SCC1 coefficient distribution when a uniform prior distribution for the herd level random effect variance was used, and this had no substantive impact on interpretation of the final model results.

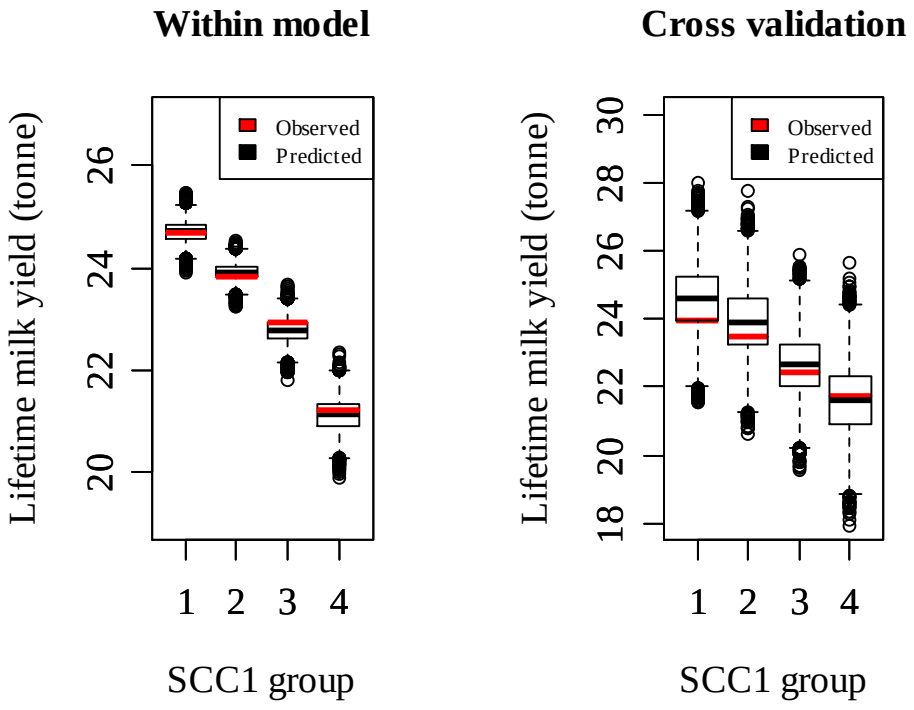

Figure 3.1. Final model predictions of lifetime milk yield from 10,000 simulations, and observed values in 2,500 Irish dairy herds used for model development, and 3,422 separate Irish dairy herds[31] used for cross validation[32]

[31] Grouped by milk somatic cell count at 5 to 30 days in milk during parity1 (SCC1); group 1; < 50,000 cells/mL, group 2; 50,000 to 99,000 cells/mL, group 3; 100,000 to 164,000 cells/mL, group 4; ≥ 164,000 cells/mL.
[32] The horizontal bold line is the median, the surrounding box contains 50% of the data, the vertical whiskers extend to 1.5 times the interquartile range, and outliers are shown beyond this.

3.3.4 Micro-simulation results

Figure 3.2 shows the probability of different levels of potential revenue change for every heifer in the herd attributable to increased LiMY, for various herd level reductions in the prevalence of cows with SCC1 ≥ 400,000 cells/mL. For example there was 75% certainty of savings of at least €97, or €115 /heifer calved into the herd, if the prevalence was reduced from ≥ 20%, to < 10%, or < 5% respectively, and at least €71 /heifer calved into the herd if the prevalence reduced from ≥ 10% to < 5% (Figure 3.2). Therefore for a herd that calves 20 heifers /year; ≥ 4 of which have SCC1 ≥ 400,000 cells/mL, there would be 75% certainty of saving at least €1,940 /year, if the number with SCC1 ≥ 400,000 cells/mL could be reduced to ≤ 1. Table 3.4 gives savings in further scenarios, and at different levels of certainty for this example herd.

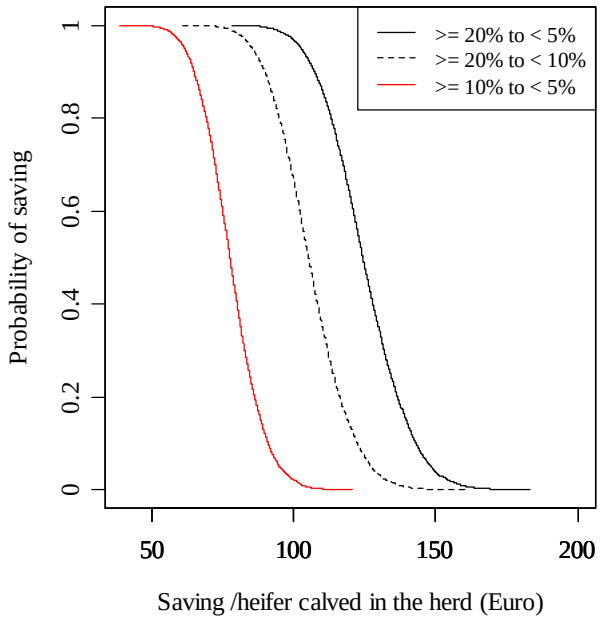

Figure 3.2. Micro-simulation over 10,000 simulations; minimum saving /heifer in the herd attributable to increased lifetime milk yield associated with specific reductions in the herd level prevalence of parity 1 cows with SCC ≥ 400,000 cells/mL between 5 to 30 days in milk

Table 3.4. Predictions for an example herd that calves 20 heifers /year; probability of annual savings through increased lifetime milk yield associated with reductions in the number of primiparous cows with SCC ≥ 400,000 cells/mL between 5 and 30 days in milk

Probability	Change in number of parity 1 cows (/20)		
	≥ 4 to ≤ 1	≥ 4 to 0	≥ 2 to 0
0.75	≥ €1,940	≥ €2,300	≥ €1,060
0.5	≥ €2,100	≥ €2,480	≥ €1,560
0.25	≥ €2,280	≥ €2,680	≥ €1,680

3.4 Discussion

This analysis in this chapter is the first to demonstrate large differences in the LiMY of cows, depending on SCC early in the first lactation. The median decrease in LiMY of 864 kg /unit increase in ln SCC early in the first lactation (for example from 55,000 to 150,000 cells/mL, or 150,000 to 400,000 cells/mL), incorporated a milk loss of 105 kg in the first lactation. For comparison, this is larger than the estimate made by De Vliegher et al. (2005a); of approximately 47 kg within 365 days of the first calving /unit increase in ln SCC at 5 to 14 DIM. Importantly, the analysis of De Vliegher et al. (2005a) was conditional on cows surviving the first lactation, and hence showed the milk loss in affected primiparous cows that survived; likely associated with residual udder pathology, but excluding milk loss associated with premature culling. Therefore, the potential decrease in LiMY was considerably more than losses in the first lactation. This highlights the usefulness of using cumulative measures of milk yield, rather than test day records alone; specifically to account for how long cows actually remain productive, in addition to decreased milk production *per se*. High SCC early in the first lactation has been associated with premature culling of cows in both Irish (chapter 5), and Belgian dairy herds (De Vliegher et al., 2005b), and this appears economically

important, through an influence on LiMY, rather than through the additional replacement costs incurred (chapter 5).

Gelman et al. (1996) have proposed that demonstrating the extent to which models are useful, rather than absolute correctness is a rational approach to model checking. Predictions of LiMY from the final model, for groups of cows (based on SCC at 5 to 30 DIM) were shown to be reliable. This not only demonstrated model fit, but that LiMY could also be predicted for cows from separate herds, not used for model development, and therefore that the results could be generalised to other Irish dairy herds, and justified the use of the micro-simulation procedure (Figure 3.1). It was important in this research to model LiMY, using only those parameters available by 30 DIM during parity 1; specifically to focus attention on potential ppp mastitis control measures for heifers. In particular, adjustment was made for seasonal variation by including month of first calving. The calving season for the majority of Irish dairy herds is from January to April (chapter 2), to allow best use of pasture for milk production. It can be hypothesised that calving earlier in spring allowed optimal use of pasture (as herd energy requirements more closely matched feed supply). Therefore, January calving heifers were possibly in better energy balance, and easier to re-breed, increasing LiMY. A subset of Irish dairy herds calves cows in autumn, to supply domestic winter milk; this could explain the relatively high LiMY for heifers that calved in October, as such herds feed more concentrates for higher milk yields. This system may be favourable for first lactation milk yield. In contrast, spring-calving primiparous cows may not have been able to make optimal use of pasture; particularly if stocking densities were high, and they were competing with mature cows. The data indicate a

trend for increased FLMY over time, and this could be due to improvements in genetics and management. The trend for decrease in LiMY with increasing year of first calving was likely due to time in the study.

The results of this chapter demonstrate that SCC1 is an economically important predictor of future productivity; at the herd level this could provide timely warning that interventions to improve management for ppp heifers are required (Table 3.4). The udder health of ppp heifers appears important to the Irish dairy industry, as the majority of herds (Table 3.1; 55%) have potential to increase revenue through reduction in the prevalence of primiparous cows with SCC1 $\geq$ 400,000 cells/mL. It was accepted in this research that not all 'milk loss' attributable to mastitis can be recovered; economic simulations have focussed on achievable reductions in the prevalence of high SCC early in the first lactation, based on observed values of herd prevalence (Table 3.1). With all possible variability in parameters included in predictions, it is highly likely there will be savings accrued in the majority of Irish dairy herds, through improving udder health early in the first lactation (Figure 3.2, Table 3.4). Additional increased revenue may be accrued through reduced incidence of clinical mastitis, and decreased replacement costs and this has not been included in the estimates. The savings presented do not account for the cost of interventions to reduce the incidence of heifer mastitis; they should be considered as 'scope for investment', and applied to aid decisions on how much expenditure can be justified to control mastitis in ppp heifers (Green et al., 2007b; 2008). Importantly for the simulated herd scenarios, it is very likely that savings of at least €50 /heifer calved into the herd would be achieved through reducing the prevalence of primiparous cows with high SCC1,

although there appears to be upper limits on these savings (€140 /heifer calved into the herd) for which the probability is close to 0, but the amount invested ultimately depends on decision makers' attitude to risk (Figure 3.2, Table 3.4). This chapter therefore gives details on possible returns on investment, assuming the target reduction is achieved.

Interventions are farm specific, but could aim to optimise the cleanliness of ppp heifers (Compton et al., 2007a) through improvements to environmental hygiene (De Vliegher et al., 2004b; Piepers et al., 2011). To aid decision making in practice, it would be useful to know the probability of different levels of revenue, associated with specific management interventions. This would depend on knowing the likely impact of the intervention on the prevalence of cows with SCC1 $\geq$ 400,000 cells/mL, and the intervention cost, and is explored further in chapter 7. For example, where ppp heifers are housed, simply storing bedding materials inside rather than outside could reduce the odds of high SCC within 30 days of calving by 21%, as occurred in English and Welsh dairy herds (Green et al., 2008). The results from Green et al. (2008) related to individual animals, and not the herd level prevalence; however the intervention cost is unlikely to exceed potential savings for the example herd (Table 3.4), and therefore could be cost effective if the prevalence of cows with high SCC1 reduced. With permanent improvements to farm infrastructure, savings may be ongoing, and accumulate as subsequent cohorts of heifers calve. Despite knowledge of risk factors for heifer mastitis (De Vliegher et al., 2012), information is lacking on the cost and efficacy of specific interventions in terms of tangible outcomes for particular herds. This information is important for the Irish dairy industry where herd expansion is

anticipated. Investigation of differences in the management, and environment of herds with varying prevalence of cows with high SCC early in the first lactation under Irish conditions would be of use for identifying where herd management changes should focus. Specific interventions could then be suggested for further evaluation based on the potential savings shown.

3.5 Conclusions

This chapter demonstrated that for cows in Irish dairy herds, SCC between 5 and 30 days in milk during parity 1 (SCC1) was negatively associated with LiMY. For the majority of Irish dairy herds with ≥ 10% prevalence of cows with SCC1 ≥ 400,000 cells/mL, there are likely to be large savings associated with improving udder health for *pre-* and *peri-partum* heifers.

Chapter 4: Association between somatic cell count early in the first lactation and the cumulative milk yield of cows in English and Welsh dairy herds

4.1 Introduction

Heifer mastitis has been recognised as a common problem of economic importance throughout the developed dairy industry worldwide (De Vliegher et al., 2012; Piepers et al., 2009). In chapter 3, the negative effect of elevated somatic cell count between 5 and 30 days in milk during parity 1 (SCC1) on the milk yield of Irish dairy cows persisted for their entire lifetime, and the median decrease was 864 kg per unit increase in ln SCC1. This result emphasised the importance of including milk production beyond the first lactation to fully understand the true extent of accrued losses. Considering impact on cumulative milk yield is therefore essential to evaluate the total cost of high milk somatic cell count (SCC) early in the first lactation and aid decision making around mastitis control measures for *pre-* and *peri-partum* (ppp) heifers; this has not been evaluated for dairy herds in England and Wales.

The aim of this chapter was to assess the association between SCC1 and cumulative milk yield over approximately 2 years for cows in English and Welsh dairy herds, and to evaluate the economic impact of the results.

4.2 Materials and methods

4.2.1 Data selection

English and Welsh dairy herds were selected according to the criteria used by Madouasse (2009; chapter 2). To be eligible for inclusion, cows required a first calving in 2004, followed by a record of SCC between 5 and 30 days in milk (DIM) during parity 1. There were 43,461 cows in 2,111 herds (DATASET1) that met these criteria.

4.2.2 Data analysis

Cumulative milk yield for each cow lactation in DATASET1 was calculated using the test interval method (ICAR, 2011), and these were summed to give an estimate of cumulative milk yield for each cow from the date of first calving in 2004 until the end of the study period on 31 December 2006. For the selected cows, 'survival time' was estimated as the number of days between a first calving date in 2004, and their last recording date. Cows were censored, if present at the final available recording date for their respective herd; otherwise it was assumed that disposal occurred at the last recording date for each cow. Kaplan-Meier survival curves were plotted using R (R-Development-Core-Team, 2010), and summary statistics produced for cows stratified by SCC1 (SCC1_gp; 1; < 55,000 cells/mL, 2; 55,000 to 149,000 cells/mL, 3; 150,000 to 400,000 cells/mL, 4; ≥ 400,000 cells/mL).

4.2.3 Model development

The outcome of interest was the cumulative milk yield (y_{ij}), for the ith cow, in the jth herd. The random effects model used for analysis took the form;

$$y_{ij} = \alpha + \mathbf{X_{ij}}\,\boldsymbol{\beta_1} + \mathbf{X_j}\,\boldsymbol{\beta_2} + u_j + e_{ij},$$

$$u_j \sim \text{Normal}\,(0, \sigma^2_u),$$

$$e_{ij} \sim \text{Normal}\,(0, \sigma^2_e),$$

where α = intercept value, $\mathbf{X_{ij}}$ = matrix of exposure variables for each cow, $\boldsymbol{\beta_1}$ = vector of coefficients for $\mathbf{X_{ij}}$, $\mathbf{X_j}$ = matrix of exposure variables for each herd, $\boldsymbol{\beta_2}$ = vector of coefficients for $\mathbf{X_j}$, u_j = a random effect to account for residual variation between herds (assumed to be normally distributed with mean = 0, and variance = σ^2_u), and e_{ij} = residual cow level error (assumed to be normally distributed with mean = 0, and variance = σ^2_e). SCC1 was included on a (ln) linear scale. Potential confounding variables available by 30 DIM during parity 1 were investigated for inclusion; specifically to account for variables known at this time that influence the relationship between SCC1 and cumulative milk yield. Therefore, DIM at the first recording (polynomial terms), and month of first calving (categorical terms) were the only confounding variables investigated. Biologically plausible interactions, and herd level random slopes (herd x fixed effect interactions) were assessed. Initial model exploration was conducted in MLwiN 2.22 (Rasbash et al., 2009), using the iterative generalized least squares procedure (Goldstein, 2003). To facilitate Bayesian posterior predictions from the model that incorporated all uncertainty in parameters, the model was further developed in WinBUGS 1.4.3 (Lunn et al., 2000). Parameters were estimated from 10,000 Markov chain Monte Carlo (MCMC) simulations, following a burn in of 1,000 simulations during which time chain convergence had occurred. This was assessed by inspection of 3 chains run in parallel to ensure a stationary distribution had been reached

(Gilks et al., 1996). Vague prior distributions were used for σ^{-2}_{u} ~ Gamma (0.001, 0.001), σ^{-2}_{e} ~ Gamma (0.001, 0.001), and $\boldsymbol{\beta}$ ~ Normal (0, 10^{6}), to give the major influence to the data in the estimation of parameters (Green et al., 2004). Distributions of covariates, and interaction terms were inspected, and these remained in the final model based on biological plausibility, and only if the 95% Bayesian credible interval (BCI) excluded 0. Sensitivity of the results to prior distributions for the herd level random effect variance (Spiegelhalter et al., 2004) was evaluated by repeating simulations using the prior; σ^{2}_{u} ~ Uniform (10^{-7}, 10^{7}).

4.2.4 Model checking

The posterior distribution of the mean residual was inspected to determine if the 95% BCI included 0, suggesting adequate model fit. Based on the methods proposed by Gelman and others (1996), model fit and usefulness were evaluated using fixed and random effects to predict the cumulative milk yield for each cow ($y.pred_{ij}$) as follows;

$$y.pred_{ij} \sim p(y.pred_{ij} \mid \boldsymbol{\beta}, \text{DATASET1}, u_j),$$

where *p* represents a conditional probability distribution, $y.pred_{ij}$ are posterior predictions of cumulative milk yield for the ith cow in the jth herd in DATASET1, $\boldsymbol{\beta}$ is the vector of final model coefficient distributions, u_j is the random effect for the jth herd. Mean cumulative milk yield was predicted for cows categorised by SCC1_gp; these categories were not used in the final model. Posterior predicted distributions of mean cumulative milk yield for cows in these groups were inspected to determine it the observed mean cumulative milk yields were within the 95% BCI of the posterior predictions,

indicating the extent of model usefulness for predictions based on SCC1 (Gelman et al., 1996).

4.2.5 Micro-simulation

The Bayesian model was extended and run simultaneously with a 1-step micro-simulation of cumulative milk yield for 1,000 theoretical cows, in herds with ≥ 10%, and ≥ 20% prevalence of cows with SCC1 ≥ 400,000 cells/mL (as in chapter 3). Herds in DATASET1 had a prevalence of cows with SCC1 ≥ 400,000 cells/mL up to 100% (with 1 eligible cow), although < 1% of herds had > 40% prevalence of cows with SCC1 ≥ 400,000 cells/mL. For each cow, simulated values for ln SCC1 were drawn from normal distributions determined from DATASET1 for herds grouped by prevalence of cows with SCC1 ≥ 400,000 cells/mL (Table 4.1). It was assumed that for herds with ≥ 10% prevalence of cows with SCC1 ≥ 400,000 cells/mL this reduced this to < 5%, and for herds with ≥ 20% prevalence of cows with SCC1 ≥ 400,000 cells/mL this reduced to < 5%, or < 10%, in order to evaluate the impact of achievable reductions in the prevalence of cows with SCC1 ≥ 400,000 cells/mL on cumulative milk yield at herd level (Table 4.1). For a straight forward comparison between different herd scenarios, all simulated cows had a first calving date in February 2004. At each of 10,000 MCMC simulations (following a burn in of 1,000 simulations), final model coefficients were used alongside data from the theoretical cows to generate predictions of cumulative milk yield for the ith cow in the jth herd ($y.pred_{ij}$);

$$y.pred_{ij} \sim p(y.pred_{ij} | \boldsymbol{\beta}, \mathbf{X}^{\mathbf{sim}}),$$

where $\boldsymbol{\beta}$ is the vector of final model coefficient distributions, and $\mathbf{X}^{\mathbf{sim}}$ is a matrix of data for simulated cows, including a simulated value for ln SCC1, based on the herd level prevalence of cows with SCC1 ≥ 400,000 cells/mL (Table 4.1), and indicator variables to denote a first calving in February 2004. An example of the model code is given in the appendix.

Table 4.1. Frequency of 2,111 English and Welsh dairy herds categorised by prevalence of cows with SCC1[33] ≥ 400,000 cells/mL, and mean and (variance) for ln[34] SCC1 for 43,461 parity 1 cows in these herds

	Herd level prevalence of cows with SCC1 ≥ 400,000 cells/mL			
	≥ 10%	≥ 20%	< 5%	< 10%
Percentage of herds	42%	12%	33%	58%
ln SCC1	4.59 (1.75)	4.93 (2.08)	4.00 (0.83)	4.13 (1.05)

[33] First test day milk somatic cell count record between 5 and 30 days in milk during parity 1.
[34] Natural logarithm of.

4.2.6 Change in revenue from cumulative milk yield

At each simulation, mean cumulative milk yield for cows in each herd scenario was predicted. Differences in mean cumulative milk yield associated with changes in the herd level prevalence of cows with SCC1 ≥ 400,000 cells/mL were multiplied by an estimated mean gross margin (Milk price – variable costs of production) of £0.20 /L (Kingsay dairy costings, United Kingdom, personal communication) to give the estimated difference in revenue. Posterior distributions of predicted savings /heifer calved into the herd were plotted as a cumulative frequency distribution to show the probability of different levels of financial return that should be expected by reducing the prevalence of cows with SCC1 ≥ 400,000 cells/mL.

4.3 Results

4.3.1 Descriptive results

Summary statistics for variables in the dataset are shown in Table 4.2. Cumulative milk yield decreased from 16.9 (interquartile range (IQR); 12.2 to 20.4) tonnes for cows with SCC1 < 55,000 cells/mL, to 15.8 (IQR; 8.9 to 19.8) tonnes for cows with SCC1 > 400,000 cells/mL. Overall, the median survival time was 791 (IQR; 607 to 888) days. No cows were censored within 700 days of a first calving in 2004. Median survival time decreased from 796 (IQR; 660 to 883) days for cows with SCC1 < 55,000 cells/mL, to 767 (IQR; 432 to 882) days for cows with SCC1 > 400,000 cells/mL (Table 4.2, Figure 4.1). The overall median time in the study was 874 (IQR; 813 to 971) days. Median time in the study varied from 868 (IQR; 812 to 959) days for cows with SCC1 < 55,000 cells/mL, to 881 (IQR; 814 to 979) for cows with SCC1 150,000 cells/mL to 400,000 cells/mL (Table 4.2).

Table 4.2. Descriptive results for 43,461 eligible cows[35] in 2,111 English and Welsh dairy herds

Group	Variable	Median (interquartile range)
SCC1[36] < 55,000 cells/mL	Cumulative milk yield[37] (tonne)	16.9 (12.2 to 20.4)
	Survival time[38] (days)	796 (660 to 883)
	Time in study[39] (days)	868 (812 to 959)
	SCC1 ('000 cells/mL)	31 (22 to 42)
	Number of cows	19,462
SCC1 55,000 to 149,000 cells/mL	Cumulative milk yield (tonne)	16.5 (11.5 to 20.3)
	Survival time (days)	796 (614 to 892)
	Time in study (days)	880 (815 to 974)
	SCC1 ('000 cells/mL)	83 (67 to 109)
	Number of cows	13,878
SCC1 150,000 to 400,000 cells/mL	Cumulative milk yield (tonne)	16.3 (10.6 to 20.2)
	Survival time (days)	782 (534 to 887)
	Time in study (days)	881 (814 to 979)
	SCC1 ('000 cells/mL)	221 (177 to 284)
	Number of cows	5,889
SCC1 > 400,000 cells/mL	Cumulative milk yield (tonne)	15.8 (8.9 to 19.8)
	Survival time (days)	767 (432 to 882)
	Time in study (days)	879 (805 to 997)
	SCC1 ('000 cells/mL)	845 (553 to 1,577)
	Number of cows	4,232

[35] Cows with an SCC record between 5 and 30 DIM during parity 1 during 2004.
[36] SCC between 5 and 30 DIM during parity 1.
[37] Estimated total milk yield from date of first calving to date of last recording up to 31 December 2006.
[38] Number of days from date of first calving to date of last recording on or before 31 December 2006.
[39] Number of days from date of first calving to 31 December 2006.

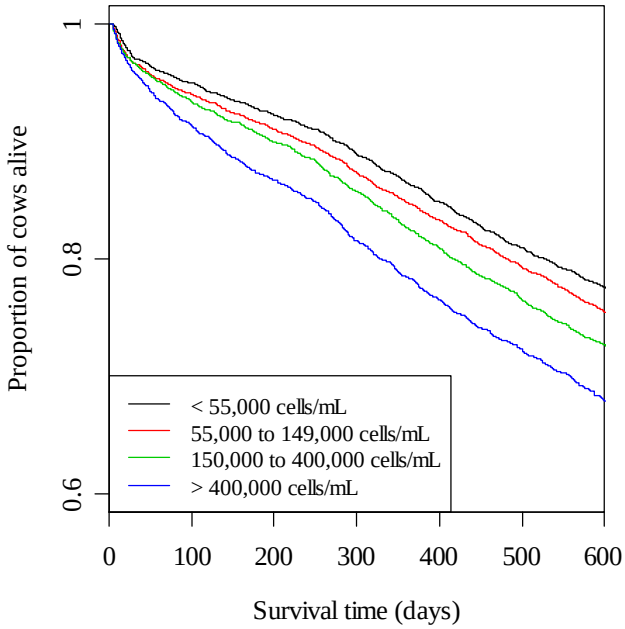

Figure 4.1. Kaplan-Meier survival curves[40] for cows in 2,111 English and Welsh dairy herds grouped by somatic cell count between 5 and 30 days in milk during parity 1

[40] Cows were censored if present at the last available recording date for their respective herd.

4.3.2 Model results

The final model is presented in Table 4.3. Cows that calved in January 2004, with mean ln SCC1 (4.33) were the baseline for comparison. Cows that calved from February to December 2004 had lower cumulative milk yields by the end of the study period. Having accounted for month of calving (which also adjusted for time in the study), a unit increase in ln SCC1 (for example from 55,000 to 150,000 cells/mL, or 150,000 to 400,000 cells/mL) was associated with a median decrease in cumulative milk yield of 482 kg (95% BCI 431 to 534) over a median of 868 days (IQR; 812 to 959) in the study.

Table 4.3. Bayesian credible intervals from 10,000 simulations of the final model; outcome cow level cumulative milk yield (kg)

Exposure (baseline)		Lower 2.5%	Median	Upper 97.5%
Intercept		17,740	17,980	18,230
ln[41] SCC1[42] (4.33)		-534	-482	-431
Month of first calving (January)	February	-813	-485	-175
	March	-1,367	-1,020	-676
	April	-1,901	-1,526	-1,153
	May	-1,916	-1,565	-1,218
	June	-2,478	-2,167	-1,858
	July	-2,610	-2,310	-2,019
	August	-3,308	-3,017	-2,732
	September	-3,633	-3,345	-3,064
	October	-4,240	-3,960	-3,676
	November	-4,625	-4,336	-4,045
	December	-5,270	-4,957	-4,636
Random effects standard deviation:		Lower 2.5%	Median	Upper 97.5%
Cow level		6,173	6,215	6,286
Herd level		2,534	2,641	2,747

[41] Natural logarithm.
[42] First test day SCC record at 5 to 30 DIM during parity 1 ('000 cells/mL).

4.3.3 Model checking

The posterior distribution of the mean residual was normal and included 0 kg (95% BCI -82 to 83), indicating the model fitted the data on which it was developed. Predictions of cumulative milk yield for cows in DATASET1, aggregated by SCC1 group also indicated good fit, and hence that the model was suitable for predictions in these herds (Figure 4.2). There was < 0.5% difference in the median, and 95% BCI limits of the ln SCC1 coefficient distribution when a uniform prior distribution for the herd level random effect variance was used, and this had no substantive impact on model interpretation.

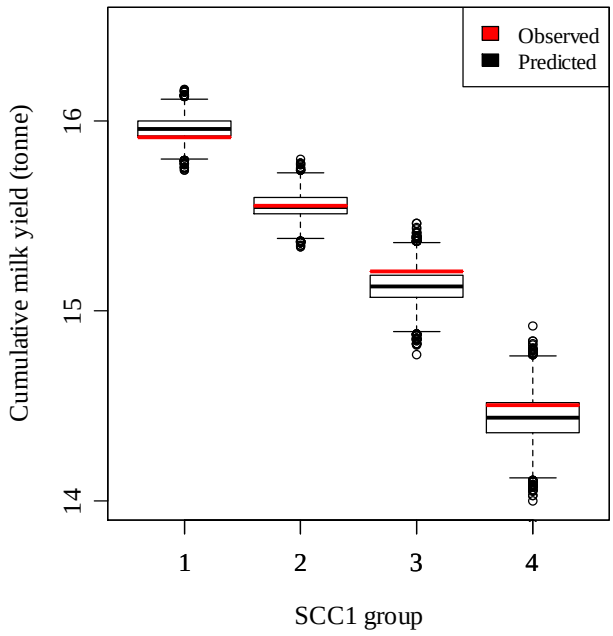

Figure 4.2. Model predictions of cumulative milk yield[43] from 10,000 simulations, and observed values in 2,111 English and Welsh dairy herds, for cows grouped by SCC at 5 to 30 days in milk during parity1 (SCC1) [44]

[43] From date of first calving in 2004 to date of last recording up to December 31, 2006.
[44] Group 1; < 55,000 /mL, group 2; 55,000 to 149,000 /mL, group 3; 150,000 to 400,000 /mL, group 4; ≥ 400,000 /mL.

4.3.4 Micro-simulation results

Figure 4.3 shows the estimated probability of different levels of potential saving attributable to increased cumulative milk yield for various herd level reductions in the prevalence of cows with SCC1 ≥ 400,000 cells/mL. For example there was 75% certainty of saving at least £73, or £85 /heifer calved into the herd over approximately 2 years, if the prevalence of cows with SCC1 ≥ 400,000 cells/mL reduced from ≥ 20%, to < 10%, or < 5% respectively, and at least £53 /heifer calved into the herd over approximately 2 years if the prevalence of cows with SCC1 ≥ 400,000 cells/mL reduced from ≥ 10% to < 5% (Figure 4.3). Therefore, for a herd that calves 20 heifers /year; ≥ 4 (≥ 20%) of which have SCC1 ≥ 400,000 cells/mL, there is 75% certainty of saving at

least £1,460 /year, if the number of heifers with SCC1 ≥ 400,000 cells/mL were reduced to ≤ 1 (≤ 10%) /year. Table 4.4 gives cost savings in further scenarios, and at different levels of certainty for this example herd.

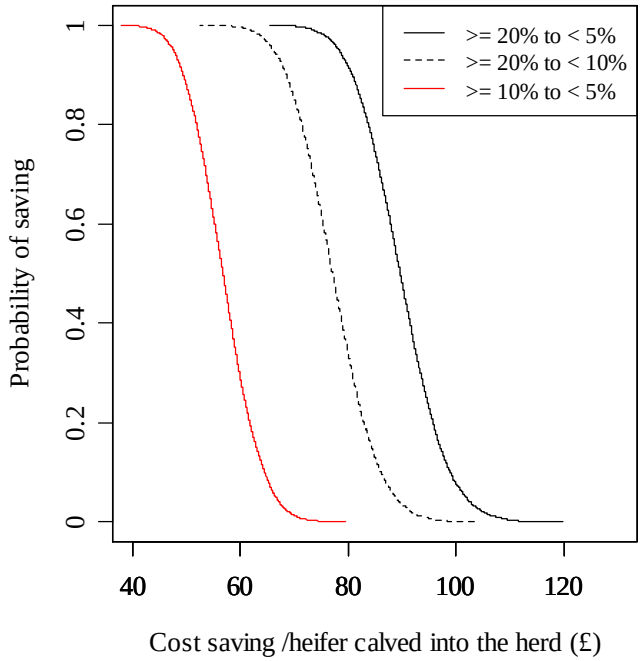

Figure 4.3. Micro-simulation over 10,000 simulations; minimum saving per heifer calved into the herd attributable to increased cumulative milk yield[45] associated with reduction in the herd level prevalence of parity 1 cows with SCC[46] ≥ 400,000 cells/mL between 5 and 30 days in milk during parity 1

[45] From date of first calving in 2004 to date of last recording up to December 31, 2006.
[46] Milk somatic cell count.

Table 4.4. Predictions for an example herd that calves 20 heifers /year; probability of annual savings through increased cumulative milk yield[47] associated with reductions in the number of parity 1 cows with SCC[48] ≥ 400,000 cells/mL between 5 and 30 DIM

Probability	Change in number (/20) and proportion of parity 1 cows		
	≥ 4 to ≤ 1 ≥ 20% to ≤ 10%	≥ 4 to 0 ≥ 20% to ≤ 5%	≥ 2 to 0 ≥ 10% to ≤ 5%
0.75	≥ £1,460	≥ £1,700	≥ £1,060
0.5	≥ £1,540	≥ £1,800	≥ £1,140
0.25	≥ £1,640	≥ £1,900	≥ £1,220

[47] Over approximately 2 years.
[48] Milk somatic cell count.

4.4 Discussion

Important losses in lifetime milk yield associated with SCC between 5 and 30 DIM (SCC1) for cows in Irish dairy herds were identified in chapter 3. In this chapter, differences in the milk yield of cows in English and Welsh dairy herds associated with changes in SCC1 were also identified. Measures of cumulative milk yield were different between these studies, however the magnitude of losses are comparable. Unit increase in ln SCC1 in both studies was associated with a 3% decrease in cumulative milk yield over approximately 2 years, assuming proportional losses over time. The estimated impact of SCC1 on cumulative milk yield for cows in English, and Welsh herds over 1 year (482 kg /2 years) was 5 times larger than an estimate of milk loss in Belgian cows of around 47 kg over 365 day /unit increase in ln SCC shortly after the first calving (De Vliegher et al., 2005a). As discussed in chapter 3, the analysis of De Vliegher et al. (2005a) included only cows that survived the first lactation, and excluded cows that were culled. Therefore the estimated milk loss is likely to be an underestimate of the true effect. Cumulative milk yield is a composite of both decreased milk yield while alive, and decreased longevity to give a realistic estimate of milk loss at cow level. Decreased milk yield attributable to high SCC between 5 and 30 DIM during parity 1 extended into subsequent lactations (as in chapter 3) and was associated with a decrease in survival time as in chapters 5, and in previous research (De Vliegher et al., 2005b).

In order to focus attention on the potential impact of mastitis control measures for ppp heifers, only parameters available by 30 DIM during parity 1

were used to model the cumulative milk yield of cows, and this could be predicted by including SCC1. At herd level, the requirement for interventions to improve the management of ppp heifers could therefore be based on the prevalence of cows with high SCC1 (Table 4.4). The simulated reductions in the herd level prevalence of cows with high SCC1 were based on observed data (Table 4.1), and therefore realistically achievable in practice. Changes in cumulative milk yield at the herd level, show that savings are very likely for 42% of English and Welsh herds with ≥ 10% prevalence of cows with SCC1 ≥ 400,000 cells/mL (Table 4.1), if this prevalence could be reduced, although upper limits on potential savings were identified (Figure 4.3, Table 4.4). Investment in control measures for heifer mastitis depends on many factors, such as the decision makers' financial situation, willingness to pay, and attitude to risk (Figure 4.3, Table 4.4). Assuming target reductions in the prevalence of cows with high SCC1 can be achieved, this chapter gives details on possible 'scope for investment'.

The savings presented do not include the costs of interventions, and they should be applied to inform decisions on rational expenditure to control mastitis in ppp heifers (Green et al., 2007b; 2008). Risk factors for heifer mastitis have been identified (De Vliegher et al., 2012), however the relative cost and efficacy of specific interventions on different farms to reduce the prevalence of cows with high SCC early in the first lactation is unknown. This information would be important to offset against the savings reported here, to assess the cost effectiveness of interventions to control heifer mastitis. For example, implementing a system of pasture rotation for *pre-partum* heifers and dry cows kept at grass (allowing ≥ 4 weeks rest between grazing sessions of ≤

2 weeks), reduced the odds of clinical mastitis, and high SCC within 30 days of calving by 68%, and 46% respectively in 52 English and Welsh dairy herds (Green et al., 2007a; 2008), but these results relate to individual animals, and not herd level prevalence. However the cost (for example in fencing materials) is unlikely to exceed potential savings (Figure 4.3, Table 4.4). Therefore the intervention could be cost effective, if the prevalence of cows with high SCC in early lactation reduced sufficiently. Ongoing savings can be expected for subsequent cohorts of heifers calved into the herd if permanent changes to farm infrastructure are made (for example improved field access, and water supply), assuming target prevalence levels for high SCC early in the first lactation are maintained. Additional savings may be accrued through reduced incidence of clinical mastitis and replacement costs that have not been considered, however the economic impact of the latter is expected to be small (chapter 5). Further research is needed into possible interventions, and knowledge of differences in the management, and environment of herds with varying prevalence of cows with high SCC early in the first lactation would be of use in formulating advice. In addition, there may have been further losses in milk production beyond the time period considered in this research (chapters 3 and 6), and this could make control of heifer mastitis even more economically favourable.

4.5 Conclusion

This chapter demonstrated that for cows in English and Welsh dairy herds, SCC between 5 and 30 DIM during parity 1 (SCC1) was negatively associated with cumulative milk yield over approximately 2 years. For dairy herds in England and Wales with ≥ 10% prevalence of cows with SCC1 ≥ 400,000 cells/mL, there are likely to be financial savings associated with improving the udder health of *pre-* and *peri-partum* heifers.

Chapter 5: Association between somatic cell count early in the first lactation and the longevity of cows in Irish dairy herds

5.1 Introduction

Mastitis is well recognized as a costly disease of dairy cows, with losses accrued mainly from decreased milk production, and discarded milk (Kossaibati and Esslemont, 1997). However, mastitis has also been associated with reduced longevity (Beaudeau et al., 1993; Seegers et al., 1998), and this has been estimated as the next biggest cost (Heikkilä et al., 2012; Huijps et al., 2008). Further losses such as the cost of drugs, veterinary services, diagnostic costs, labour, decreased milk quality, capital investments, and impact on other diseases (Halasa et al., 2007) are typically less, but may be important for particular herds (Huijps et al., 2008). Premature disposal is of particular relevance for heifers that develop mastitis (Heikkilä et al., 2012), as they must typically reach the second lactation to produce sufficient milk to break even on rearing costs (chapter 3). Increased longevity of cows reduces demand for replacement heifers giving economic benefits at the farm level, such as the opportunity costs of producing more beef calves, selling surplus heifers, increasing the size of the milking herd, or leasing resources. Alternatively, a surplus of replacement heifers creates the opportunity for increased voluntary culling, and selective breeding to improve the genetic merit of the herd.

Premature culling in the first lactation has been associated with IMI at calving in pasture-based herds in New Zealand (Compton et al., 2007b). In Belgian herds, first lactation culling hazard increased by 11% per unit increase in the natural logarithm of (ln) somatic cell count (SCC) for primiparous cows

at 5 to 14 days in milk (DIM), and by 32% when only culling for udder health reasons were considered (De Vliegher et al., 2005b). However, the impact of SCC early in the first lactation on lifetime survival has not been evaluated and this is important because the full repercussions of IMI in early life may not become evident until later in life (chapter 3). As heifers make up the largest parity group in many Irish herds (ICBF, 2011), especially following expansion (a trend that may continue in anticipation of the abolition of European Union milk quotas in 2015), understanding the repercussions of heifer IMI is of particular importance.

The aim of this chapter was to assess the association between SCC1, and survival over a 5 year period for cows in Irish dairy herds. A Bayesian approach was taken, and posterior predictions were used to evaluate the magnitude, and financial relevance of this effect, in the context of particular herd scenarios.

5.2 Materials and methods

5.2.1 Data selection

To be eligible for inclusion in the analysis, cows in Irish dairy herds required at least one SCC recording between 5 and 30 DIM during parity 1; 233,176 cows in 7,423 herds were included (DAT), as used in chapter 3. Two random samples of 1,000 of these herds were taken, and all records for eligible cows were extracted using R (R-Development-Core-Team, 2010). Not all herds sampled had dates of birth available for cows. For those that did, minimum age at first calving (AFC) was 371 days. Heifers with AFC < 700 days were deemed at increased risk of culling independent of SCC1 because of dystocia (Berry and Cromie, 2009), and individual cows with AFC < 700 days (6% of the total population) were discarded to remove this effect. Following selection there were 147,458 records from 7,537 cows in 812 herds in the first sample dataset (sample_1), used for model development, and 144,113 records from 7,353 cows in 808 herds in the second (sample_2), used for cross validation.

5.2.2 Definition of disposal

Survival time was estimated as the number of days between the dates of first calving and the last recording, and was aggregated into 50 day intervals. Disposal (death or culling) was assumed to occur in the last 50 day interval for each cow, in the absence of censoring. In survival analysis, censoring accounts for those cows in the dataset for which disposal (the event of interest) may occur when not under observation. This allows them to contribute to the denominator population at risk during the study period (Dohoo et al., 2009). There were 3 reasons for censoring in this analysis. Firstly, this related to the

dataset structure; disposal is a terminal event, and could only occur in the last 50 day interval for each cow, therefore censoring occurred in every interval survived until the last. Secondly, cows were censored at the last 50 day interval, if identified at a later time in other herds (assumed sold). Thirdly, cows were censored at the last 50 day interval if they were present at the last available test date for the respective herd. Median and interquartile range (IQR) for variables in sample_1, and sample_2 were determined.

5.2.3 Model development

Cow disposal was coded as a binary outcome. The discrete time logistic survival model used for analysis took the form;

$$disposed_{ijk} \sim \text{Bernoulli (probability} = \pi_{ijk}),$$

$$\text{logit}(\pi_{ijk}) = \alpha + int_{ijk} + int_{ijk}^2 + int_{ijk}^3 + \mathbf{X_{ijk}}\boldsymbol{\beta_1} + \mathbf{X_{jk}}\boldsymbol{\beta_2} + \mathbf{X_k}\boldsymbol{\beta_3} + v_k + u_{jk},$$

$$v_k \sim \text{Normal}(0, \sigma^2_v),$$

$$u_{jk} \sim \text{Normal}(0, \sigma^2_u),$$

where the subscripts i, j, and k denote the ith 50 day interval, for jth cow, in the kth herd respectively, α = intercept value, int = 50 day interval numbered from first calving (included on a ln scale centred on the mean interval number), $\mathbf{X_{ijk}}$ = matrix of exposure variables for each interval, $\boldsymbol{\beta_1}$ = vector of coefficients for $\mathbf{X_{ijk}}$, $\mathbf{X_{jk}}$ = matrix of exposure variables for each cow, $\boldsymbol{\beta_2}$ = vector of coefficients for $\mathbf{X_{jk}}$, $\mathbf{X_k}$ = matrix of exposure variables for each herd, $\boldsymbol{\beta_3}$ = vector of coefficients for $\mathbf{X_k}$, v_k = random effect to account for residual variation between herds (assumed to be a normal distribution with mean = 0 and variance σ^2_v), u_{jl} = random effect to account for residual variation between

cows (assumed to be a normal distribution with mean 0 and variance σ^2_u). Covariates tested in the model were ln SCC1, together with milk, fat and protein proportions recorded between 5 and 30 DIM in parity 1 (TDY1, TDF1, and TDP1 respectively); these continuous variables were centred on their mean value. DIM at the first recording was also tested, and this was centred on 5 DIM. Month and year of first calving, and month of final recording were investigated as categorical variables.

Time-varying covariates are those that can take different values depending on the 50 day interval they refer to for a particular cow, and are an important consideration in survival analyses (Gröhn et al., 1997). To investigate the impact of time-varying covariates on disposal from the herd, categorical variables were constructed such that missing values in particular 50 day intervals could be included as categories, to maintain the structure of the dataset, and hence represent time at risk of disposal for each cow. Lagged time-varying covariates from the 2 previous 50 day intervals were investigated for inclusion in the model. The time-varying covariates were; SCC group (1; < 55,000 cells/mL, 2, 55,000 to 147,000 cells/mL 3; ≥ 148,000 cells/mL, and missing), TDY group (1; < 20 kg, 2; 20 to < 30 kg, 3; ≥ 30 kg, and missing), and DIM group (1; < 100 d, 2; 100 to 199 d, 3; 200 to 399 d, 4; > 399 d, and missing).

To avoid biased parameters associated with likelihood methods (Browne and Draper, 2006), the final model was estimated in WinBUGS 1.4.3 (Lunn et al., 2000), using 10,000 Markov chain Monte Carlo (MCMC) simulations for parameter estimation, following a burn in of 1,000 MCMC simulations during which time chain convergence occurred. Initial values for all covariates were

generated in MLwiN 2.22 using penalised quasi-likelihood (Rasbash et al., 2009). Vague prior distributions were used for $\sigma^{-2}_{v} \sim$ Gamma (0.001, 0.001), $\sigma^{-2}_{u} \sim$ Gamma (0.001, 0.001), and $\boldsymbol{\beta} \sim$ Normal (0, 10^6), which meant the data had overriding influence for estimation of parameters (Green et al., 2004). Covariates and interaction terms were selected based on biological plausibility, and when the 95% Bayesian credible interval (BCI) for the posterior odds ratio distribution excluded 1. Sensitivity of the results to prior distributions for the random effect variances (Spiegelhalter et al., 2004) was evaluated by repeating simulations using the following priors; $\sigma^2_{v} \sim$ Uniform (10^{-6}, 10^6), and $\sigma^2_{u} \sim$ Uniform (10^{-6}, 10^6).

5.2.4 Model assessment

In order to assess model fit (Green et al., 2009), and usefulness (Gelman et al., 1996), posterior predicted distributions of disposal risk for sub-sets of cows from sample_1 and sample_2, were generated during the MCMC simulation. The posterior predictive binomial distribution for the occurrence of disposal in each interval for each cow (y_{ijk}) can be summarized as;

$$y_{ijk} \sim p(y_{ijk} | \boldsymbol{\beta}, \text{sample\_1}, v_k, u_{jk}),$$

where *p* represents a conditional probability distribution, $\boldsymbol{\beta}$ is the vector of coefficient distributions, sample_1 is the data in the first sample dataset, v_k and u_{jk} are conditional probability distributions;

$$v_k \sim p(v_k | \sigma^2_{v}),$$

$$u_{jk} \sim p(u_{jk} | \sigma^2_{u}),$$

and σ^2_v, and σ^2_u, are posterior predictive distributions for herd, and cow level random effect variances respectively. To assess model fit, the sub-sets of sample_1 used for posterior predictions were data relating to parameters from the final model; 50 day interval from first calving, calendar month of first calving, and calendar month of last recording. To assess model usefulness, the sub-sets of sample_1 used for posterior predictions were data that were not used for parameter estimation in the final model, these were; SCC1 group (1; < 50,000 /mL, 2; 50,000 to 99,000 /mL, 3; 100,000 to 164,000 /mL, 4; ≥ 164,000 /mL), estimated bulk milk SCC (BMSCC) group (geometric mean BMSCC estimated from all cow test day SCC records available for each herd, weighted by all TDY records in the full dataset; 1; < 200,000 /mL, 2; 200,000 /mL to 249,000 /mL, 3; 250,000 /mL to 399,000 /mL, 4; > 399,000 /mL), and AFC group (1; < 730 days, 2; 730 to 759 days, 3; 760 to 849 days, 4; > 849 days). To investigate whether results could potentially be generalised to other Irish herds, prediction of the conditional binomial distribution for the occurrence of disposal (y_{ijk}^{xval}), for cows in sub-sets based on SCC1 and BMSCC in sample_2, were made thus;

$$y_{ijk}^{xval} \sim p(y_{ijk}^{xval} \mid \boldsymbol{\beta}, \text{sample\_2}),$$

where $\boldsymbol{\beta}$ is the vector of coefficient distributions, and sample_2 is the data in the second sample dataset, which was not used for estimating parameters in the final model.

5.2.5 Micro-simulation of herd scenarios

To illustrate the impact of SCC1 on survival at herd level, and to demonstrate financial relevance micro-simulation was used as in chapters 3 and

4. Therefore, the Bayesian model was extended to include a one-step micro-simulation of disposal risk for 1,000 theoretical cows with different characteristics, in herds with ≥ 10%, or ≥ 20% initial prevalence of cows with SCC1 ≥ 400,000 cells/mL. Initial herd level prevalence group cut offs were selected based on the observed distributions of cows with SCC1 ≥ 400,000 cells/mL in DAT (Table 5.1), and data from the first milk recording at 5 to 30 DIM for heifers in these groups were simulated from normal distributions determined from DAT (Table 5.1). Scenarios were used such that for herds with ≥ 10% of cows with SCC1 ≥ 400,000 cells /mL this was reduced this to < 5%, and for herds with ≥ 20% of cows with SCC1 ≥ 400,000 cells/mL this was reduced to < 5%, or < 10%. To model these changes, distributions for ln SCC1 were used as shown in Table 5.1. Distributions for TDY1, and TDF1 remained unchanged in order to demonstrate solely the impact on disposal risk of achievable reductions in the herd level prevalence of cows with SCC1 ≥ 400,000 cells/mL. For a straight forward comparison between different herd scenarios, all simulated heifers had a first calving in February aged 24 months, and a final recording in December. The conditional predicted binomial distribution for the occurrence of disposal ($pred_{ij}$), in each 50 day interval (i), for each cow (j) was;

$$pred_{ij} \sim p(pred_{ij} | \boldsymbol{\beta}, \mathbf{X}^{\mathbf{sim}}),$$

where $\boldsymbol{\beta}$ is the vector of coefficient distributions in the final model, and $\mathbf{X}^{\mathbf{sim}}$ is a matrix of simulated exposure variables for the cows; which included ln SCC1, TDY1, and TDF1 that were drawn from the distributions in Table 5.1, 50 day interval, month of last recording, and DIM category. Risk of disposal from the herd within the 350, 700, 1,050, 1,400, and 1,750 days after first

calving was calculated from the conditional probability of each cow surviving subsequent 50 day intervals, thus;

$$\text{disposal risk within n 50 day intervals}_j = 1-(\prod_{i=1}^{n} 1-\text{pred}_{ij}),$$

where pred_{ij} is the probability of disposal in the ith interval for the jth cow. An example of this calculation is given in Figure 5.1 for disposal risk within 350 days from first calving (n = 7). The calculated disposal risk for each cow (j) was then used to draw from a Bernoulli distribution if each cow would be disposed (as a binary outcome) within n 50 day intervals (i) from first calving, thus;

$$\text{disposed within n intervals}_j \sim \text{Bernoulli (probability} = 1-(\prod_{i=1}^{n} 1-\text{pred}_{ij})).$$

The difference in the number of cows disposed over time in the simulated herds following reductions in the prevalence of cows with SCC1 ≥ 400,000 cells/mL was multiplied by a replacement cost of €1,451 /cow (Kennedy et al., 2011) to give an estimated reduction in herd disposal cost attributable to changes in herd level prevalence of cows with SCC1 ≥ 400,000 cells/mL, and this was expressed as the cost (€) /heifer calved in the herd. Following 10,000 MCMC simulations, the posterior probabilities of magnitudes of saving within 1,750 days of first calving were plotted as a cumulative frequency distribution to illustrate the likelihood of different cost benefits. The posterior probability (Bayesian *p* value (Gelman et al., 1995)) that disposal risk was higher for cows in herds with ≥ 20% initial prevalence of cows with SCC1 ≥ 400,000 cells/mL,

compared to the same herds following reduction in prevalence to < 5% was determined.

5.2.6 Micro-simulation of baseline disposal risk

In order to aid interpretation, the impact of SCC1 on disposal risk was investigated on a continuous scale with ln $SCC1^{SIM}$ defined as uniform (0, 9.2), to include the full range of possible values. Predictions were based on draws from this distribution for baseline cows (**base**; February calving, AFC = 24 months, last recording in December, < 100 DIM, TDY1; 23 kg/d, TDF1; 0.04, at 450 to 500 days from first calving), thus;

$$\text{base} \sim p(\text{base}|\ \boldsymbol{\beta}, \mathbf{X}^{\mathbf{SIM2}}),$$

where p represents a conditional probability distribution, $\boldsymbol{\beta}$ is the vector of coefficient distributions, and $\mathbf{X}^{\mathbf{SIM2}}$ is a matrix of data for the simulated cows. Simulations were repeated for groups of cows that were 100 to 199, and 200 to 304 DIM. Regression lines were estimated for the posterior relationship between ln $SCC1^{SIM}$, and risk of disposal in each group. Examples of the model code are given in the appendix.

Table 5.1. Frequency of 7,423 Irish herds categorised by prevalence of cows with SCC1[49] ≥ 400,000 cells/mL, and mean and (variance) for normally distributed variables measured at 5 to 30 days in milk in 233,176 parity 1 cows in these herds; values were used to simulate economic impact of herd level reductions in the prevalence of cows with SCC1 ≥ 400,000 cells/mL

	Herd level prevalence of cows with SCC1 ≥ 400,000 cells/mL			
	≥ 10%	≥ 20%	< 5%	< 10%
Percentage of herds	55%	19%	25%	45%
SCC1[50] (cells/mL)	120,000	170,000	71,000	81,000
ln SCC1	4.79 (1.52)	5.11 (1.78)	4.26 (0.80)	4.39 (1.04)
Milk1[51]	22.4 (30.0)	21.6 (32.3)	Unchanged	
Fat1[52]	0.041 (0.00007)			

[49] First test day somatic cell count record between 5 and 30 days in milk during parity 1.
[50] Geometric mean.
[51] First test day milk yield record (kg) between 5 and 30 days in milk during parity 1.
[52] First test day fat record (proportion) between 5 and 30 days in milk during parity 1.

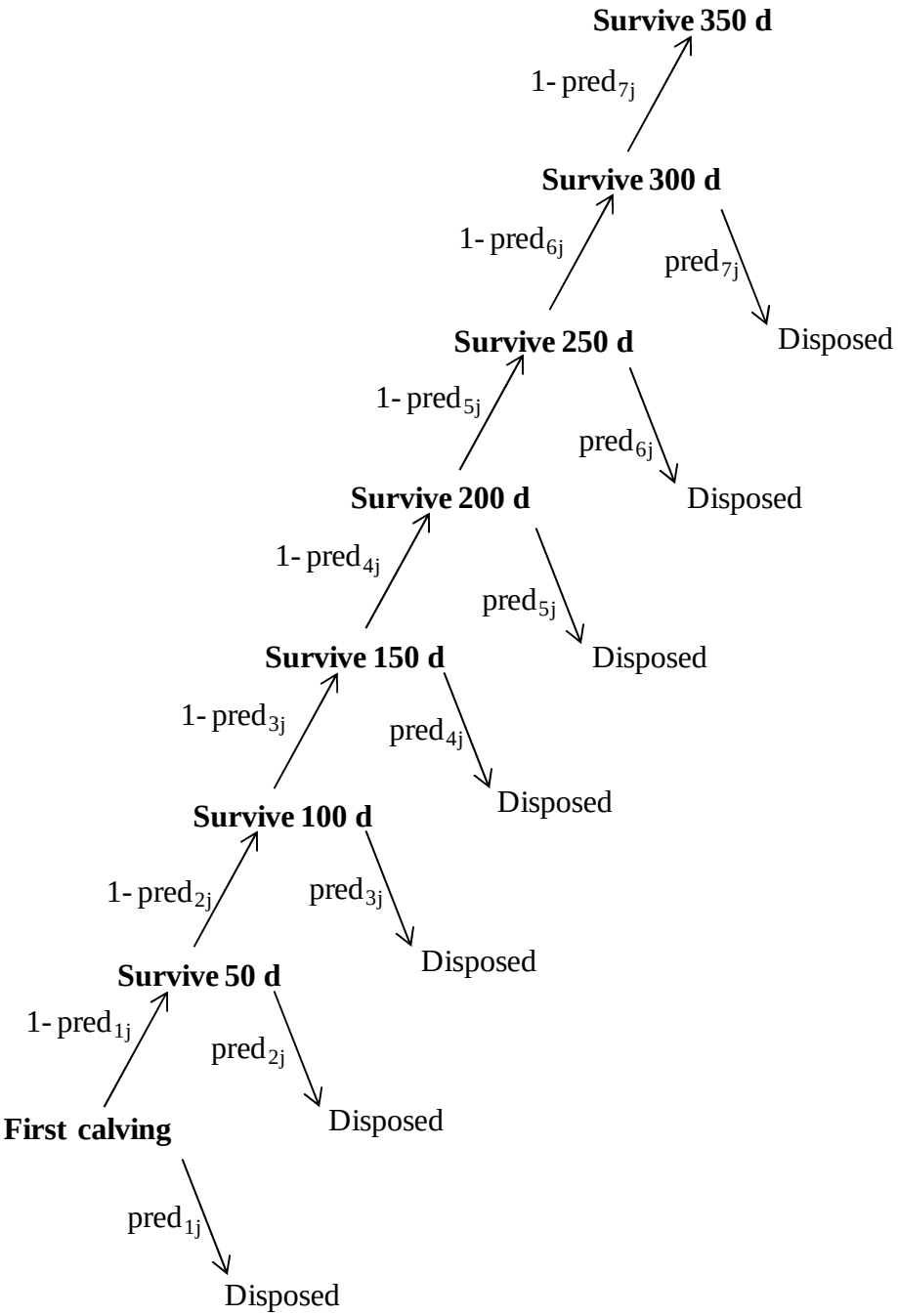

Figure 5.1. Diagram to represent calculation of the probability[53] of cow disposal within 350 days (d) from first calving[54]

[53] Probability of disposal in the ith interval for the jth cow = $pred_{ij.}$

[54] Probability of disposal within 350 days from first calving = 1 – (probability of surviving 350 days).

5.3 Results

5.3.1 Descriptive results

Descriptive statistics for sample_1 and sample_2 were similar (Table 5.2). In sample_1, median (IQR) SCC1 was 86,000 (51,000 to 172,000) cells/mL, and 54% of cows were disposed of during the study period after a median (IQR) time at risk of 3.7 (2.8 to 4.5) years. Distributions of SCC1, time at risk, and AFC were right skewed. Distributions of other variables from the first recording (TDY1, TDF1, and TDP1) were normal. Four per cent of cows moved to other herds and were censored.

Table 5.2. Descriptive statistics for cows with a recording 5 to 30 days in milk from first calving in random samples of 812, and 808 Irish dairy herds[55]

	812 herds used for model[56] development	808 herds used for cross validation of model
Number of cows	7,537	7,353
Number of cows disposed	4,101	3,944
Median month of first calving (IQR[57])	April 2005 (February 2005 to February 2006)	April 2005 (February 2005 to February 2006)
Median age at first calving (IQR)	2.1 (2.0 to 2.3) years	2.1 (2.0 to 2.4) years
Median SCC[58] (IQR)	86,000 (51,000 to 172,000) cells/mL	85,000 (49,000 to 176,000) cells/mL
Median test day milk yield[2] (IQR)	23 (19 to 26) kg	23 (19 to 26) kg
Median test day fat proportion[2] (IQR)	0.040 (0.036 to 0.045)	0.040 (0.036 to 0.045)
Median test day protein proportion[2] (IQR)	0.032 (0.030 to 0.034)	0.032 (0.030 to 0.034)
Median time at risk[59] (IQR)	3.7 (2.8 to 4.5) years	3.7 (2.8 to 4.5) years

[55] Based on herd test day data from 2005 to 2009.
[56] Discrete time logistic survival model for cow disposal in any 50 day interval from first calving.
[57] Interquartile range.
[58] At the first recording between 5 and 30 days in milk during parity 1.
[59] Time between date of first calving and date of last recording.

5.3.2 Model results

The final models are presented in Table 5.3. For Model 5.1, that focussed on SCC1 as the exposure of interest, the median odds of a cow disposal in any 50 day interval increased by 5% (median odds ratio (MOR) = 1.05 (95% BCI; 1.02 to 1.09)), with every unit increase in SCC1 (as a ln linear score). Increased milk and fat proportions (TDY1 and TDF1), were negatively associated with disposal; unit and 0.01 unit increases were associated with 2% (MOR = 0.98 (95% BCI; 0.97 to 0.98)), and 7% (MOR = 0.93 (95% BCI; 0.89 to 0.98)) reductions in the odds of disposal in each interval respectively. Decrease in AFC from 27 to 24 months was associated with a 10% reduction in the odds of disposal (MOR = 0.90 (95% BCI; 0.93 to 0.88)). Cows with a first calving in November had the highest odds of disposal, 39% (MOR = 1.39 (95% BCI; 1.12 to 1.73)) greater than those calving in February, and cows with their last recording in March had the highest odds of disposal, this was 10 times higher (MOR = 9.90, (95% BCI 8.04 to 12.16)) than in December. Random effect variance was greater at herd level than cow level (Table 5.4), indicating there was more variation in cow disposal between herds, than between cows within herds. There was < 3% difference in the MOR, and limits of the 95% BCI when the uniform prior distribution for the random effect variances was used, and this had no substantive impact on model interpretation.

Table 5.3. Model 5.1[60]; 95% Bayesian credible intervals for the odds ratios for cow disposal from 812 Irish dairy herds

Exposure (baseline)		Lower 2.5%	Median	Upper 97.5%
Intercept		0.002	0.002	0.003
ln SCC1[61] (4.64)		1.020	1.052	1.085
TDY1[62] (23 kg)		0.968	0.976	0.983
TDF1[63] (0.04)		0.000	0.001	0.081
Month of first calving (February)	January	0.896	1.010	1.135
	March	1.106	1.222	1.343
	April	1.132	1.287	1.460
	May	1.187	1.436	1.720
	June	0.885	1.160	1.503
	July	0.909	1.398	2.073
	August	0.967	1.486	2.187
	September	1.176	1.465	1.802
	October	1.069	1.317	1.603
	November	1.121	1.393	1.728
	December	0.927	1.164	1.442

Exposure (baseline)		Lower 2.5%	Median	Upper 97.5%
Month of last recording (December)	January	6.514	7.973	9.786
	February	6.234	7.691	9.450
	March	8.045	9.905	12.158
	April	7.207	8.962	11.090
	May	6.315	7.885	9.786
	June	5.140	6.398	7.885
	July	4.595	5.568	6.686
	August	4.154	4.968	5.918
	September	2.214	2.625	3.096
	October	2.140	2.479	2.875
	November	1.539	1.791	2.080
ln AFC[64] (6.70)		1.753	2.275	2.927
[ln interval[65]]^1 (2.28)		1.260	1.363	1.473
[ln interval]^2 (2.28)		1.849	1.972	2.102
[ln interval]^3 (2.28)		1.198	1.247	1.299
DIM[66] (< 100 days (d))	100 to 199 d	2.654	2.939	3.267
	200 to 304 d	5.291	5.900	6.567

[60] Discrete time logistic survival models for cow disposal in any 50 day interval from first calving including SCC1 only. Estimates based on 10,000 simulations.
[61] First test day somatic cell count record between 5 and 30 days in milk (DIM) in parity 1.
[62] First test day milk yield record (kg) between 5 and 30 DIM in parity 1.
[63] First test day fat record (proportion) between 5 and 30 DIM in parity 1.
[64] Age at first calving (days).
[65] 50 day intervals from first calving. Included as polynomials.
[66] Days in milk (DIM) category in the penultimate interval for each cow. Missing category not shown.

Table 5.3 continued. Model 5.2[67]; 95% Bayesian credible intervals for the odds ratios for cow disposal from 812 Irish dairy herds

Exposure (baseline)		Lower 2.5%	Median	Upper 97.5%
Intercept		0.002	0.002	0.003
ln SCC1 (4.64)		1.010	1.043	1.075
TDY1 (23 kg)		0.971	0.978	0.986
TDF1 (0.04)		0.000	0.001	0.126
Month of first calving	January	0.897	1.010	1.138
(February)	March	1.106	1.213	1.337
	April	1.110	1.264	1.433
	May	1.176	1.417	1.699
	June	0.873	1.137	1.459
	July	0.911	1.400	2.086
	August	1.021	1.535	2.270
	September	1.197	1.490	1.848
	October	1.075	1.319	1.611
	November	1.134	1.405	1.737
	December	0.935	1.166	1.458
ln AFC (6.70)		1.752	2.263	2.907
Month of last recording	January	6.554	8.037	9.885
(December)	February	6.284	7.714	9.516
	March	8.125	9.924	12.170
	April	7.272	8.971	11.067
	May	11.067	7.846	9.757
	June	5.155	6.398	7.909
	July	4.641	5.590	6.746
	August	4.233	5.033	5.983
	September	2.235	2.636	3.114
	October	2.157	2.485	2.883
	November	1.558	1.801	2.083
DIM (< 100 d)	100 to 199 d	2.336	2.615	2.907
	200 to 304 d	4.250	4.811	5.452
ln SCC (< 4 /mL)	4 to < 5 /mL	0.993	1.101	1.223
	≥ 5 /mL	1.132	1.258	1.401
TDY (< 20 kg)	20 to < 30 kg	0.897	0.976	1.062
ln SCC (< 4 /mL)	4 to < 5 /mL	0.993	1.101	1.223
	≥ 5 /mL	1.132	1.258	1.401
[ln interval[68]]^1 (2.28)		1.299	1.412	1.526
[ln interval]^2 (2.28)		1.809	1.923	2.057
[ln interval]^3 (2.28)		1.182	1.229	1.282

[67] Discrete time logistic survival models for cow disposal in any 50 day interval from first calving including SCC1 and further time-varying covariates. Estimates based on 10,000 simulations
[68] 50 day intervals from first calving. Included as polynomials.

Table 5.4. Random effect variances from final models[69]; 95% Bayesian credible intervals for ln[70] odds of cow disposal from 812 Irish dairy herds, based on 10,000 simulations

	Model 5.1			Model 5.2		
	Lower 2.5%	Median	Upper 97.5%	Lower 2.5%	Median	Upper 97.5%
Cow level	0.0003	0.0009	0.009	0.0005	0.001	0.003
Herd level	0.225	0.284	0.352	0.233	0.291	0.356

[69] Discrete time logistic survival model for cow disposal in any 50 day interval from first calving.
[70] Natural logarithm of.

5.3.3 Inclusion of time-varying covariates

Cows in late lactation had higher median odds of disposal in the subsequent 50 day interval (Table 5.3); median odds ratios were 2.9 (95% BCI; 2.65 to 3.27), and 5.9 (95% BCI; 5.29 to 6.57) for those cows 100 to 199 DIM, and over 199 DIM respectively, compared to those < 100 DIM (Model 5.1; Table 5.3). With the other time-varying covariates added (Model 5.2; Table 5.3), results were similar, and cows in late lactation also had higher odds of disposal in the subsequent 50 day interval; median odds ratios were 2.6 (95% BCI; 2.34 to 2.91), and 4.8 (95% BCI; 4.25 to 5.45) for those cows 100 to 199 DIM, and over 199 DIM respectively, compared to those < 100 DIM. In Model 5.2, the time-varying SCC categories; 55,000 to 147,000 cells/mL, and ≥ 148,000 cells/mL were associated with 10% (MOR = 1.10, (95% BCI; 1.00 to 1.22)) and 26% (MOR = 1.26, (95% BCI; 1.13 to 1.40)) increased odds of disposal in the subsequent 50 day interval respectively, compared to cows with SCC < 55,000 cells/mL. With these time-varying SCC categories added, the strength of association (MOR) between ln SCC1 and disposal decreased by 0.9% compared to Model 5.1, indicating that part of this impact is mediated through an association with SCC at later recordings (Table 5.3). Time-varying covariates for TDY were associated with 2% (MOR = 0.98 (95% BCI; 0.90 to

1.82)) and 38% (MOR = 0.62 (95% BCI; 0.53 to 0.73) decreased odds of disposal in the subsequent 50 day interval for cows with test day milk yield (TDY) of 20 to < 30 kg, and ≥ 30 kg respectively, compared to cows with TDY < 20 kg. Association of TDY1 with disposal from the herd was unchanged. Two models are presented, because the impact of time-varying covariates in Model 5.2 is useful to identify possible reasons for disposal of particular cows (Table 5.3). However, the main aim of the research was to evaluate the impact of SCC1 on cow disposal risk, specifically to focus on information available by 30 DIM in parity 1. It was therefore decided that further predictions and simulations would be based on Model 5.1.

5.3.4 Model assessment

To demonstrate the internal fit of Model 5.1 (Table 5.3) to sample_1, posterior predicted risk of disposal by interval, is shown in Figure 5.2. The observed data had a cyclical pattern, with higher risk of disposal in particular intervals. Although the 50 day time intervals relate to cow-time from the date of first calving, the majority of cows in the dataset calved in spring (February to April), and thus cyclicity occurred because of an increased risk of disposal each autumn, when cows were in late lactation. A time-varying term for DIM was required to improve model fit to the observed data, although small discrepancies remained for certain intervals. Categorical terms for 7 intervals were added to the model which improved the fit, shown as the alternative model (Figure 5.2); however odds ratio distributions were not deemed to change by a meaningful extent (< 1% difference in MOR and limits of the 95% BCI), and the parsimonious model was retained. Predictions of disposal risk by

month of first calving, and month of last recording in sample_1 indicated good model fit (not shown).

In terms of the usefulness of Model 5.1, there was close agreement between predicted and observed disposal risk for cows grouped by SCC1, by BMSCC (Figure 5.3), and by AFC (not shown). Within model fit was good, as observed values were within the 95% BCI of predictions. This was also the case on cross validation, and these results indicate that in terms of SCC1, BMSCC, and AFC, Model 5.1 appeared to be generalisable to other Irish herds.

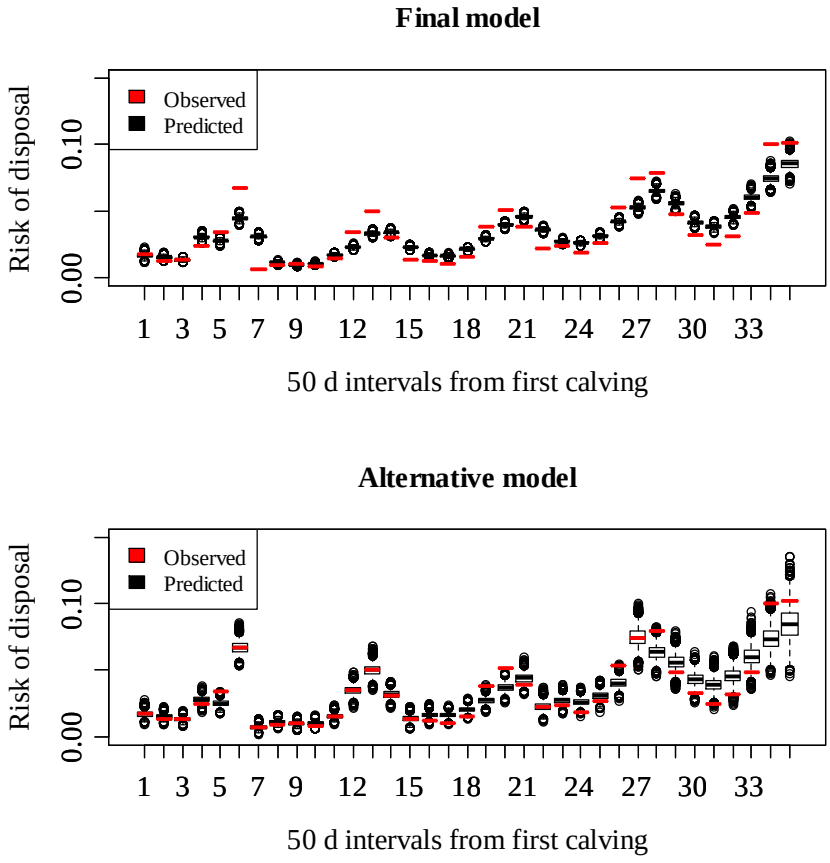

Figure 5.2. Predictions from 10,000 simulations of the final and alternative[71] versions of Model 5.1[72] to assess internal fit; disposal risk in each 50 day (d) interval from first calving, and observed values in 812 Irish dairy herds used for model development

[71] The alternative model includes 7 additional categorical terms to improve fit to observed data.
[72] Discrete time logistic survival model for cow disposal in any 50 day interval from first calving.

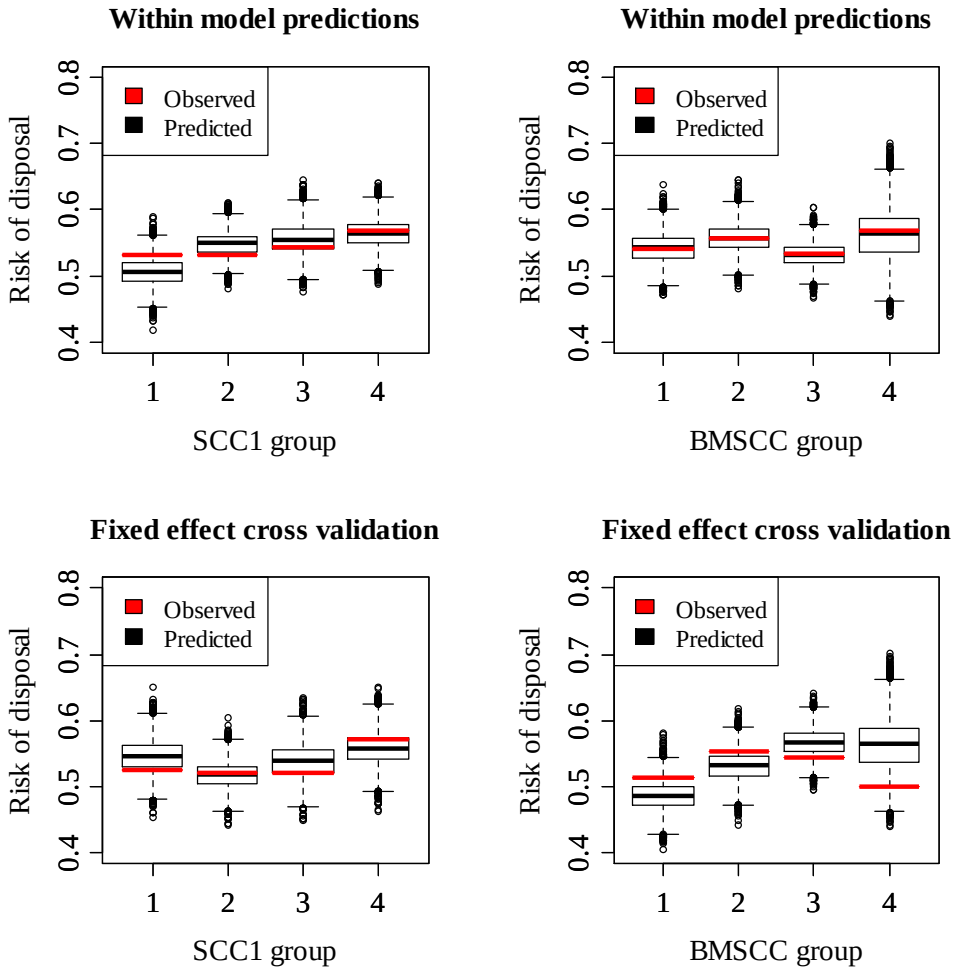

Figure 5.3. Model 5.1[73] predictions of disposal risk from 10,000 simulations, and observed values in 812 Irish dairy herds used for model development to assess model fit, and in 808 separate Irish dairy herds used for cross validation[74], grouped by milk somatic cell count at 5 to 30 days in milk during parity 1 (SCC1)[75] and estimated bulk milk somatic cell count (BMSCC) group[76]

[73] Discrete time logistic survival model for cow disposal in any 50 day interval from first calving.
[74] Using fixed effects from the model. Indicates results can be generalized to other Irish dairy herds.
[75] Group 1; < 50,000 cells/mL, group 2; 50,000 to 99,000 cells/mL, group 3; 100,000 to 164,000 cells/mL, group 4; ≥ 164,000 cells/mL.
[76] Geometric mean BMSCC estimated from cow test day SCC, and milk records. Group 1; < 200,000 cells/mL, group 2; 200,000 to 249,000 cells/mL, group 3; 250,000 to 399,000 cells/mL, group 4; > 399,000 cells/mL.

5.3.5 Micro-simulation of baseline disposal risks

Results based on Model 5.1 are presented graphically; the relationship between risk of disposal from the herd, and SCC1 is shown for cows in different stages of lactation (Figure 5.4). Regression lines (on a ln scale), had slopes of 0.0011, 0.00061, and 0.0031, and intercepts of 0.0022, 0.013, and 0.0064 for cows that were < 100, 100 to 199, and 200 to 304 DIM respectively.

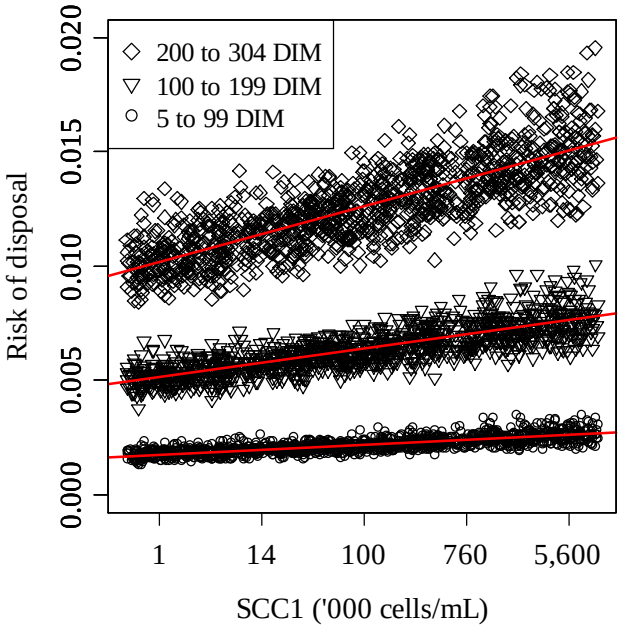

Figure 5.4. Scatter plot and regression lines from 1,000 simulations of Model 5.1[77]; posterior predicted risk of disposal between 450 and 500 days from first calving, against milk somatic cell count at 5 to 30 days in milk (SCC1)[78] for cows at different stages of lactation

[77] Discrete time logistic survival model for cow disposal in any 50 day interval from first calving.
[78] For cows with a first calving in February, aged 24 months, that produced 23 kg/day of milk with 4% fat between 5 and 30 days in milk, and had their last recording in December.

5.3.6 Micro-simulation of herd scenarios

The posterior probability of disposal was greater for cows in herds ≥ 20% initial prevalence of cows with SCC1 ≥ 400,000 cells/mL, compared to the same herds after a reduction in prevalence to < 5%, for 65%, 68%, 74%, 75%, and 73% of simulations within 350, 700, 1,050, 1,400, and 1,750 days from first calving respectively (Figure 5.5). Figure 5.6 shows the estimated probability of different levels of potential savings /heifer in the herd attributable to reduced replacement costs within 1,750 days of first calving, for various herd level reductions in the prevalence of cows with SCC1 ≥ 400,000 cells/mL. Herds with ≥ 20% prevalence of heifers with SCC1 ≥ 400,000 cells/mL had 54% probability of a cost saving ≥ €10 / heifer in the herd through reducing the prevalence of heifers with SCC1 ≥ 400,000 cells/mL to < 10% (Figure 5.6). For an example herd that calves 20 heifers /year, this equates to a saving of €200 /year through decreased replacement rate; further scenarios for the example herd are given in Table 5.5. When only the first 350 days from first calving (first lactation) are included in the economic simulation, the results are ostensibly the same, indicating that at herd level, the impact of SCC1 on disposal risk is greater over a shorter time period, or conversely, other reasons for disposal become more important as the time period considered increases.

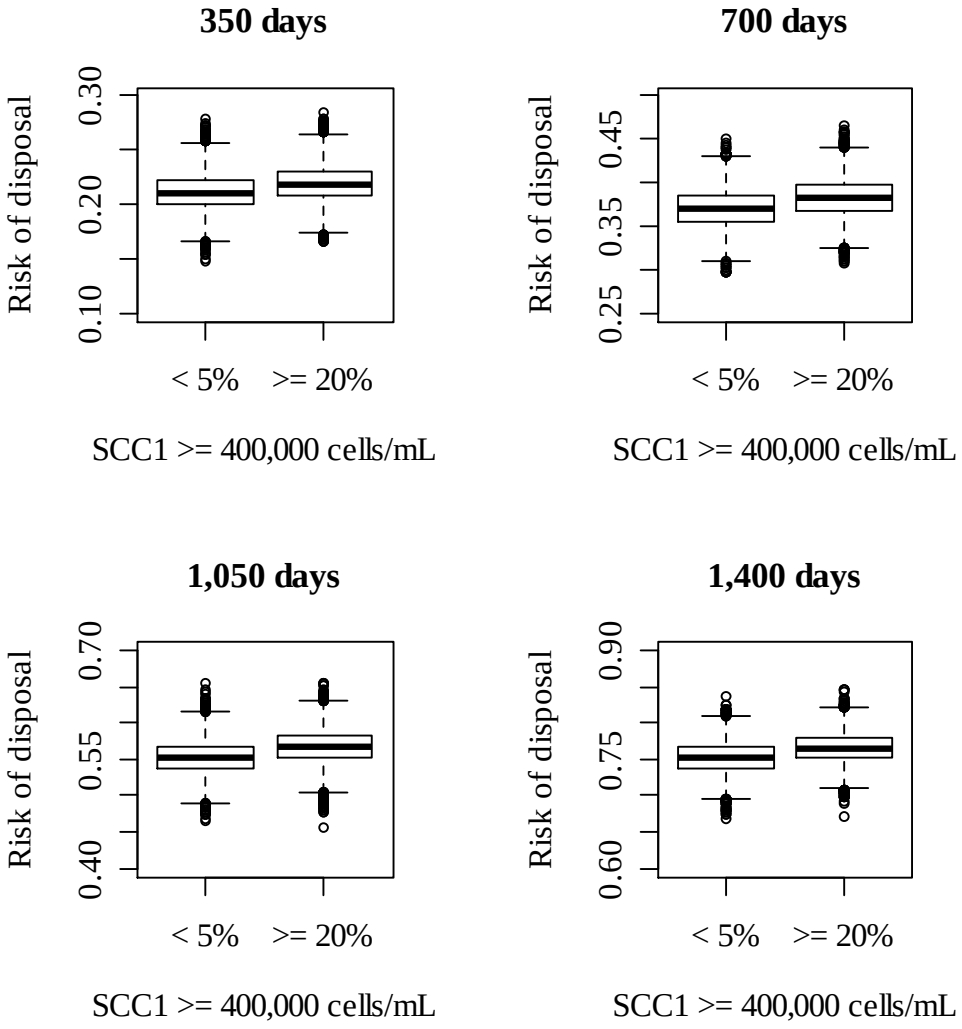

Figure 5.5. Micro-simulation over 10,000 simulations of Model 5.1[79]; cow disposal risk within 350, 700, 1,050, and 1,400 days from first calving in herds with an initial prevalence of cows with milk somatic cell count at 5 to 30 days in milk during parity 1 (SCC1) ≥ 400,000 cells/mL of ≥ 20% and following a reduction to < 5%

[79] Discrete time logistic survival model for cow disposal in any 50 day interval from first calving.

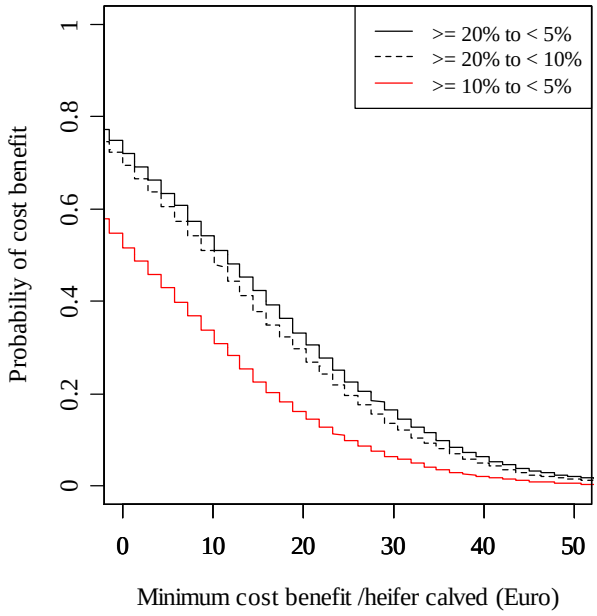

Figure 5.6. Micro-simulation over 10,000 simulations of Model 5.1[80]; minimum cost saving per heifer calved attributable to reduction in replacement costs[81] over 1,750 days from first calving, for changes in the herd level prevalence of cows with SCC at 5 to 30 days in milk during parity 1 ≥ 400,000 cells/mL

[80] Discrete time logistic survival model for cow disposal in any 50 day interval from first calving.
[81] At €1,451 /cow replaced.

Table 5.5. Model 5.1[82] predictions for an example herd that calves 20 heifers /year; probability of annual savings through decreased replacement costs within 1,750 days of first calving associated with reductions in the number of heifers with milk somatic cell count ≥ 400,000 cells/mL between 5 and 30 days in milk

Saving (€)	Change in number of parity 1 cows (/20)		
	≥ 4 to ≤ 1	≥ 4 to 0	≥ 2 to 0
≥ 0	0.68	0.70	0.52
≥ 200	0.54	0.55	0.38
≥ 400	0.40	0.41	0.24

[82] Discrete time logistic survival model for cow disposal in any 50 day interval from first calving.

5.4 Discussion

SCC in the first month of lactation during parity 1 was positively associated with risk of disposal from the herd, although the size of this effect appeared relatively small, and was therefore of limited financial importance. A possible reason for this is that in practice other considerations have an overriding influence on cow disposal decisions. The impact of time-varying covariates in explaining cow disposal risk was demonstrated (Model 5.2), and this emphasises the importance of recent health, and production records in making disposal decisions. In seasonally calving herds, those cows not pregnant at the end of the breeding season may be at higher disposal risk at the end of lactation (Pinedo et al., 2010), and in some herds this may limit the number of cows that can be removed for other reasons. Herd circumstances, such as the availability of replacement heifers, and space in the dairy unit may also influence disposal decisions (Lehenbauer and Oltjen, 1998), and for European Union herds, milk quota availability may also require consideration. In this research more variation in cow disposal risk was identified between herds, than within herds, indicating that decisions on cow disposal do appear to be herd specific (Weigel et al., 2003), and may therefore reflect the underlying management objectives, or other farm factors such as disease incidence.

When the cost of potential interventions are considered, as a result of the small effect size, and uncertainty in the outcome, reductions in the prevalence of cows with SCC1 $\geq$ 400,000 cells/mL appear only marginally beneficial in terms of reduced disposal costs for less than 1 in 5 Irish dairy herds with a prevalence $\geq$ 20% (Table 5.1). However this judgement depends on decision

makers' attitude to risk (Figure 5.6), and hence how much uncertainty in a particular outcome they are comfortable with. If control measures to reduce SCC1 were to be considered, they should focus on the *pre-* and *peri-partum* period (Green et al., 2007b; 2008). However, chapter 3 shows the importance of considering the impact of SCC1 on lifetime milk yield before the cost-effectiveness of specific interventions can be properly evaluated. There may also be additional benefit through reduced incidence of clinical mastitis in particular herds.

This chapter highlights the usefulness, and importance of generating predictions from statistical models to show the impact of results, because 'significant' findings may not be biologically, or economically meaningful when considered in context. Use of MCMC for predictions allows variability in parameters to be included, and therefore the full uncertainty in possible outcomes, as well as the central tendency can be explored. For example, a conventional approach may base conclusions on the mean effect of SCC in early lactation on culling risk (De Vliegher et al., 2005b), but variation in model parameters can affect the inference from these results (Green et al., 2010). The mean association of SCC early in the first lactation, and disposal in this research (Table 5.3) was less than that previously observed based on recorded culling dates over the first lactation (De Vliegher et al., 2005b). However cows in our study were followed up for over 5 years, and up to a maximum of 6 lactations, and therefore the impact of SCC1 on disposal risk over a longer time period was less. This was also shown by introducing time-varying covariates for SCC (Table 5.3), and the associated reduction in the

coefficient for SCC1, which was consistent with previous work (De Vliegher et al., 2005b).

With a more specific outcome definition (culling for udder health reasons only), strength of association with SCC early in the first lactation increased 3 fold (De Vliegher et al., 2005b), emphasising the importance of how an outcome is defined, although definitions, and reliability of recording may also vary between herds. In the current study it was assumed that cows were disposed when recordings ceased (unless censored), although in reality it would likely be after this, because of the logistics of economic carcase salvage. Despite this, trends in cow disposal appeared consistent with previous studies, indicating this was a reasonable proxy for culling. Specifically, cows in late lactation were at higher risk of disposal (Rajala-Schultz and Gröhn, 1999), which varied seasonally (Anderson, 1985; Crosse and O' Donovan, 1989), and this could relate to an overall increased risk of disposal in the autumn for those cows in spring-calving herds that failed to conceive (Pinedo et al., 2010). Despite this, increasing milk yield decreased disposal risk (Beaudeau et al., 1994; Rajala-Schultz and Gröhn, 1999).

Assessment of model fit, for logistic regression models is not straight forward, and is often neglected (Green et al., 2009). Demonstrating the extent to which models are useful, rather than simply ‘correctness’ has been proposed as a rational approach to model assessment (Gelman et al., 1996). In this research, assessments of Model 5.1 based on aggregated predictions in groups of magnitude of SCC1 suggested that predictions were reliable, and were likely generalisable to other Irish herds. This permitted use of a micro-simulation procedure in order to present the study results in a meaningful context.

5.5 Conclusions

Despite negative association of SCC between 5 and 30 DIM during parity 1 (SCC1) and longevity, the effect was small, and therefore unlikely to be economically important when considered in isolation. Economic evaluation of potential savings attributable to reducing the prevalence of cows with high SCC1 should also therefore consider lifetime milk yield as shown in chapter 3.

Chapter 6: Association between somatic cell count during the first lactation and the cumulative milk yield of cows in Irish dairy herds

6.1 Introduction

In chapters 3 and 5, increased somatic cell count between 5 and 30 days in milk during parity 1 (SCC1) was reported to have a negative impact on both cumulative milk yield, and risk of disposal for cows in Irish dairy herds. Early lactation milk somatic cell count (SCC) in heifers is considered a reflection of the adequacy of control measures during the ppp period (De Vliegher et al., 2012), and improving management for ppp heifers to reduce the prevalence of cows with SCC1 $\geq$ 400,000 cells/mL would be expected to have an economically important impact on lifetime milk yield (chapter 3). In Belgian heifers, increased SCC early in the first lactation has been associated with increased SCC at subsequent test days throughout the first lactation (De Vliegher et al., 2004a). For cows that survive, SCC beyond 'early lactation' therefore gives information on the legacy of intramammary infection (IMI) from the ppp period, as well as IMI originating while heifers are in milk. A negative relationship between geometric mean first lactation SCC, and cumulative first lactation milk yield has been reported (Hortet and Seegers, 1998; Raubertas and Shook, 1982). However no studies have investigated the association between numeric summaries of first lactation SCC, and cumulative milk yield beyond the first lactation. Furthermore, the impact of SCC1 and SCC over the entire first lactation on cumulative milk yield has not been compared in the same study, and the association between SCC1 and SCC

throughout the entire first lactation has not been investigated for cows in Irish dairy herds. These relationships will help understand the relative importance of the ppp, and lactating period for the control of heifer mastitis.

The aims of this chapter were twofold. Firstly, to compare associations between SCC1, and SCC throughout the entire first lactation, on cumulative milk yield over both the first lactation and the subsequent lifetime of cows in Irish dairy herds. Micro-simulation was then used to show the financial impact of herd level reductions in the prevalence of cows with high SCC during the first lactation in terms of lifetime milk yield. The second aim was to assess the association between SCC1 and SCC throughout the entire first lactation of cows in Irish dairy herds.

6.2 Materials and methods

6.2.1 Data selection

The data selection procedure used in chapter 3 was extended to include only cows with ≥ 2 SCC recordings during parity 1 (the first at 5 to 30 days in milk (DIM) between January 2005 and March 2007); 51,483 cows in 5,900 Irish dairy herds were therefore available for analysis. Cumulative milk yields for all lactations up to July 25, 2012 were calculated using a recognised method (Olori et al., 1999), and provided by ICBF. Lactation milk yields were summed for each cow to give an estimate of 'lifetime milk yield' from the date of first calving, to the end of the study period. Number of cows, first lactation SCC parameters, proportion of cows with SCC1 ≥ 400,000 cells/mL, number of recordings in the first lactation, proportion of cows surviving the first and

fourth lactation, and first lactation and lifetime milk yield were summarised for a subset of 5,413 herds (with ≥ 2 eligible cows /herd) that was split into quartiles based on herd level geometric mean first lactation SCC (herd_gSCC_p1; quartile 1; < 72,000 cells/mL, quartile 2; 72,000 to 93,000 cells/mL, quartile 3; 94,000 to 119,000 cells/mL, quartile 4; ≥ 120,000 cells/mL).

6.2.2 First lactation somatic cell count and cumulative milk yield; statistical analysis

For comparison, the outcomes of interest (y_{ij}) were 1) First lactation milk yield (FLMY), or 2) Lifetime milk yield (LiMY), for the ith cow, in the jth herd. Random effects models were developed that took the form;

$$y_{ij} = \alpha + \mathbf{X_{ij}}\,\boldsymbol{\beta_1} + \mathbf{X_j}\,\boldsymbol{\beta_2} + u_j + e_{ij},$$

$$u_j \sim \text{Normal}\,(0, \sigma^2_u),$$

$$e_{ij} \sim \text{Normal}\,(0, \sigma^2_e),$$

where α = intercept value, $\mathbf{X_{ij}}$ = matrix of exposure variables for each cow, $\boldsymbol{\beta_1}$ = vector of coefficients for $\mathbf{X_{ij}}$, $\mathbf{X_j}$ = matrix of exposure variables for each herd, $\boldsymbol{\beta_2}$ = vector of coefficients for $\mathbf{X_j}$, u_j = a random effect to account for residual variation between herds (assumed to be normally distributed with mean = 0, and variance = σ^2_u), and e_{ij} = residual level 1 error (assumed to be normally distributed with mean = 0, and variance = σ^2_e). To determine the relative importance of SCC early in the first lactation, compared to SCC over the entire first lactation in terms of future milk yield, separate models for FLMY and LiMY were developed in which the exposure of interest was ln SCC1. SCC variables and age at first calving (AFC) were investigated for inclusion as

polynomial terms (to powers of 5) on a ln scale to account for non-linear associations with cumulative milk yield. Month and year of first calving were investigated for inclusion as linear or categorical terms. Biologically plausible interactions, and herd level random slopes (herd x fixed effect interactions) were assessed. Initial values for all covariates were generated in MLwiN (Rasbash et al., 2009), with the iterative generalized least squares procedure (Goldstein, 2003). To facilitate posterior predictions from the models that incorporated all uncertainty in parameters, the models for FLMY and LiMY were developed in a Bayesian framework using WinBUGS 1.4.3 (Lunn et al., 2000). Parameters were estimated from 10,000 MCMC simulations, following a burn in of 1,000 simulations during which time chain convergence occurred, determined by visual inspection of 3 chains to ensure a stationary distribution had been reached (Gilks et al., 1996). Vague prior distributions were used for the random effect variances; $\sigma^{-2}_{u} \sim$ Gamma (0.001, 0.001), $\sigma^{-2}_{e} \sim$ Gamma (0.001, 0.001), and $\boldsymbol{\beta} \sim$ Normal (0, 10^{6}), to give the major influence to the data in the estimation of parameters (Green et al., 2004). Distributions of covariates, and interaction terms were inspected; these remained in the model based on biological plausibility, and if the 95% Bayesian credible interval (BCI) excluded 0. Sensitivity of the results to prior distributions for the herd level random effect variance (Spiegelhalter et al., 2004) was evaluated by repeating simulations using the prior; $\sigma^{2}_{u} \sim$ Uniform (10^{-9}, 10^{9}).

6.2.2.1 Model checking

To evaluate model fit and usefulness, fixed and random effects were used to predict cow FLMY and LiMY ($y.pred_{ij}$) thus (Gelman et al., 1996);

$$y.pred_{ij} \sim p(y.pred_{ij} \mid \boldsymbol{\beta}, data, u_j)$$

where *p* represents a conditional probability distribution, $y.pred_{ij}$ are posterior predictions of cumulative milk yield for the ith cow in the jth herd, $\boldsymbol{\beta}$ is the vector of model coefficient distributions, and u_j is the random effect for the jth herd. Predicted and observed mean FLMY and LiMY were calculated at the cow level for quartiles of cows categorized by geometric mean of first lactation SCC (quartile 1; < 55,000 cells/mL, quartile 2; 55,000 to 90,000 cells/mL, quartile 3; 91,000 to 149,000 cells/mL, quartile 4 ≥ 150,000 cells/mL), or grouped by SCC1 (group 1; < 55,000 cells/mL, group 2; 55,000 to 149,000 cells/mL, group 3; 150,000 to 399,000 cells/mL, group 4; ≥ 400,000 cells/mL); these categories were not in the final models. Posterior predicted distributions of mean cumulative milk yield for cows in these groups were inspected to determine if the observed values were within the 95% BCI of the posterior predictions, as an indication of internal model fit and usefulness (Gelman et al., 1996).

6.2.2.2 Micro-simulation

Management changes to improve mastitis have an impact on the whole herd rather than individual cows. Therefore, to illustrate the potential impact of reductions in herd_gSCC_p1 on the mean LiMY of cows, and to demonstrate financial relevance micro-simulation was carried out as was conducted in chapters 3 to 5 for herd level reductions in the prevalence of cows with high SCC1. The Bayesian model was therefore extended to include a one-step micro-simulation of LiMY for 1,000 simulated cows with different characteristics, based on herd_gSCC_p1 quartile. Increase in the mean and

variance of ln SCC for first lactation cows by herd was associated with increase in the between herd variance of these parameters. For each simulated cow, values for the mean and variance of ln SCC over the first lactation were therefore drawn from normal distributions based on the observed data, to give a realistic distribution of values. In order to demonstrate the impact of an achievable reduction in herd_gSCC_p1 on LiMY, herds with herd_gSCC_p1 in quartile 4 were assumed to move to quartiles 1 or 2, and herds in quartile 3 were assumed to move to quartile 1. For ease of comparison, all simulated cows were assumed to have a first calving date in February 2007. At each of 10,000 MCMC simulations (following a burn in of 1,000), final model coefficients were combined with data from the simulated cows to generate predictions of lifetime milk yield for the ith cow in the jth herd ($y.pred_{ij}$);

$$y.pred_{ij} \sim p(y.pred_{ij}| \boldsymbol{\beta}, X^{sim}),$$

where $\boldsymbol{\beta}$ is a vector of model coefficient distributions, and X^{sim} is a matrix of data for simulated cows, including simulated values for the mean and variance of ln SCC over the first lactation, based on herd_gSCC_p1 quartile, and indicator variables to denote a first calving in February 2007.

The mean LiMY for simulated cows in each herd scenario was calculated following each MCMC simulation. Differences in mean LiMY were multiplied by an estimated gross margin (milk price – variable costs of production), that was drawn from a normal distribution with mean = 0.17 €/L, and standard deviation = 0.03 €/L for each cow (Hennessy et al., 2011), to give the difference in expected revenue associated with reductions in herd_gSCC_p1. Posterior distributions of mean savings /heifer calved into the herd were plotted

as a cumulative probability distribution to show the probability of different levels of financial return for changes in herd_gSCC_p1. An example of the model code is given in the appendix.

6.2.3 Somatic cell count legacy during the first lactation; statistical analysis

Proportions of cows in each SCC1 group and first lactation geometric mean quartile were determined. The association between ln SCC1, and the subsequent mean and variance of ln SCC during the first lactation was of interest, to determine the possible legacy of IMI in early lactation. As the mean and variance of ln SCC during the first lactation were positively associated, the related responses; mean and variance of ln SCC during the entire first lactation, for the jth cow in the kth herd ($Resp_{ijk}$) were analysed in the following bivariate linear model (Rasbash et al., 2009);

$$Resp_{ijk} = (\beta_0 + v_{0k} + u_{0jk}).z_{0jk} + (\beta_1 + v_{1k} + u_{1jk}).z_{1jk}$$

$$+ (\boldsymbol{\beta_2} + v_{2k}).z_{0jk}.\mathbf{X_{jk}} + (\boldsymbol{\beta_3} + v_{3k}).z_{1jk}.\mathbf{X_{jk}}$$

$$\begin{bmatrix} v_{0k} \\ v_{1k} \\ v_{2k} \\ v_{3k} \end{bmatrix} \sim \text{Normal}\,(0, \sum_v),\ \sum_v = \begin{bmatrix} \sigma^2_{v0} & & & \\ \sigma^2_{v01} & \sigma^2_{v1} & & \\ \sigma^2_{v011} & \sigma^2_{v12} & \sigma^2_{v2} & \\ \sigma^2_{v01112} & \sigma^2_{v122} & \sigma^2_{v23} & \sigma^2_{v3} \end{bmatrix}$$

$$\begin{bmatrix} u_{0jk} \\ u_{1jk} \end{bmatrix} \sim \text{Normal}\,(0, \sum_u),\ \sum_u = \begin{bmatrix} \sigma^2_{u0} & \\ \sigma^2_{u01} & \sigma^2_{u1} \end{bmatrix}$$

where z_{0jk}, and z_{1jk} are binary response indicator variables for mean and variance of ln SCC during the first lactation respectively for the jth cow in the kth herd. The model intercepts β_0, and β_1 for the mean and variance of first lactation ln SCC respectively, were allowed to vary randomly to account for

lack of independence between cows (u_{0k}, and u_{1k}), and herds (v_{0k}, and v_{1k}). Exposure variables for each cow (matrix $\mathbf{X_{jk}}$) had corresponding vectors of coefficients $\boldsymbol{\beta_2}$ and $\boldsymbol{\beta_3}$ for the mean and variance of first lactation ln SCC respectively which could vary randomly at the herd level, as defined by v_{2k}, and v_{3k}. Cow level random effects (u_{0k}, and u_{1k}) were assumed to have a multivariate normal distribution with mean = 0, and covariance matrix = $\sum_u$, consisting of variances for the mean and variance of ln SCC throughout the first lactation; σ^2_{u0}, and σ^2_{u1} respectively, and their covariance σ^2_{u01}. The herd level random effects covariance matrix ($\sum_v$) had an expanded structure to include variances for random coefficients, in addition to the intercepts, and hence additional covariance terms. The model was fitted using MCMC in MLwiN (Browne, 2012), with vague prior distributions for $\sigma^{-2}_{vi} \sim$ Gamma (0.001, 0.001), $\sigma^{-2}_{ui} \sim$ Gamma (0.001, 0.001), and $\boldsymbol{\beta} \sim$ Normal (0, 10^6). SCC1 was the exposure of interest and this was included as ln scale polynomials. In order for the model to be useful for predictions of the mean and variance of first lactation ln SCC by 30 DIM, only confounding variables deemed to be operating by 30 DIM were assessed. These were month of first calving, AFC, and DIM at the first recording. Biologically plausible interactions and herd level random slopes (for SCC1) were investigated for inclusion. Parameters were included in the model if the 95% BCI excluded 0, and there was a reduction in the deviance information criteria (Spiegelhalter et al., 2002). Convergence was assessed by inspection of chains to ensure a stationary distribution had been reached (Browne, 2012), and model fit was assessed by checking distributions of cow and herd level mean residuals for normality (Goldstein, 2003).

6.3 Results

6.3.1 Descriptive results

Lifetime milk yield (LiMY) for all 51,483 cows was evaluated over maximum follow up times between 5.3 and 7.5 years. Descriptive statistics, grouped by herd_gSCC_p1 quartile are shown in Table 6.1. There were trends for decreased FLMY, LiMY, and proportions of cows surviving with increasing herd_gSCC_p1. These changes were associated with an increase in the herd level proportion of recordings with high SCC, both at 5 to 30 DIM (≥ 400,000 cells/mL), and throughout the first lactation (≥ 400,000 and ≥ 200,000 cells/mL), and also increased variability in these proportions between herds (Table 6.1). Increasing herd_gSCC_p1 was associated with increasing variance in the mean of ln SCC for cows over the first lactation both between and within herds (Table 6.1). Forty six per cent of cows had SCC1 < 150,000 cells/mL and geometric mean first lactation SCC ≤ 90,000 cells/mL, and only 5% of cows had SCC1 ≥ 150,000 cells/mL and geometric mean first lactation SCC ≤ 90,000 cells/mL (Table 6.2). Twenty four per cent of cows had SCC1 < 150,000 cells/mL, and geometric mean first lactation SCC > 90,000 cells/mL (Table 6.2). Similarly, 25% of cows had SCC1 ≥ 150,000 cells/mL, and geometric mean first lactation SCC > 90,000 cells/mL (Table 6.2).

Table 6.1. Descriptive data from 5,413 Irish herds categorised by first lactation geometric mean SCC, and for 50,996 parity 1 cows in these herds

	Herd level first lactation geometric mean SCC (cells/mL)			
	< 72,000	72,000 to 93,000	94,000 to 119,000	≥ 120,000
Quartile of herds	Best 25%	Middle 50%		Worst 25%
Total number of cows	11,709	14,172	13,516	11,599
Number of recordings in the first lactation (interquartile range (IQR))	7 (5 to 9)	7 (5 to 9)	7 (5 to 9)	7 (5 to 8)
Proportion of cows that survive the first lactation	0.83	0.84	0.84	0.81
Proportion of cows that survive the fourth lactation	0.36	0.37	0.36	0.34
Median lifetime milk yield (IQR) (tonne)	24.0 (12.0 to 36.7)	23.2 (12.0 to 35.8)	23.1 (12.0 to 35.6)	21.0 (11.0 to 33.2)
Median first lactation milk yield (IQR) (tonne)	5.3 (4.2 to 7.3)	5.2 (4.1 to 6.9)	5.2 (4.0 to 7.0)	5.0 (3.8 to 6.8)
Mean of the natural logarithm of (ln) SCC over the first lactation for cows in the mean herd (between herd variance)	4.01 (0.37)	4.42 (0.42)	4.67 (0.45)	5.07 (0.61)
Variance of ln SCC over the first lactation for cows in the mean herd (between herd variance)	0.58 (0.96)	0.60 (0.96)	0.61 (1.22)	0.69 (1.49)
Proportion of recordings ≥ 200,000 cells/mL during the first lactation for cows in the median herd (interquartile range between herds)	0.08 (0.04 to 0.12)	0.14 (0.10 to 0.19)	0.21 (0.16 to 0.25)	0.33 (0.27 to 0.41)
Proportion of recordings ≥ 400,000 cells/mL during the first lactation for cows in the median herd (interquartile range between herds)	0.02 (0.00 to 0.05)	0.05 (0.02 to 0.08)	0.08 (0.04 to 0.11)	0.15 (0.10 to 0.21)
Proportion of cows with SCC1_hi[83] in the median herd (interquartile range between herds)	0.00 (0.00 to 0.10)	0.07 (0.00 to 0.17)	0.11 (0.00 to 0.20)	0.19 (0.00 to 0.31)

[83] Somatic cell count (SCC) ≥ 400,000 cells/mL between 5 and 30 DIM during parity 1.

Table 6.2. Proportion of 51,483 Irish dairy cows in each SCC1 group[84] and first lactation geometric mean somatic cell count (SCC) quartile

		First lactation geometric mean SCC quartile (thousand cells/mL)				
		< 55	55 to 90	91 to 149	≥ 150	Total
SCC1 group (thousand cells/mL)	< 55	0.15	0.09	0.04	0.01	0.29
	55 to 149	0.07	0.15	0.13	0.06	0.41
	150 to 399	0.01	0.03	0.06	0.08	0.18
	≥ 400	0.00	0.01	0.03	0.08	0.12
	Total	0.23	0.28	0.26	0.23	1.00

[84] SCC between 5 and 30 days in milk during parity 1 (SCC1).

6.3.2 First lactation Somatic cell count and cumulative milk yield; model results

6.3.2.1 Outcome 1; first lactation milk yield

The final models for FLMY (Models 6.1 and 6.2; Table 6.3) accounted for month, and AFC. Cows that calved in June 2007, aged 27 months were used as the baseline for comparison. In Model 6.1, a 1-unit increase in mean ln SCC over the first lactation (for example an increase in first lactation geometric mean SCC from 50,000 to 150,000 cells/mL, or 150,000 cells/ml to 400,000 cells/mL) was associated with a median decrease in FLMY of 135 kg (95% BCI 108 to 163 kg). Variance in ln SCC over the first lactation was not associated with changes in FLMY. There was an interaction between ln SCC over the first lactation and AFC, and FLMY decreased with decreasing AFC (Figure 6.1). In Model 6.2 a 1-unit increase in ln SCC1 was associated with a median decrease in FLMY of 7l kg (95% BCI 54 to 88 kg). Decrease in AFC from 27 to 24 months was associated with a median decrease in FLMY of 232 kg (95% BCI 217 to 247 kg; Table 6.3).

Table 6.3. Bayesian credible intervals from 10,000 simulations of the final models; outcome cow level first lactation milk yield (kg)

		Model 6.1[85]			Model 6.2[86]		
Exposure (baseline)		Lower 2.5%	Median	Upper 97.5%	Lower 2.5%	Median	Upper 97.5%
Intercept		954	3,068	5,228	974	3,127	5,268
Mean ln SCC[87] (4.54)^1		-163	-135	-108	NA	NA	NA
ln SCC1 (4.66)		NA[88]	NA	NA	-88	-71	-54
ln AFC[89] (6.7)^1		1,976	2,169	2,362	1,991	2,181	2,374
ln AFC (6.7)^2		-1,569	-1,131	-695	-1,787	-1,377	-957
Mean ln SCC^2 x ln AFC		-440	-272	-110	NA	NA	NA
Month of first calving (June)	January	623	784	942	640	800	961
	February	269	428	580	293	445	598
	March	-51	106	258	-33	119	270
	April	-326	-170	-13	-323	-165	-6
	May	-494	-320	-150	-494	-322	-152
	July	365	627	881	382	634	890
	August	865	1,121	1,376	882	1,142	1,397
	September	1,203	1,399	1,588	1,219	1,413	1,606
	October	1,222	1,415	1,601	1,244	1,433	1,619
	November	1,053	1,259	1,453	1,063	1,269	1,466
	December	946	1,178	1,414	964	1,196	4,137
Year of first calving (2007)	2004	-124	2,039	4,164	-176	1,960	4,137
	2005	182	2,335	4,448	142	2,269	4,424
	2006	274	2,424	4,541	229	2,362	4,512
Random effect standard deviation:		Lower 2.5%	Median	Upper 97.5%	Lower 2.5%	Median	Upper 97.5%
Cow level		2,037	2,051	2,064	2,038	2,052	2,065
Herd level		1,265	1,297	1,330	1,265	1,297	1,331

[85] Impact of mean natural logarithm of (ln) milk somatic cell count (SCC) over the first lactation.
[86] Impact of SCC at 5 to 30 days in milk during parity 1 (SCC1).
[87] Over the entire first lactation, centred on the mean (4.54).
[88] Not applicable.
[89] Age at first calving (days); included as polynomial terms, centred on the mean (6.7)

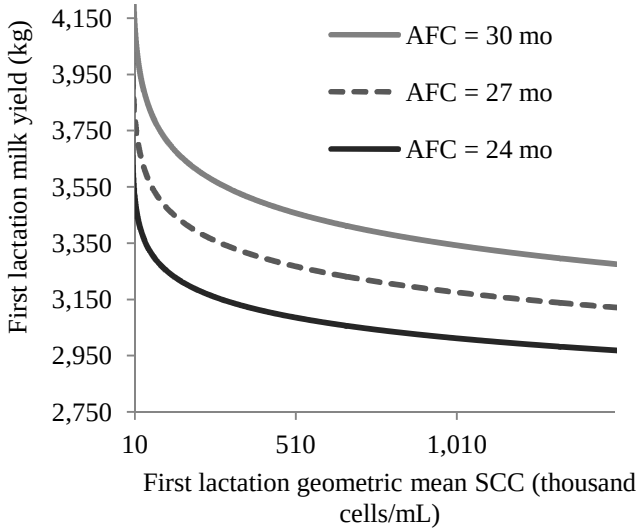

Figure 6.1. Median predictions of first lactation milk yield for specific cows[90] from Model 6.1 (exposure; mean ln[91] SCC[92] over the first lactation) to show the impact of age at first calving (AFC)

[90] First calving in February 2007
[91] Natural logarithm of.
[92] Milk somatic cell count.

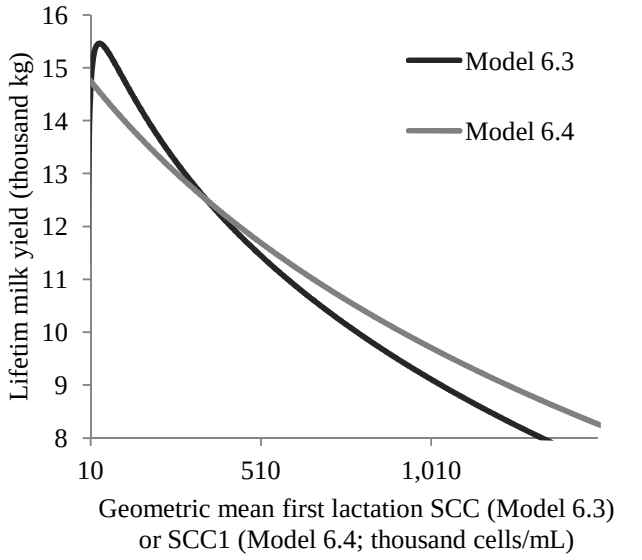

Figure 6.2. Median predictions of lifetime milk yield for specific cows[93] from Model 6.3 (exposure; mean and variance of ln[94] SCC[95] over the first lactation) and Model 6.4 (exposure; ln SCC between 5 and 30 days in milk during the first lactation; SCC1)

[93] First calving in February 2007, aged 27 months, variance in mean ln SCC during the first lactation = 0.62.
[94] Natural logarithm of.
[95] Milk somatic cell count.

6.3.2.2 Outcome 2; lifetime milk yield

The final models for LiMY (Models 6.3 and 6.4; Table 6.4) were adjusted for month, and year of first calving, and AFC. The relationship between geometric mean first lactation SCC and LiMY (Model 6.3) is shown in Figure 6.2. A 1-unit increase in the mean of ln SCC over the first lactation was associated with a median decrease in lifetime milk yield of 1,663 kg (95% BCI 1,347 to 1,986 kg; calculated by adding the polynomial terms for ln SCC over the first lactation from Model 6.3; Table 6.4). A 1-unit increase in the variance of ln SCC over the first lactation was associated with a median decrease in LiMY of 719 kg (95% BCI 553 to 888 kg; Figure 6.3). With SCC1 as the exposure of interest (Model 6.4; Table 6.4), a 1-unit increase in ln SCC1 was associated with a median decrease in LiMY of 633 kg (95% BCI 533 to 733 kg; Figure 6.2). In contrast to Models 6.1 and 6.2, decreased AFC was associated with increased LiMY, and the impact differed between Models 6.3 and 6.4. In Model 6.3, AFC was included as a 4^{th} order polynomial; decrease in AFC, from 27 to 24 months was associated with a median increase in LiMY of 99 kg (95% BCI 33 to 160 kg). In Model 6.4, decrease in AFC from 27 to 24 months was associated with a median increase in LiMY of 574 kg (95% BCI 483 to 663 kg). No biologically plausible interactions, for example between ln SCC1 and ln AFC were identified.

Table 6.4. Bayesian credible intervals from 10,000 simulations of the final models; outcome cow level lifetime milk yield (kg)

		Model 6.3[96]			Model 6.4[97]		
Exposure (baseline)		Lower 2.5%	Median	Upper 97.5%	Lower 2.5%	Median	Upper 97.5%
Intercept		-3,378	9,770	22,460	-4,040	8,976	22,020
ln SCC1 (4.66)		NA[98]	NA	NA	-733	-633	-533
Mean ln SCC[99] (4.54)^1		-1,279	-1,090	-910	NA	NA	NA
Mean ln SCC[95] (4.54)^2		-707	-573	-437	NA	NA	NA
Variance ln SCC[95] (0.62)		-888	-719	-553	NA	NA	NA
ln AFC[100] (6.7)^1		-4,798	-3,396	-1,976	-6,633	-5,735	-4,833
ln AFC (6.7)^2		-30,510	-21,240	-12,280	NA	NA	NA
ln AFC (6.7)^3		13,660	26,940	40,500	NA	NA	NA
ln AFC (6.7)^4		-12,730	-7,969	-3,273	NA	NA	NA
Month of first calving (June)	January	5,448	6,446	7,386	5,295	6,245	7,220
	February	4,225	5,171	6,060	4,079	4,992	5,937
	March	2,694	3,626	4,513	2,593	3,505	4,434
	April	886	1,845	2,771	825	1,756	2,719
	May	-222	813	1,854	-333	702	1,730
	July	-1,552	24	1,587	-1,572	-5	1,575
	August	1,326	2,879	4,451	1,328	2,871	4,436
	September	3,706	4,907	6,049	3,555	4,721	5,907
	October	3,986	5,133	6,249	3,859	4,986	6,140
	November	3,082	4,290	5,492	2,896	4,114	5,326
	December	1,950	3,347	4,710	1,775	3,182	4,565
Year of first calving (2007)	2004	1,394	14,240	27,510	1,347	14,500	27,540
	2005	-1,379	11,280	24,420	-1,480	11,500	24,390
	2006	-3,757	8,894	22,060	-3,889	9,154	22,050
Random effect standard deviation:		Lower 2.5%	Median	Upper 97.5%	Lower 2.5%	Median	Upper 97.5%
Cow level		12,418	12,498	12,578	12,458	12,538	12,621
Herd level		6,899	7,094	7,294	6,961	7,153	7,352

[96] Impact of first lactation mean and variance in the natural logarithem of (ln) milk somatic cell count (SCC) over the whole first lactation.
[97] Impact of SCC at 5 to 30 days in milk during the first lactation (SCC1).
[98] Not applicable.
[99] Over the entire first lactation.
[100] Age at first calving (days); included as polynomial terms.

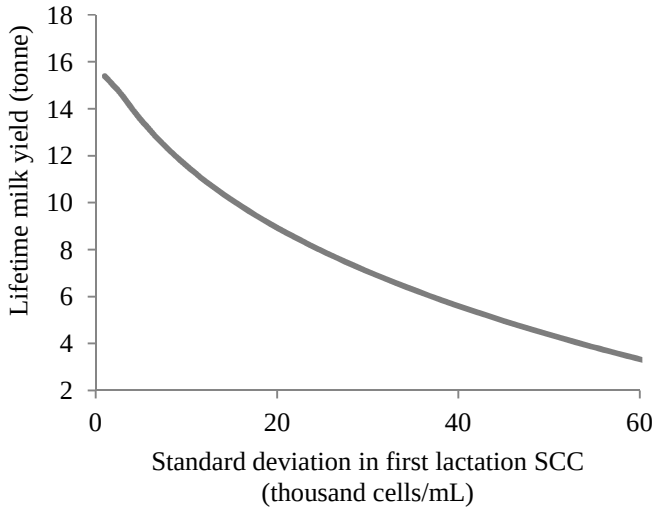

Figure 6.3. Median predictions of lifetime milk yield for specific cows[101] from Model 6.3 (exposure; mean and variance of ln[102] SCC[103] over the first lactation)

[101] First calving in February 2007, aged 27 months, and mean ln SCC during the first lactation = 4.54.
[102] Natural logarithm of.
[103] Milk somatic cell count.

6.3.2.3 Model checking

Predictions of FLMY or LiMY for cows aggregated in quartiles by geometric mean first lactation SCC, indicated good fit, and hence that Models 6.1 and 6.3 were adequate for predictions in these herds (Figure 6.4). This was also the case for predictions of lifetime and first lactation milk yield aggregated by SCC1 group from Models 6.2 and 6.4 (not shown). There was < 1% difference in the median, and 95% BCI limits of the coefficient distributions for exposures of interest when a uniform prior distribution for the herd level random effect variance was used, indicating that choice of prior distribution had no substantive impact on model interpretation.

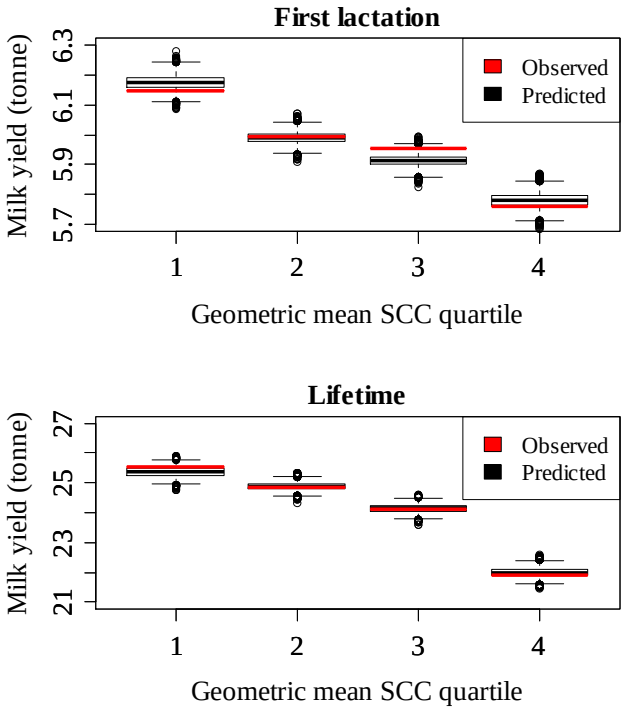

Figure 6.4. Assessment of model fit and usefulness; predictions of first lactation and lifetime milk yield from 10,000 simulations of Model 6.1[104] and Model 6.3[105] respectively[106] , and observed values in 5,900 Irish dairy herds grouped by first lactation geometric mean SCC[107] (Group 1; < 55,000 cells/mL, group 2; 55,000 to 89,000 cells/mL, group 3; 90,000 to 149,000 cells/mL, group 3; ≥ 150,000 cells/mL).

[104] Outcome; first lactation milk yield. Exposures; First lactation mean of ln SCC over the first lactation.
[105] Outcome; lifetime milk yield. Exposures; First lactation mean and variance of ln SCC over the first lactation.
[106] The horizontal line is the median, the surrounding boxes contain 50% of the data, the vertical whiskers extend to 1.5 times the interquartile range, and outliers are shown beyond this.
[107] Milk somatic cell count.

6.3.2.4 Micro-simulation

Figure 6.5 shows the cumulative probability distribution of potential cost savings for every heifer in the herd attributable to increased LiMY, associated with reductions in herd_gSCC_p1. Direct probabilities for different levels of saving can be read from Figure 6.5. For example, there was 75% certainty of cost savings of at least €199 /heifer in the herd, if herd_gSCC_p1 reduced from ≥ 120,000 cells/mL to ≤ 72,000 cells/mL. That would be equivalent to moving from the upper to the lower quartile for herd_gSCC_p1 (Table1). For a herd in

which 20 heifers complete the first lactation, this is equivalent to a saving of €3,980 associated with moving from the highest to the lowest herd_gSCC_p1 quartile. Further scenarios for the example herd are given in Table 6.5.

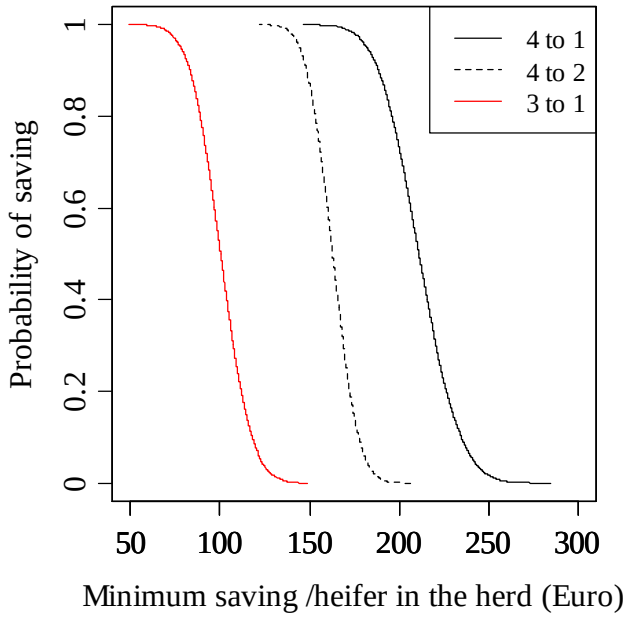

Figure 6.5. Micro-simulation over 10,000 simulations of Model 6.3[108]; minimum cost saving /heifer in the herd attributable to increased lifetime milk yield associated with reduction in herd quartile[109] for first lactation geometric mean SCC[110]

[108] Exposures; First lactation mean and variance in ln SCC over the first lactation.
[109] 1; ≤ 72,000 cells/mL, 2; 72,000 to 93,000 cells/mL, 3; 93,000 cells/mL to 120,000 cells/mL, 4; ≥ 120,000 cells/mL.
[110] Milk somatic cell count.

Table 6.5. Simulated cost savings through increased lifetime milk yield[111] associated with specific reductions in herd level first lactation geometric mean SCC[112], for an example herd in which 20 heifers complete the first lactation

Probability	Herd level geometric mean first lactation SCC (quartiles[113])		
	4 to 1	4 to 2	3 to 1
0.75	≥ €3,980	≥ €3,100	≥ €1,820
0.5	≥ €4,220	≥ €3,260	≥ €2,020
0.25	≥ €4,460	≥ €3,420	≥ €2,200

[111] Milk margin was drawn from a normal distribution with mean = 0.17 €/L, and standard deviation = 0.03 €/L for each cow.
[112] Milk somatic cell count.
[113] 1; ≤ 72,000 cells/mL, 2; 72,000 to 93,000 cells/mL, 3; 93,000 cells/mL to 120,000 cells/mL, 4; ≥ 120,000 cells/mL.

6.3.3 Somatic cell count legacy during the first lactation; model results

The association between ln SCC1 and both the mean, and variance of ln SCC throughout the first lactation varied by herd (Model 6.5; Tables 6.6 and 6.7), and the between herd variation in the relationship between SCC1 and geometric mean SCC during the first lactation was large (Table 6.7, Figure 6.6). For most herds, increase in SCC1 was associated with increase in geometric mean first lactation SCC, but this was not always the case (Figure 6.6). The mean (baseline) AFC in Model 6.5 was 27 months, and this interacted with SCC1 (Table 6.6). With SCC1 unchanged, a 3 month change in AFC was positively associated with a 1.3% (95% BCI 0.5 to 2.1 %) change in geometric mean SCC during the first lactation.

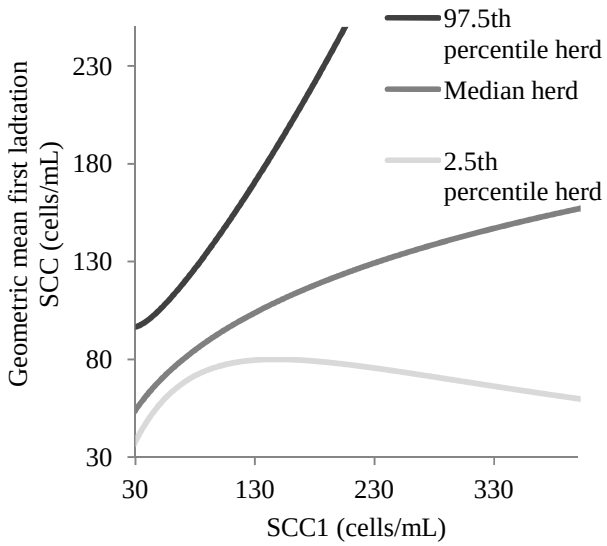

Figure 6.6. Predictions of median geometric mean SCC[114] during the first lactation from Model 6.5[115] for different herds based on magnitude of relationship with SCC at 5 to 30 days in milk (SCC1)

[114] Milk somatic cell count.
[115] Multivariate normal model with mean and variance of ln SCC during the first lactation as outcomes, and herd level random slopes for SCC1 (exposure of interest). The predictions refer to cows with a first calving during February aged 27 months.

Table 6.6. Bayesian credible intervals for fixed effects following 10,000 simulations of the final model for the mean and variance of ln[116] SCC[117] during the first lactation (Model 6.5).

Fixed effects (baseline)		Mean ln SCC during p1[118] Lower 2.5%	Median	Upper 97.5%	Variance ln SCC during p1 Lower 2.5%	Median	Upper 97.5%
Intercept		4.591	4.628	4.665	0.441	0.449	0.457
Month of first calving (June)	January	-0.106	-0.067	-0.028	NA[119]	NA	NA
	February	-0.107	-0.069	-0.032	NA	NA	NA
	March	-0.081	-0.043	-0.006	NA	NA	NA
	April	-0.039	0	0.038	NA	NA	NA
	May	-0.02	0.023	0.065	NA	NA	NA
	July	-0.09	-0.026	0.038	NA	NA	NA
	August	-0.14	-0.077	-0.015	NA	NA	NA
	September	-0.122	-0.076	-0.028	NA	NA	NA
	October	-0.132	-0.085	-0.039	NA	NA	NA
	November	-0.081	-0.03	0.018	NA	NA	NA
	December	-0.113	-0.064	-0.015	NA	NA	NA
ln AFC[120] (6.7)		0.12	0.161	0.205	0.06	0.104	0.148
ln SCC1[121] (4.66)^1		0.406	0.413	0.419	0.044	0.051	0.058
ln SCC1 (4.66)^2		-0.033	-0.03	-0.026	0.132	0.137	0.141
ln SCC1 (4.66)^1.ln AFC (6.7)		-0.069	-0.036	-0.001	-0.097	-0.059	-0.023
ln SCC1 (4.66)^2.ln AFC (6.7)		0.016	0.034	0.052	-0.048	-0.027	-0.006

[116] Natural logarithm of.
[117] Milk somatic cell count.
[118] First lactation.
[119] Not applicable.
[120] Age at first calving.
[121] SCC at 5 to 30 days in milk during parity 1.

Table 6.7. Matrices of median (co)variances (95% Bayesian credible interval) following 10,000 simulations of the final model; outcomes mean and variance of ln[122] SCC[123] during the first lactation (Model 6.5).

Cow level	
σ^2_{u0} [124]	σ^2_{u1} [125]
0.278	
(0.274 to 0.282)	
0.065	0.310
(0.062 to 0.068)	(0.306 to 0.314)

Herd level					
σ^2_{v0} [126]	σ^2_{v1} [127]	σ^2_{v2} [128]	σ^2_{v3} [129]	σ^2_{v4} [130]	σ^2_{v5} [131]
0.049					
(0.045 to 0.052)					
-0.007	0.031				
(-0.009 to -0.004)	(0.028 to 0.034)				
0.008	-0.008	0.014			
(0.006 to 0.01)	(-0.01 to -0.006)	(0.012 to 0.015)			
-0.001	0.003	0.000	0.002		
(-0.002 to 0.000)	(0.002 to 0.004)	(0.00 to 0.001)	(0.002 to 0.002)		
-0.023	0.002	-0.001	-0.002	0.019	
(-0.025 to -0.021)	(0.000 to 0.004)	(-0.003 to 0.000)	(-0.003 to -0.002)	(0.017 to 0.022)	
0.001	-0.004	-0.004	-0.002	0.000	0.008
(0.000 to 0.003)	(-0.005 to -0.003)	(-0.005 to -0.003)	(-0.002 to -0.001)	(-0.001 to 0.001	(0.008 to 0.009)

[122] Natural logarithm of.
[123] Milk somatic cell count.
[124] Cow level variance in the intercept for mean of ln SCC during the first lactation.
[125] Cow level variance in the intercept for variance of ln SCC during the first lactation.
[126] Herd level variance in the intercept for mean of ln SCC during the first lactation.
[127] Herd level variance in the intercept for variance of ln SCC during the first lactation.
[128] Herd level variance in the coefficient for SCC at 5 to 30 DIM during parity 1 (SCC1) for the mean of ln SCC during the first lactation.
[129] Herd level variance in the coefficient for SCC1 for the variance of ln SCC during the first lactation.
[130] Herd level variance in the coefficient for SCC1^2 for the mean of ln SCC during the first lactation.
[131] Herd level variance in the coefficient for SCC1^2 for the variance of ln SCC during the first lactation.

6.4 Discussion

To the author's knowledge, the analyses in this chapter is the first to demonstrate large differences in both FLMY and subsequent LiMY of cows, associated with the geometric mean and variance of SCC during the first lactation. The results highlight that in addition to the importance of optimizing the udder health of heifers in early lactation, as found in chapter 3 (De Vliegher et al., 2005a), this is also vital throughout the remainder of the first lactation in terms of lifetime productivity. The median decrease in LiMY of 1,663 kg /1-unit increase in mean ln SCC over the first lactation in this chapter, was much larger than the median first lactation milk loss of 135 kg associated with 1-unit increase in mean ln SCC over the first lactation. This first lactation milk loss was similar to the estimate made by Raubertas and Shook (1982), however it exceeded estimates from higher yielding cows in more recent studies based on test day recordings, in which 1-unit increase in mean ln SCC over the first lactation was associated with losses of 85 to 120 kg over 305 d for primiparous cows (Dürr et al., 2008; Halasa et al., 2009). Importantly, previous analyses based on test day recordings only show the milk loss in affected cows that survive; probably associated with residual udder pathology, but exclude milk loss associated with premature culling. In contrast cumulative measures of milk yield take cow longevity into account to give a more realistic estimate of milk loss. The trend for higher FLMY with increased AFC was consistent with previous research (Berry and Cromie, 2009). As seen in chapters 3 and 5, it was also likely that decreased AFC was associated with increased longevity (Berry and Cromie, 2009), and hence LiMY.

A 1-unit increase in ln SCC1 was associated with median decreases in FLMY and LiMY of 71 and 633 kg respectively. This was slightly less than estimates for the reduction in FLMY and subsequent LiMY from chapter 3 of 105 and 864 kg respectively associated with 1-unit increase in SCC1, probably because inclusion criteria for heifers were different. To enable mean and variance in ln SCC over the first lactation to be calculated for all heifers, the current study only included data for heifers that survived for at least 2 recordings in the first lactation. Therefore compared to chapter 3, the total impact of mastitis early in the first lactation on cumulative milk yield has probably been underestimated, because heifers that were culled soon after calving were excluded. However, models for FLMY and LiMY with SCC1 as the exposure of interest were included in this research to make valid comparisons with the models that had mean ln SCC over the entire first lactation as the exposure of interest, as the same dataset was used.

Heifers with high SCC in early lactation may have high geometric mean SCC throughout the entire first lactation (De Vliegher et al., 2004a; Santman-Berends et al., 2012), as a result of failure to cure from early lactation IMI or subsequent new IMI. However, the association between cow level SCC1 and geometric mean SCC over the first lactation in this chapter varied between herds (Figure 6.6), suggesting that differences in the dynamics of IMI, and the management of heifers between herds has an important impact on patterns of SCC during the first lactation. High cow SCC early in the first lactation, and throughout the whole of the first lactation were both associated with reduced FLMY and LiMY. Therefore, control measures to reduce SCC in an individual herd may be relatively more important during either the ppp period (Green et

al., 2008), or the lactating period (Barkema et al., 2009). A rational approach to managing heifer mastitis in herds with high first lactation geometric mean SCC would be to identify if this may be a result of high SCC during the ppp or the lactating period, and prioritize control measures accordingly, as both scenarios appear equally likely in Irish dairy heifers (Table 6.2). Further investigations should evaluate risk factors for heifer mastitis in terms of impact on SCC throughout the entire first lactation in order to develop herd specific management interventions to optimise the lifetime milk yield of dairy cows

6.5 Conclusions

This chapter demonstrated that for cows in Irish dairy herds, geometric mean and variance of first lactation SCC, and SCC1 were negatively associated with both first lactation and lifetime milk yield. The apparent legacy of SCC early in the first lactation on SCC for the remainder of the first lactation was highly herd dependent. Approximately 50% of Irish dairy herds have potential to make savings through reducing SCC throughout the first lactation. This could involve preferentially targeting mastitis control measures in a herd specific manner towards the ppp period, or towards the lactating period, depending on individual herd SCC patterns. Further research is needed to define the most cost effective control measures in different circumstances.

Chapter 7: Bayesian evaluation of budgets for endemic disease control; an example using management changes to reduce milk somatic cell count early in the first lactation of cows in Irish dairy herds

7.1 Introduction

Chapters 3 and 5 showed that for 50% of Irish dairy herds, reducing the prevalence of cows with high somatic cell count at 5 to 30 days in milk during parity 1 (SCC1) would be associated with savings through increased longevity, and lifetime milk yield (LiMY). This reduction may be achieved through herd level management interventions targeted at ppp heifers (Green et al., 2008). Previous studies have identified risk factors for mastitis in primiparous cows (De Vliegher et al., 2012), however the cost and efficacy of particular management changes have yet to be evaluated in the field. Data on the likely cost effectiveness of management interventions is therefore unavailable. However, potentially effective interventions may not be deemed 'cost effective' if they are too expensive to implement, or the desirable outcome is too uncertain for particular decision makers (Spiegelhalter et al., 2004). It is therefore unrealistic for economic analyses to assume an unlimited 'willingness to pay' for each Euro saved through reduced disease costs. Rational budgets for management interventions are unknown, and this information would facilitate the development of practical advice to control heifer mastitis on Irish dairy farms and elsewhere.

Uncertainty and variability in parameters can be handled with Bayesian analyses, which can be extended with micro-simulation to generate posterior predictions for particular scenarios as used in chapters 3 to 6. Making distributional assumptions can be avoided, with all uncertainty, and relationships between variables propagated through to the final outcome by using a 1-step procedure (Chessa et al., 1999; Spiegelhalter et al., 2004). However, a 2-step micro-simulation procedure, where distributions for parameters are obtained from other research or expert opinion is more common, and has been used to estimate average costs of high milk somatic cell count (SCC) in early lactation of €31 (range 0 to 220) / heifer in the herd (Huijps et al., 2009a). The integrated 1-step procedure has been applied previously to investigate the impact of management interventions in dairy herds, with iterations propagated from a single model (Green et al., 2010). However, the approach can be extended to synthesise evidence from multiple sources, as used in cost effectiveness analyses for human medical treatments (O' Hagan and Stevens, 2001; Spiegelhalter and Best, 2003). To the author's knowledge this extension of the methodology has not been applied in a veterinary context, and control of heifer mastitis is taken as an example.

The aim of this chapter was to use 1-step Bayesian micro-simulation to synthesise evidence, and determine budgets for specific management interventions to control heifer mastitis in Irish dairy herds under different circumstances.

7.2 Materials and methods

7.2.1 Overview

An overview of the 1-step micro-simulation procedure is provided in Figure 7.1. This procedure was used to estimate the likely economic impact of specific interventions to control heifer mastitis, in terms of changes in lifetime milk yield and cow disposal risk. Therefore, models for lifetime milk yield (chapter 3), and disposal risk (chapter 5) were run in parallel using their respective data for 10,000 Markov chain Monte Carlo (MCMC) iterations using WinBUGS 1.4.3 (Lunn et al., 2000). At each iteration after burn in, coefficient estimates from the models were taken forward and combined with simulated data for theoretical cows (based on ≥ 20%, and ≥ 30% herd level prevalence of cows with SCC1 ≥ 200,000 cells/mL) to predict lifetime milk yield and the occurrence of disposal within 1,750 days of first calving. Management interventions thought to reduce SCC1 were assumed to be implemented. Potential financial savings associated with applying the interventions were determined from the mean difference in lifetime milk yield, and disposal risk at herd level. The probability of cost effectiveness, and maximum rational spend for implementing the management interventions was estimated for different decision makers based on their expected minimum return on investment and willingness to pay.

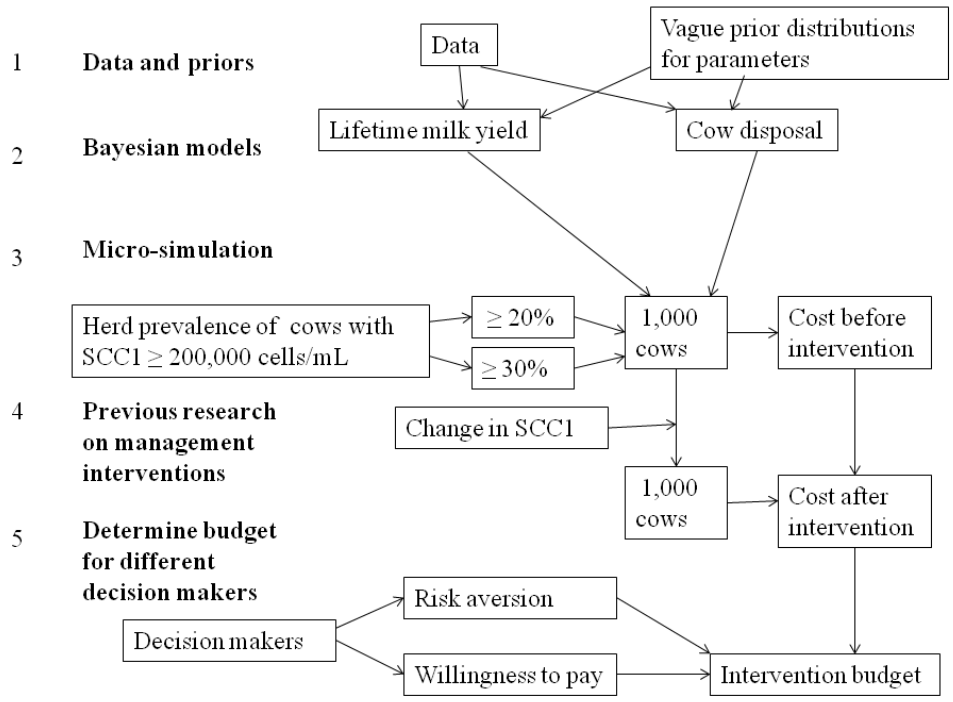

Figure 7.1. A diagrammatic representation of the 1-step micro-simulation procedure. Iterations from Bayesian models for lifetime milk yield and disposal risk from separate analyses were run in parallel, and applied to 1,000 theoretical cows in herds with ≥ 20%, and ≥ 30% of cows with high somatic cell count (≥ 200,000 cells/mL) between 5 and 30 days in milk during parity 1 (SCC1). Savings associated with keeping bedding materials inside rather than outside (change in ln SCC1 ~ Normal(-0.15, 0.02)), increasing transition yard area from < 1.25 m^2 to > 1.25 m^2 / 1,000 kg of milk production (change in ln SCC1 ~ Normal(-0.12, 0.01)), and ensuring that bedding in the calving area was spread evenly instead of unevenly (change in ln SCC1 ~ Normal(-0.19, 0.05)) were simulated. The assumed distribution of revenue from milk margin was ~ Normal (mean = 0.17, standard deviation = 0.03) €/L, replacement cost was €1,451 /cow disposal, and decision maker willingness to pay for interventions (k) was between €0 and €1 /€1 of potential saving. Savings were accrued through increased lifetime milk yield, and decreased disposal risk of cows. Interventions were deemed cost effective for a particular decision maker when incremental net benefit (INB) ≥ 0, where INB = (k x savings) – costs. The maximum budget for the interventions was therefore the savings when INB = 0, and this was determined at different levels of certainty for each value of k.

7.2.2 Lifetime milk yield model (Model 3.1)

This model evaluated the association between the SCC1, and lifetime milk yield for cows in Irish dairy herds and is described fully in chapter 3. The results used in the micro-simulation are shown in Table 7.1. Overall, one unit increase in ln SCC1 (natural logarithm of SCC1) was associated with a median

decrease in lifetime milk yield of 865 kg (95% Bayesian credibility interval (BCI) 702 to 1,025 kg).

Table 7.1. Lifetime milk yield model (Model 3.1; Chapter 3)[132]; parameters used in the micro-simulation procedure

	95% Bayesian credibility interval		
Exposure (baseline)	Lower 2.5%	Median	Upper 97.5%
Intercept	-4,819	10,950	26,260
ln[133] SCC1[134] (4.65)	-1,025	-865	-702
First calving February 2007	2,979	4,418	5,832
ln AFC[135] (6.71)	-8,302	-6,906	-5,484

[132] Only relevant parameters shown.
[133] Natural logarithm of.
[134] First test day somatic cell count record at 5 to 30 days in milk during parity 1.
[135] Age at first calving (days).

7.2.3 Cow disposal model (Model 5.1)

This model evaluated the association between SCC1, and survival over a 5 year period from 2005 to 2009, for cows in Irish dairy herds and is described fully in chapter 5. The results used in the micro-simulation are shown in Table 7.2. Disposal odds increased by 5% (BCI 2 to 9%) per unit increase in ln SCC1.

Table 7.2. Cow disposal model (Model 5.1; chapter 5)[136]; parameters used in the micro-simulation procedure

		95% Bayesian credibility interval (odds ratio)		
Exposure (baseline)		Lower 2.5%	Median	Upper 97.5%
Intercept		0.002	0.002	0.003
ln[137] SCC1[138] (4.64)		1.020	1.052	1.085
TDY1[139] (23 kg)		0.968	0.976	0.983
TDF1[140] (0.04)		0.000	0.001	0.090
ln AFC[141] (6.70)		1.770	2.263	2.930
[ln interval[142]]^1 (2.28)		1.260	1.361	1.473
[ln interval]^2 (2.28)		1.847	1.970	2.100
[ln interval]^3 (2.28)		1.198	1.247	1.298
DIM[143] (< 100)	100 to 199	2.642	2.939	3.264
	200 to 304	5.280	5.883	6.554

[136] Only relevant parameters shown.
[137] Natural logarithm of.
[138] First test day somatic cell count record between 5 and 30 days in milk (DIM) during parity 1.
[139] First test day milk yield record (kg) between 5 and 30 DIM in parity 1.
[140] First test day fat record (proportion) between 5 and 30 DIM in parity 1.
[141] Age at first calving (days).
[142] 50 day intervals from first calving. Included as polynomials.
[143] DIM category in the penultimate interval for each cow. Missing category not shown.

7.2.4 One-step micro-simulation

7.2.4.1 Simulation of individual cows

To account for the variability in parameters, coefficient values from Models 3.1 and 5.1 were propagated onward (at each iteration) and applied to 1,000 simulated cows kept in herds that housed *pre-partum* heifers, and did not apply the specific management interventions to be tested. At each of 10,000 MCMC simulations, coefficients from Models 3.1 and 5.1 were combined with data from the theoretical cows to generate predictions of lifetime milk yield and the occurrence of disposal within 1,750 days from first calving for the i^{th} cow in the j^{th} herd ($y.pred_{ij}$);

$$y.pred_{ij} \sim p(y.pred_{ij} | \boldsymbol{\beta}, X^{sim}),$$

where *p* represents a conditional probability distribution, **β** is a vector of coefficient distributions from Model 3.1 or 5.1, and X^{sim} was a matrix of data for simulated cows. This included an indicator variable to denote a first calving in February 2007 (aged 24 months), and data from a first milk recording (including ln SCC1) at 5 to 30 DIM simulated from observed normal distributions based on the initial herd level prevalence of cows with SCC1 ≥ 200,000 cells/mL (Table 7.3). Interval dependent indicator variables were used for month of final recording and DIM category in the penultimate interval.

Table 7.3. Observed herd frequency[144], and cow level[145] means (variances) categorised by high SCC1[146] prevalence

		Herd level prevalence of cows with SCC1 ≥ 200,000 cells/mL	
		≥ 20%	≥ 30%
Observed data	Percentage of herds	59%	26%
	ln[147] SCC1	4.82 (1.47)	5.06 (1.56)
	Milk1[148]	23 (30.0)	22 (33.3)
	Fat1[149]	0.04 (0.00007)	0.04 (0.00007)

[144] Based on 7,423 Irish dairy herds.
[145] Based on 233,176 parity 1 cows in 7,423 Irish dairy herds.
[146] First test day somatic cell count record (cells/mL) between 5 and 30 days in milk (DIM) during parity 1.
[147] Natural logarithm of.
[148] First test day milk yield record (kg) between 5 and 30 DIM during parity 1.
[149] First test day fat record (proportion) between 5 and 30 DIM during parity 1.

7.2.4.2 Economic simulation

At every iteration, the difference in lifetime milk yield for each cow in these scenarios, before and after applying the management interventions, was multiplied by the estimated gross margin (Milk price – variable costs of production) ~ Normal (mean = 0.17, standard deviation = 0.03) €/L (Hennessy et al., 2011), to give the predicted difference in milk revenue. In addition, at every iteration, the difference in the number of cows disposed within 1,750 days from first calving for each scenario was multiplied by €1,451 (Kennedy et

al., 2011), to estimate replacement costs. Following the assumed management interventions, the cost differences associated with increased lifetime milk yield and decreased cow disposal risk were expressed as a mean financial value per heifer in the herd (Figure 7.1). Posterior distributions of total savings per heifer in the herd were plotted as a cumulative frequency distribution to show the probability of different levels of return in an intuitive form.

7.2.4.3 Simulation of management interventions

Three interventions applicable to mastitis control for housed ppp heifers to improve environmental hygiene, and therefore reduce the risk of new intramammary infections were selected from previous research based on farm observations (Table 7.4, (Green et al., 2008)). The interventions were storage of bedding inside, decreasing transition yard stocking density (from < 1.25 m^2 to > 1.25 m^2 /1,000 kg of annual mean milk production for the herd), and spreading of bedding evenly in the calving area. Storage of bedding material inside implies it is more likely to be dry when used, and therefore less able to support microbial growth. Increase in transition yard area /cow implies the yard has less contamination. Spreading of bedding material evenly in the calving area provides a more hygienic environment compared to if the bedding material is clumped. Normal distributions for change in ln SCC1 associated with these interventions were assumed (Table 7.4); the mean was available, and the variance was estimated given that the 95% BCIs reported were equivalent to 2 standard deviations (Green et al., 2008). Draws from these distributions were added to the simulated ln SCC1 for each cow (Figure 7.1), to determine the impact of the 3 interventions when applied together for herds

with ≥ 20%, or ≥ 30% initial prevalence of cows with SCC1 ≥ 200,000 cells/mL.

Table 7.4. Change in Normal distribution parameters for ln SCC1 (natural logarithm of SCC1)[150] associated with management interventions (Green et al., 2008)

Management intervention	Mean	Variance
Storage of bedding material inside	-0.15	0.02
Decreased transition yard[151] stocking density	-0.12	0.01
Even spreading of bedding in calving area	-0.19	0.02

[150] Somatic cell count at 5 to 30 days in milk during parity 1.
[151] From < 1.25 m^2 to > 1.25 m^2 per 1,000 kg of herd annual mean milk production /cow.

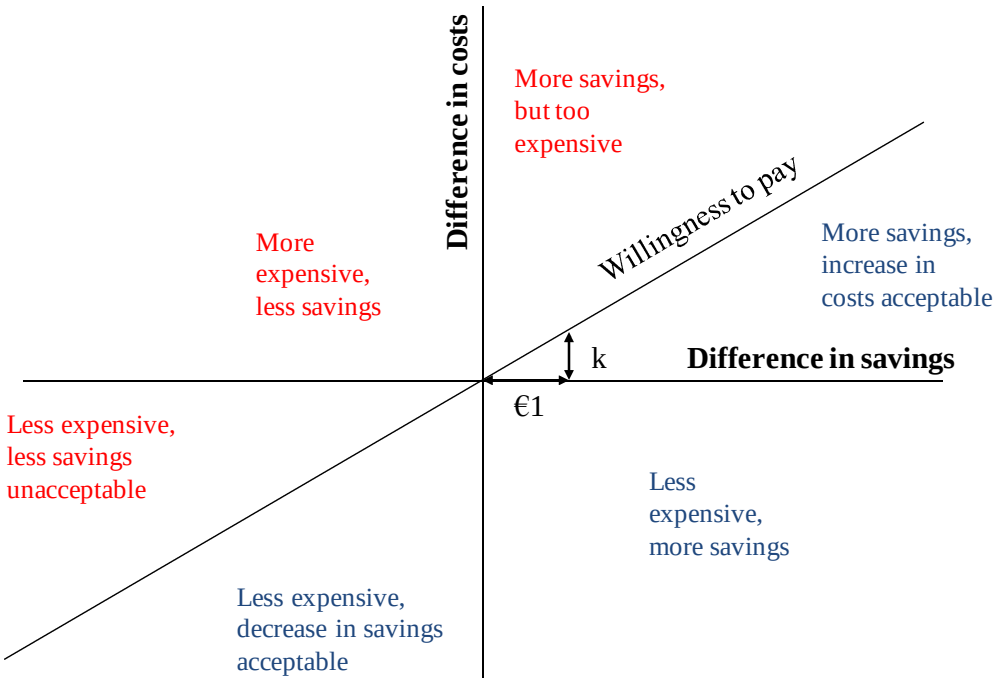

Figure 7.2. A diagrammatic representation of a cost effectiveness plane. The horizontal and vertical axes show differences in savings, and costs respectively. These axes are bisected by a line with slope (k), representing willingness to pay / €1 of saving. Points below the willingness to pay line are cost effective, and those above it are not. The outcome from economic models can be plotted on the cost effectiveness plane, producing a density map over multiple iterations, to show the likelihood of cost effectiveness for specific interventions (Spiegelhalter et al., 2004).

7.2.5 Willingness to pay

A cost effectiveness plane is illustrated in Figure 7.2; position on the plane is determined by the difference in costs and savings for a particular intervention. The axes of Figure 7.2 are bisected by a line that divides the plane into quadrants; the slope of the line (k) represents 'willingness to pay' for an intervention. In the context of changes to dairy herd management in this research, costs and savings are in monetary units. Therefore k represents the amount that a particular decision maker is prepared to invest for every €1 of saving, and hence the return on investment that would be acceptable for a particular management change at a particular cost. Points above the 'willingness to pay' line would not be considered cost effective by the decision maker (as costs are more than the acceptable savings). Conversely, points below the 'willingness to pay' line are considered cost effective. The cost effectiveness of interventions therefore depends on the slope k, which is determined by the attitude of the decision maker. For example, if savings that merely cover the intervention cost are acceptable; k = €1. Values of k ≥ €1, imply the decision maker is willing to pay more than the expected return on saving. With a willingness to pay (k) of > €0 and < €1 /€1 of saving, the value chosen reflects the minimum return on investment the decision maker expects over and above the intervention cost in order that they would consider the intervention to be cost effective, and therefore be a rational choice. The relationship between willingness to pay, and the minimum expected return on investment is shown in Figure 7.3; for example, if k = €0.5 /1€ saving, the decision maker would not accept a return < €1 for every €1 invested. 'Willingness to pay', and hence the slope of the line that bisects Figure 7.2

changes for different decision makers. Position on the plane of Figure 7.2 relative to this line determines if a particular intervention is likely to be deemed 'cost effective', and therefore a rational choice for a particular decision maker. For example, an effective intervention could be to build additional housing to increase space allowance for *pre-partum* heifers, however if the most likely combination of costs and savings falls in the top right quadrant of Figure 7.2, above the willingness to pay threshold, the intervention would be deemed too expensive, and would not be implemented. Conversely, the most likely economic outcome for a less effective but cheaper intervention, such as buying a tarpaulin to keep bedding material dry could fall below the willingness to pay threshold in the top right quadrant of Figure 7.2, and therefore be deemed cost effective. Decision makers typically do not divulge their willingness to pay for interventions; therefore, a sensitivity analysis is often required to evaluate how the incremental net benefit (INB) varies with willingness to pay (k) /€1 of potential saving (Spiegelhalter et al., 2004). This is equivalent to varying the slope of the willingness to pay line in Figure 7.2, and assessing the impact on likely cost effectiveness, where;

INB[k] = k x difference in savings – difference in costs, and

k = (0:10) x €0.1.

Appropriate levels of spending for the control of mastitis in heifers during the ppp period are unknown. Therefore, posterior distributions for the maximum intervention cost (when INB[k] = 0) were determined. The maximum intervention cost determines the budget available for implementing

the interventions in order that they are considered 'cost effective' by a particular decision maker.

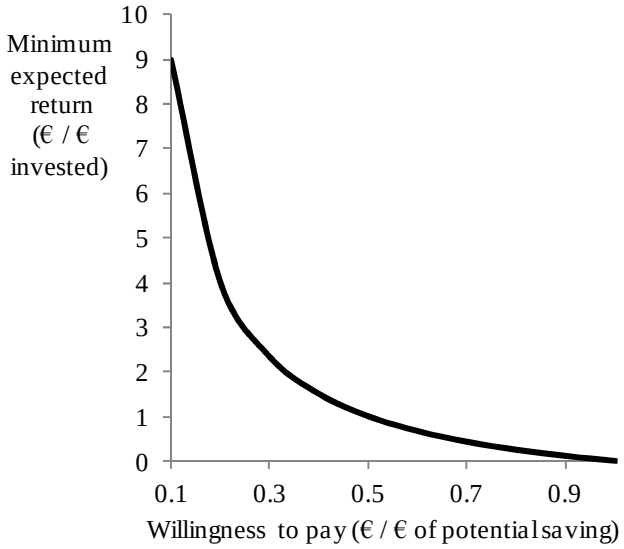

Figure 7.3. Relationship between 'willingness to pay' and minimum acceptable return on investment. With regard to management interventions to control heifer mastitis, 'willingness to pay' (k) is the maximum amount (€) a decision maker will pay / €1 of potential saving, in order that an intervention is considered cost effective. If k = 1, it is acceptable to at least break even, cover the intervention cost only and make no additional financial return for the intervention to be deemed cost effective. If k = 0.5, the decision maker would not accept < 100% return on investment, after covering the intervention costs. Values of k > 1 imply the decision maker is willing to pay more than the likely return.

7.3 Results

7.3.1 Potential savings

The prevalence of simulated cows with SCC1 ≥ 200,000 cells/mL reduced by between 12 and 14% following the interventions. This section reports potential savings, through increased lifetime milk yield, and decreased disposal risk following the management interventions, but before the expected minimum return on investment of different decision makers is considered.

For herds with ≥ 20%, or ≥ 30% of parity 1 cows with SCC1 ≥ 200,000 cells/mL that applied all three interventions, there was 75% certainty of total

savings of at least €24 or €61 /heifer calved into the herd respectively; the full range of possibilities is shown in Figure 7.4. It follows that for an example herd of 80 cows, that incorporates 20 new heifers /year, ≥ 6 of which with SCC1 ≥ 200,000 cells/mL, there would be a 75% probability of saving at least €1,220 through these interventions; further scenarios for the example herd, and an identical herd with ≥ 4 new heifers with SCC1 ≥ 200,000 cells/mL /year are shown in Table 7.5. Components of the savings are also shown in Table 7.5. Importantly, most savings are through increased revenue from the higher lifetime milk yield of cows following the interventions. There is 75% certainty of a maximum expected loss ≤ €40 through change in cow disposal risk following the interventions tested (Table 7.5). To put potential savings through decreased cow disposal risk in context; a herd in which 20 heifers calve /year, ≥ 6 of which with SCC1 ≥ 200,000 cells/mL has only 50% chance of avoiding the disposal of around 1 cow every 12 years (€1,451 / €120 (Section 1.3.4.2, Table 7.5)) through applying the specific interventions to reduce the prevalence of cows with SCC1 ≥ 200,000 cells/mL. However, there would be 50% probability of saving at least €1,360 through increased lifetime milk yield alone through applying the specific interventions (Table 7.5).

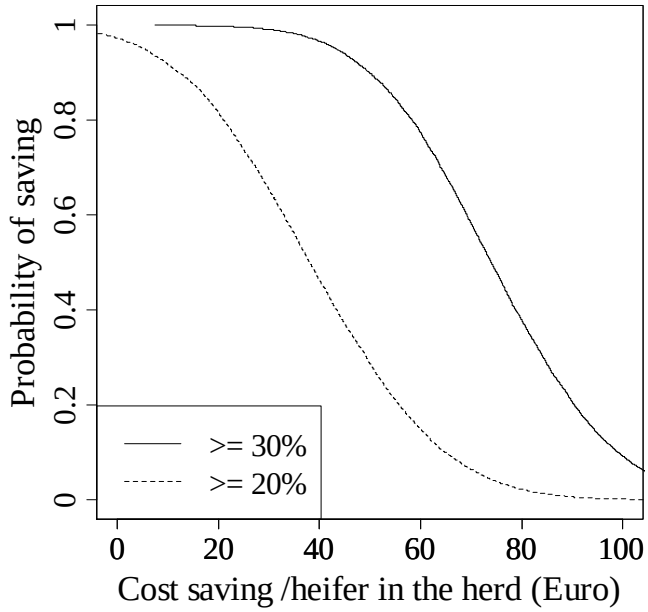

Figure 7.4. Posterior predictions of cost saving at herd level. Bayesian models for lifetime milk yield, and the binomial occurrence of disposal of cows in any 50 day interval from first calving were run in parallel. Vague prior distributions were used for all parameters, and the models were both run for 10,000 Markov chain Monte Carlo iterations following a burn-in of 1,000 iterations to allow chain convergence to occur. Model coefficients were applied to data from 1,000 theoretical cows in herds with ≥ 20%, and ≥ 30% of cows with high somatic cell count (≥ 200,000 cells/mL) between 5 and 30 days in milk during parity 1 (SCC1). Possible savings associated with keeping bedding materials inside rather than outside (change in ln SCC1 ~ Normal(-0.15, 0.02)), increasing transition yard area from < 1.25 m2 to > 1.25 m2 / 1,000 kg of milk production (change in ln SCC1 ~ Normal (-0.12, 0.01)), and ensuring that bedding in the calving area was spread evenly, instead of unevenly (change in ln SCC1 ~ Normal (-0.19, 0.05)) were simulated, assuming milk margin ~ Normal (mean 17, standard deviation = 0.03) €/L, and €1,451 /cow disposal.

Table 7.5. Components of savings associated with interventions[152] for an example herd that calves 20 heifers /year

Probability of saving	Component of saving	Initial number of parity 1 cows with SCC1 ≥ 200,000 cells/mL (/20)	
		≥ 6 (≥ 30%)	≥ 4 (≥ 20%)
0.75	Lifetime milk[153]	≥ €1,260	≥ €520
	Disposal[154]	≥ €-40	≥ €-40
	Total	**≥ €1,220**	**≥ €480**
0.5	Lifetime milk	≥ €1,360	≥ €640
	Disposal	≥ €120	≥ €120
	Total	**≥ €1,480**	**≥ €760**
0.25	Lifetime milk	≥ €1,440	≥ €760
	Disposal	≥ €300	≥ €280
	Total	**≥ €1,740**	**≥ €1,040**

[152] For housed *pre-partum* heifers the following interventions to improve environmental hygiene were implemented; bedding material storage was inside instead of outside (change in the natural logarithm of (ln) somatic cell count between 5 and 30 days in milk during parity 1 (SCC1) ~ Normal(-0.15, 0.02)), transition yard area increased from < 1.25 m^2 to > 1.25 m^2 / 1,000 kg of herd mean annual milk production (change in ln SCC1 ~ Normal(-0.12, 0.01)), and bedding in the calving area was spread evenly, instead of unevenly (change in ln SCC1 ~ Normal(-0.19, 0.05)).
[153] Minimum increase in revenue attributable to lifetime milk yield assuming a margin ~ Normal(0.17, 0.03^2) €/L
[154] Minimum increase in revenue attributable to cow disposal assuming a cost of €1,451 / cow disposed. Negative values indicate that increased cow disposal risk is possible following the interventions.

7.3.2 Cost effectiveness of interventions

This section presents the probability that interventions are 'cost effective', in terms of the maximum amount that should be spent on implementation, given the minimum expected return on investment of a decision maker.

Table 7.6 shows that for a given probability of cost effectiveness, as the minimum expected return on investment increases (decrease in willingness to pay), the potential budget for all 3 management interventions for the control of heifer mastitis decreases, and this appears more sensitive to the expected minimum return of the decision maker, than to the desired level of probability that the interventions would be cost effective. For example, in herds with ≥ 30% of cows with high SCC1, and 70%, 80% or 90% required probability that

the interventions would be ‘cost effective’, for a decision maker who would be content to at least break even, the budget for implementing the management changes should not exceed €64, €58, or €50 /heifer calved into the herd respectively (Table 7.6). However for a decision maker who requires a return on investment of at least 100%, after recovering intervention costs, the budget for implementing the management changes should not exceed €32, €29, or €25 /heifer calved into the herd for 70%, 80%, and 90% probabilities respectively of meeting this objective (Table 7.6). For herds with ≥ 20% of cows with high SCC early in the first lactation, the budget for implementing the interventions would be less, due to lower potential savings (Table 7.5). The results in herds with a lower prevalence cut off were also more sensitive to the expected minimum return of the decision maker, than to the probability of ‘cost effectiveness’ (Table 7.6). For 70%, 80%, and 90% probabilities that the interventions would be ‘cost effective’, and for a decision maker who would be content to at least break even, the budget for implementing the management changes should not exceed €27, €21, or €12 /heifer calved into the herd respectively (Table 7.6). However for a decision maker who requires a return on investment of at least 100%, after recovering intervention costs, the budget for implementing the management changes should not exceed €14, €11 or €6 /heifer calved into the herd for 70%, 80%, and 90% probabilities of ‘cost effectiveness’ respectively.

Table 7.6. Potential budgets[155] (€ /heifer calved in the herd) for varying expected minimum returns[156] against probability of the cost effectiveness[157] for specific interventions[158] to reduce the prevalence of cows with high milk somatic cell count between 5 and 30 days in milk during parity 1 (SCC1)

Initial herd prevalence of cows with SCC1 ≥ 200,000 cells/mL	Willingness to pay (k) for intervention (€ / €1 of potential saving)	Minimum expected return over intervention cost to be deemed 'cost effective' (%)	Probability of cost effectiveness			
			0.60	0.70	0.80	0.90
≥ 0.3	1.0	0	69.12	63.96	58.32	49.88
	0.9	11	62.21	57.57	52.49	44.82
	0.8	25	55.30	51.17	46.66	39.90
	0.7	43	48.38	44.77	40.82	34.92
	0.6	67	41.47	38.38	34.99	29.93
	0.5	100	34.56	31.98	29.16	24.94
	0.4	150	27.65	25.58	23.33	19.95
≥ 0.2	1.0	0	32.96	27.41	21.04	12.07
	0.9	11	29.67	24.67	18.94	10.87
	0.8	25	26.37	21.93	16.83	9.66
	0.7	43	23.07	19.19	14.73	8.45
	0.6	67	19.78	16.45	12.62	7.24
	0.5	100	16.48	13.71	10.52	6.04
	0.4	150	13.19	10.96	8.42	4.83

[155] Determined from potential change in the lifetime milk yield and disposal risk of cows.
[156] Where expected return over intervention cost = (1 – k)/k.
[157] (k x Difference in savings) - Difference in costs ≥ €0.
[158] For housed *pre-partum* heifers the following interventions to improve environmental hygiene were implemented; bedding material storage was inside instead of outside (change in the natural logarithm of (ln) somatic cell count between 5 and 30 days in milk during parity 1 (SCC1) ~ Normal(-0.15, 0.02)), transition yard area increased from < 1.25 m^2 to > 1.25 m^2 / 1,000 kg of herd mean annual milk production (change in ln SCC1 ~ Normal(-0.12, 0.01)), and bedding in the calving area was spread evenly, instead of unevenly (change in ln SCC1 ~ Normal(-0.19, 0.05)).

7.4 Discussion

This chapter has shown that the perceived 'cost effectiveness' of interventions to control mastitis in heifers is highly dependent on decision makers' willingness to pay, and hence their minimum expected return on investment. In addition, the efficacy of interventions is initially uncertain, meaning they may not always be 'cost effective' on particular farms (Green et al., 2010). Attitude to risk varies between individuals, and decisions about implementing interventions to control disease must be made based on a level of risk regarding the economic outcome that is deemed acceptable. However, 'willingness to pay' potentially has a larger impact on the intervention budget than 'attitude to risk'. The expectations of farmers when making decisions around mastitis control are not well understood, and could be affected by their psychological, physiological, and emotional state (Hastie and Dawes, 2001). For instance, pride in keeping cows healthy was an important motivator for mastitis control in Dutch dairy herds (Valeeva et al., 2007). It is hard to put an economic value on emotions such as 'pride' attributable to controlling mastitis, and this could mean 'willingness to pay' exceeds what seems rational based on changes in lifetime milk yield and disposal risk alone, as the minimum expected return is non-tangible. Farmers have cost preferences for mastitis interventions which effectively weight costs based on factors such as the practicality of implementing the changes (Huijps et al., 2009b). Decision making is therefore complicated by variation in what is deemed 'cost effective' by different individuals and this may explain the low compliance seen with voluntary mastitis control programmes (Green et al., 2007a; Valeeva et al.,

2007). Importantly, advice on ‘effective’ interventions for mastitis control may not be implemented if ‘cost effectiveness’ is ignored (Huijps et al., 2010).

Cost effectiveness analyses in human health economics often assess the relative benefit of treatments for a particular condition based on improvements in quality and quantity of life, measured subjectively as ‘quality adjusted life years (QUALYs; NICE, 2010)’. Developing similar methods to assess non-tangible benefits could help to better understand decision maker characteristics, and refine budgets for endemic disease control in livestock. However, despite use of complex modelling and sensitivity analysis in the human field (O' Hagan and Stevens, 2001; Spiegelhalter and Best, 2003), a decision must still be made on which treatment is most cost effective, determined by the maximum amount the decision maker is willing to pay per additional QALY. Although this threshold is subjective, where multiple decisions around new treatments for multiple conditions have to be made by health-care providers, cost effectiveness analyses can be used to maximise benefits from limited funds, by informing decisions on where to invest (Spiegelhalter et al., 2004). Such an economic decision tool would be invaluable to inform livestock farmers when faced with making decisions around which endemic disease(s) are a priority for investment in control measures, to maximise savings. This approach is dependent on extensive future intervention studies and cost effective analyses.

Intervention studies for the control of heifer mastitis have so far mainly focussed on individual animal treatments (Nickerson, 2009; Parker et al., 2007b; 2008). However, these require the handling of heifers, which can be dangerous for operators and animals, and there are public health risks associated with the use of prophylactic antibiotics (Borm et al., 2006;

Nickerson, 2009). These factors may limit cost effectiveness, and individual treatments are best applied alongside herd management changes (Green et al., 2007a). The majority of Irish dairy heifers calve in early spring (chapter 2), meaning they are housed during the ppp period, and poor environmental hygiene at this time is a risk factor for mastitis (De Vliegher et al., 2012). Interventions to improve the environmental hygiene of housed heifers were therefore selected for the example, from the limited available literature on the effect of management changes on SCC in early lactation (Green et al., 2007a; 2008). As a multi-factorial approach to mastitis control is advised (Anon, 2013; Green et al., 2007a), it was assumed that 3 management changes to improve environmental hygiene were applied simultaneously for ppp heifers. In practice the findings from this chapter only inform rational levels of expenditure for mastitis control in heifers through the specific management changes tested. However, even with considerable variation between decision makers, there was still potential to invest €5 per heifer in the herd for the control of heifer mastitis in a worst case scenario where the decision maker must be 90% certain of $\geq$ 150% return, in lower prevalence herds (Table 7.6); for example this could cover the cost of basic protection to keep bedding dry. Potential budgets were higher in herds with higher prevalence of cows with SCC1 $\geq$ 200,000 cells/mL, and in the best scenario investigated where the decision maker was content to be 60% certain of at least breaking even, a budget of €69 per heifer in the herd could make investment in improvements to buildings and facilities feasible (Table 7.6). For interventions to be perceived as 'cost effective', farmers should aim to implement changes for the least possible cost but within budget. In addition to the importance of environmental hygiene for heifers housed

during the ppp period, factors affecting host defences have also been identified as risk factors for mastitis, including udder oedema, nutrition, and factors relating to social integration into the herd following calving (De Vliegher et al., 2012). Furthermore, contagious transmission of pathogens between heifers can occur by cross suckling and via flies (McDougall et al., 2009). For a holistic approach to the control of heifer mastitis, further research should consider the efficacy, and hence likely budgets for different decision makers to implement management changes based on all risk factors.

The Bayesian methods used in this research differ from the classical statistical approach, which is based only on current data, and ignores any prior information. The outcome from a classical analysis gives the probability of obtaining particular study data, given a hypothesis, if the study were repeated on many occasions. Importantly, probabilities from classical analyses do not refer to parameters themselves, and therefore should not be applied in a predictive sense to inform decisions (Berry and Stangl, 1996; Bolstad, 2007). For example, a classical analysis may give a parameter mean and confidence interval (for the mean). In contrast, a Bayesian analysis gives a probability distribution for the parameter directly, that can be used for prediction and onward simulation. In this chapter the micro-simulation procedure allowed synthesis of evidence from different sources, to enable immediate predictions of the likely impact of interventions over the lifetime of cows. It also facilitated comparison between scenarios with and without an intervention over the same time period to study the effect in isolation, as if a controlled trial had been carried out. Micro-simulation is therefore a useful technique for investigation

of scenarios that would be impractical or expensive in reality (Spiegelhalter et al., 2004).

Although the underlying models have been shown to be useful and generalisable in chapters 3 and 5, further work is needed to validate the cost effectiveness analysis and budgets presented here. Ultimately, this requires observed data from management intervention studies on Irish dairy farms to compare with model predictions. In addition other costs should be considered, for instance the impact of reducing the prevalence of heifers with high SCC in early lactation on lifetime clinical mastitis costs, and milk quality. Impact on clinical mastitis was included in the costs of high SCC early in the first lactation estimated by Huijps et al. (2009a); although these were still lower than some of the potential budgets in this chapter as follow up time was only 1 year. For the cost effectiveness analysis in this chapter to be useful for decision support in practice, it should be extended to consider other endemic diseases so the relative benefits of control can be compared. A quantitative approach to determining priorities for investment would avoid reliance on subjective opinion (More et al., 2010), and this would be particularly useful for Irish farmers to inform decisions on disease control investments in conjunction with national control plans for several endemic diseases (Anon, 2013). There may be overlapping benefits of certain management changes on multiple endemic diseases which would make them even more economically favourable. A survey of Irish farmers would be useful to further evaluate their ‘risk aversion’ and ‘willingness to pay’ for disease control. This information would help refine budgets, and therefore identify achievable farm management changes for validation of efficacy in future studies.

7.5 Conclusions

Potential budgets for specific management interventions to reduce the herd level prevalence of cows with SCC between 5 and 30 DIM during parity 1 ≥ 200,000 cells/mL increase with initial prevalence. Budgets appear more dependent on the expected minimum return on investment of decision makers, than the probability of achieving the desired outcome, and hence perceived 'cost effectiveness' to the decision maker. Factors affecting the willingness of decision makers to pay for control measures require further investigation, as knowledge of rational spending limits is useful for the development of specific interventions for particular farms to control heifer mastitis, and other endemic diseases of livestock.

Chapter 8: General discussion and conclusions

8.1 Data quality versus quantity

The emphasis of the analyses in this thesis was to consider data from as many herds and cows in Ireland as possible, such that the results could potentially be generalised widely. This decision was made to produce evidence that could be applied to mastitis control on a national level (Anon, 2013). However milk recording test days occur infrequently for many Irish herds, and the raw datasets contained discrepancies. Judgements had to be made between the quality and quantity of the data used for analysis. These decisions were initially based on biological plausibility and inspection of frequency distributions. For example, the proportion of cows per herd that were recorded at each test day had a bimodal distribution that represented both routine herd recording, and test days associated with purchased cows that occurred in the herd of origin. Therefore, a graphical approach was used for selection. A herd test day was deemed to occur when at least 10% of cows were recorded (Section 2.2.1) to select a parametric distribution from the initial bimodal shape. At the cow level, there were many ways in which to summarise milk somatic cell count (SCC) during the first lactation. In order to be meaningful, some of these are conditional on a fixed number of recordings, such as the proportion of SCC recordings exceeding a threshold. In chapter 6 first lactation SCC was summarised using the geometric mean and variance to account for variable numbers of recordings, make use of the detail in continuous scales, and to avoid omitting data. To validate this approach, analyses were repeated with data that was conditional on cows having up to 8 recordings during the

first lactation but this did not substantially alter the results compared to selecting a minimum of 2 recordings. Cows with clinical mastitis were probably absent at herd test days, and would have fewer SCC records, meaning that even the large impact of high SCC during the first lactation on lifetime milk yield reported in this thesis is likely to underestimate the true cost of heifer mastitis. Inclusion of data for cases of clinical mastitis would therefore reinforce the inference that mastitis control during the first lactation is important. Additional data on cow fertility would potentially be useful to further describe the relationship between first lactation SCC and lifetime milk yield. However the focus of this thesis was to produce results that could be generalised rather than investigate more detailed biology in a smaller subset of herds with the necessary records.

8.2 Insights on aetiology

In previous research, heifers with intramammary infection (IMI) attributable to Coagulase-Negative *Staphylococcus spp.* (CNS) had higher milk yield during the first lactation than both uninfected heifers, and those infected with major pathogens; partly through reduced incidence of clinical mastitis and culling risk through the first lactation (Piepers et al., 2010). This observation highlights a further limitation of using SCC data as a proxy for IMI, as high SCC early in the first lactation may not have the same impact on milk yield for all pathogens. One way to investigate this observation using SCC data would be through considering the timing of high SCC early in the first lactation. This was investigated between 5 and 30 days in milk (DIM), but did not influence

cumulative milk yield or disposal risk. However, in previous research the impact of high SCC between 5 and 15 DIM on first lactation milk yield and culling risk depended on when it was measured (De Vliegher et al., 2005a; 2005b). This is possibly because IMI associated with CNS in heifers is likely to be present at calving, and would be relatively more prevalent before 10 DIM, as self cure can be rapid (Barkema et al., 1999a). In contrast if major pathogens are present at calving these are less likely to self cure and therefore persist beyond 10 DIM, with a more severe negative influence of milk yield and survival. When a longer early lactating period is considered, IMI are more likely to have occurred after calving, compared to during the *pre-* and *peri-partum* (ppp) period. Alternatively, the dynamics of IMI early in the first lactation may differ between countries or over time and this could explain differences between studies. In this thesis 'early lactation' for heifers was taken as 5 to 30 DIM to facilitate the simulation of management interventions in chapter 7 which was a primary aim.

8.3 Apparent prevalence of heifer mastitis

This research has used SCC as a proxy for IMI. Although this provides a slightly biased assessment for the reasons highlighted in Section 1.1.2, the approach has enabled data from thousands of farms to be evaluated. SCC during the first lactation has been shown to be economically important in terms of lifetime milk yield. Since SCC data are widely available for monitoring, and in the absence of clinical mastitis records for many herds, there is value in describing the prevalence of putative IMI in heifers throughout lactation based

on SCC. This has been included in chapter 2, and is a first step in assessing the importance of heifer mastitis in Ireland, England, and Wales. Figure 2.3 indicates a large range in the apparent herd level prevalence of heifer mastitis, which generally appears high in the first month of lactation in Ireland, England, and Wales, as has been indicated elsewhere (De Vliegher et al., 2012). However for the Irish herds the median prevalence of primiparous cows with high SCC appeared maximal and most variable towards the end of the first lactation; this was not apparent for the English and Welsh herds (Figure 2.3; Table 2.4). A possible explanation for the increased variability could be there were fewer cows in late lactation for the Irish compared to the English and Welsh herds at each milk recording date. The trend in the median prevalence highlights the importance of mastitis monitoring and control throughout the first lactation (chapter 6). Higher and more variable geometric mean SCC through lactation for heifers and cows in the Irish, compared to the English and Welsh herds (Figure 2.2; Table 2.3) may relate to differences in production systems (chapters 1 and 2), and payment schemes for milk. In Ireland, bonuses for low SCC milk are less common than in England and Wales, meaning the economic importance of mastitis may be less tangible to farmers. Furthermore, a derogation exists in Ireland that permits the 3 month rolling geometric mean bulk milk SCC to exceed 400,000 cells/mL from November to February, if this is deemed to have a ‘physiological’ basis (More, 2009). However there are no clear guidelines around how a ‘physiological’ increase in bulk milk SCC should be distinguished from a ‘pathological’ increase. The current pricing structure for milk in Ireland therefore does little to encourage mastitis control, and the subliminal message to farmers through milk pricing may be that SCC

does not matter. However, given the economic importance of low SCC in heifers identified in this thesis, it is possible that current policies are in fact counterproductive to the Irish dairy industry.

8.4 Importance of monitoring mastitis

8.4.1 Seasonal variation in milk somatic cell count

The data analysis in chapter 2 highlighted the relationship of season with individual cow SCC. After accounting for stage of lactation and milk yield, SCC for cows in Irish, English, and Welsh herds was found to be higher during spring and summer than during autumn and winter. This was consistent with higher bulk milk SCC during spring and summer for the English and Welsh herds (Green et al., 2006b), but was inconsistent with lower bulk milk SCC during spring and summer for the Irish herds (Berry et al., 2006). Spring-calving predominated in the Irish herds, whereas the English and Welsh herds predominantly had year-round calving patterns, suggesting that the difference between the datasets may be due to dilution of cells, as a result of increased milk yield during spring and summer for the Irish herds. Importantly, a high risk of new IMI during spring and summer may be overlooked if only bulk milk SCC is monitored. Furthermore, increase in bulk milk SCC during winter may be driven by IMI dynamics during spring and summer if the new IMI rate exceeds the cure rate, and Irish farmers should therefore monitor data from individual cows in addition to bulk milk SCC. This could include monitoring proportions of cows with putative IMI based on SCC thresholds for cows and heifers; both over time, and by stage of lactation. The association between

season, and cow SCC varied between herds (chapter 2), therefore herd specific target and interference levels are required. Clinical mastitis rates should be monitored in a similar manner. For cows with 2 consecutive recordings in lactation or spanning the dry period, monitoring can incorporate SCC dynamics; such as proportions of cows moving from low to high SCC, high to low SCC, remaining high, or remaining low. This approach has been applied in UK herds where monthly recording is common (Bradley and Green, 2005), and can be considered as supplementary to other monitoring methods as it is based on a subset of cows. However at present, many Irish dairy farms do not milk record frequently enough to make assessment of SCC dynamics useful for early identification of problems (chapter 2). Monitoring mastitis is of limited value unless prompt action is to be taken if herd specific targets are exceeded.

8.4.2 Herd expansion

Increase in the size of Irish, English, and Welsh dairy herds was associated with increase in cow SCC. Higher stocking rates in larger herds, and increased cow traffic could contribute to increased risk of IMI with environmental mastitis pathogens. Alternatively, cows could be more susceptible to IMI in larger herds due to stress through group changes and bullying. Larger herds may also have increased risk of IMI with contagious mastitis pathogens as more susceptible quarters could be exposed during milking. More labour units are required by larger herds. As herd size increases in line with industry trends, the number of labour units per cow decreases which may limit attention to detail in the application of mastitis control

measures. Therefore it is important that mastitis is monitored during expansion, as outlined in section 8.2.1 in order to adapt control measures as risks change.

8.5 Somatic cell count early in the first lactation and lifetime milk yield

8.5.1 Importance of heifer mastitis control

Trends for increase in herd size emphasise the importance of achieving optimal milk production and longevity from replacement heifers. This thesis has indicated that high SCC between 5 and 30 DIM during parity 1 (SCC1) had a substantial negative impact on milk production beyond the first lactation, that persisted for the entire lifetime of cows in Irish herds (chapter 3), and for at least 2 years for cows in English and Welsh herds (chapter 4). Estimates of first lactation milk loss were much larger than in previous research that considered only the impact of SCC early in the first lactation on the test day milk yield of cows that survived (De Vliegher et al., 2005a). In chapters 4 and 5, SCC1 was also associated with increased risk of disposal of cows, in agreement with previous research (De Vliegher et al., 2005b). Through the impact of SCC1 on lifetime milk yield, control measures for mastitis in ppp heifers are likely to be economically advantageous for many herds. These should involve both decreasing the risk of new IMI from environmental and contagious pathogens, as well as increasing host resistance, but further work is needed to define the relative cost effectiveness of specific interventions in different circumstances (chapter 7). Having raised awareness of the economic importance in this thesis, proportions of heifers with high SCC1 (Table 2.4; Figure 2.3) can be used to

set target and interference prevalence levels in herd monitoring schemes. This will be particularly important in Irish dairy herds where expansion is anticipated.

8.5.2 Reasons for change in lifetime milk yield

Decreased lifetime milk yield attributable to high SCC1 could be due to decreased daily milk yield while cows are alive, as a result of persistent fibrosis of the mammary parenchyma, limiting its functional capacity. The impact of high SCC1 may also be mediated through decreased longevity of cows related to ongoing poor udder health (chapter 6), increased risk of other diseases, or impaired fertility. This thesis did not identify when losses in lifetime milk yield occur, or reasons for the loss. Lifetime milk yield is correlated with survival time and in the English and Welsh herds, no differences in the relationship between survival time and cumulative milk yield for cows grouped by SCC1 were identified (Figure 8.1). This assessment was not possible for cows in the Irish herds, as test day milk recording data (required to estimate survival time) were not available beyond 2009, whereas 'lifetime milk yield' was determined up to 2012 using a separate dataset. Cow disposal depends on many factors in addition to SCC1 such as the availability of replacements, and the likely marginal profit from a replacement heifer compared to the culled cow. Herd circumstances that may relate to milk quota constraints or expansion plans, and the attitude of the decision maker are further considerations. Therefore, reasons for the reduction in lifetime milk yield associated with high SCC1 would need to be investigated in research herds to control disposal decisions. However this

approach would mean the results could not be generalised to other herds, and would be of limited use, as well as costly. Regardless of the causal pathway, this thesis has identified adverse economic consequences of high SCC1 in terms of lifetime milk yield, which emphasises the importance of heifer mastitis control.

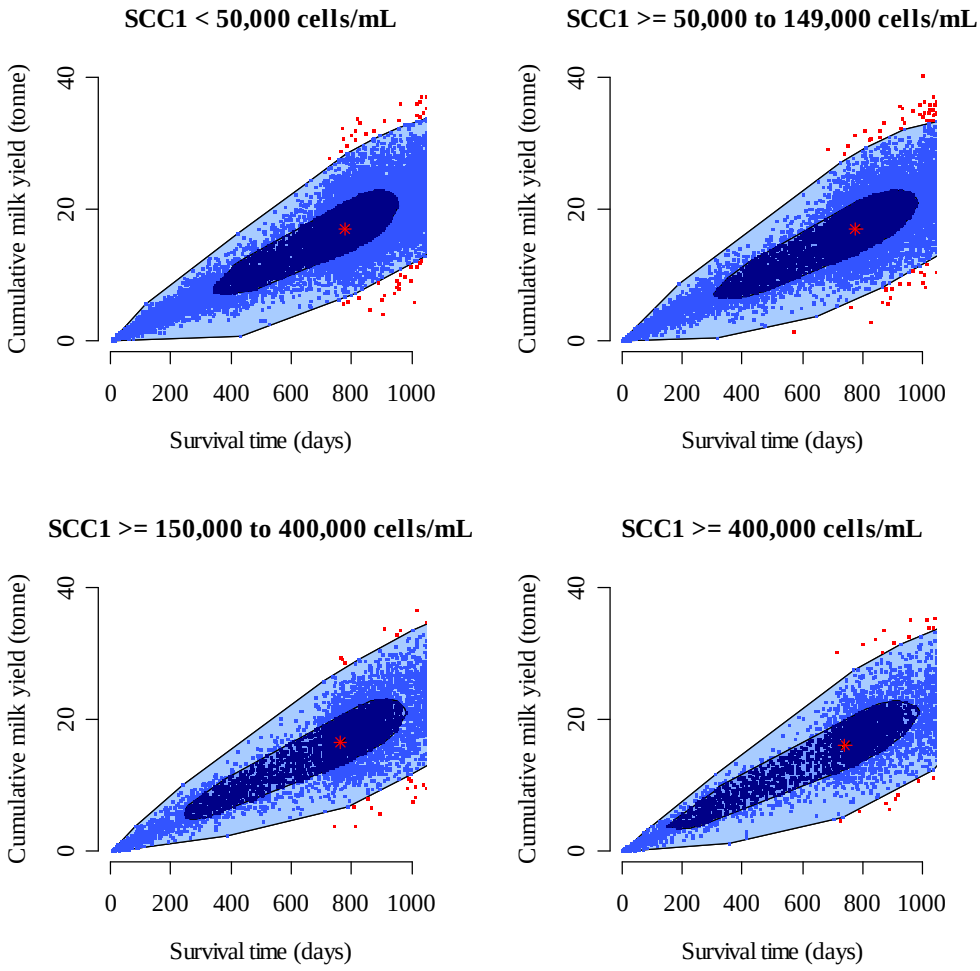

Figure 8.1. Bag plots[159] of the relationship between survival time[160] and cumulative milk yield[161] for 43,461 eligible cows[162] by SCC1[163] for in 2,111 English and Welsh dairy herds

[159] The star is the bivariate median, the inner dark blue 'bag' contains 50% of the data, and the outer boundary contains 95% of the data with outliers marked beyond this.
[160] Number of days between a first calving date in 2004, and last recording date.
[161] Estimated from a first calving in 2004 until 31 December 2006.
[162] Cows with an SCC record between 5 and 30 DIM during parity 1 during 2004.
[163] Milk somatic cell count between 5 and 30 days in milk during parity 1.

8.6 Somatic cell count legacy throughout the first lactation

In general cows with high SCC1 are more likely to have high than low geometric mean first lactation SCC, but the relationship was herd dependent (chapter 6). However, cows with high geometric mean first lactation SCC were equally likely to have either high or low SCC1, indicating that heifer mastitis may not always be associated with the ppp period. The findings from chapter 6 therefore highlight the importance of investigating patterns in first lactation SCC on a herd specific basis, in order to ensure heifer mastitis control is targeted at the appropriate risk period, being either the ppp period, or the lactating period from the second month of lactation. Targeted mastitis control throughout the first lactation is important as increases in both SCC1, and the geometric mean and variance of first lactation SCC were associated with large reductions in milk yield over the first lactation and the subsequent lifetime of cows in Irish dairy herds (chapters 3 and 6). Potential savings through heifer mastitis control give an indication of the 'scope for investment' in potential control measures. This highlights the importance of monitoring proportions of heifers with high SCC throughout the entire first lactation (Table 2.4; Figure 2.3) and setting target and interference prevalence levels.

8.7 Importance of low somatic cell count in heifers

In chapters 3, 4, and 6, relatively small changes in cow SCC during the first lactation; for example from 50,000 cells/mL to 150,000 cells/mL had a large impact on the cumulative milk yield of cows. This information can be

used to guide decisions on thresholds for putative IMI in heifers. For example as SCC1 is economically important, it may be advantageous to use low diagnostic thresholds early in the first lactation (such as < 100,000 cells/mL) to increase sensitivity, meaning more cows with IMI would be identified based on SCC1. If this led to introducing control measures sooner at the start of an 'outbreak', savings through increased lifetime milk yield could be greater. However, this decision depends on the cost effectiveness of control measures, as the loss of specificity when using a lower threshold for putative IMI, may overestimate the scale of a heifer mastitis problem and lead to unnecessary investment. A relatively small increase in SCC1 could be due to IMI with minor pathogens such as CNS in one or more quarters (Barkema et al., 1999a). Control plans targeted at specific CNS species may be cost effective for herds with heifer mastitis problems not associated with major pathogens.

8.8 Budgets for mastitis control in pre and peri-partum heifers

Rational budgets for specific interventions to control heifer mastitis are useful to ensure cost-effectiveness is achieved according to the requirements of the decision maker. The cost-effectiveness of interventions pertaining to the ppp period were investigated for sub-sets of herds (chapter 7). Budgets for specific management interventions to reduce the herd level prevalence of cows with SCC1 ≥ 200,000 cells/mL increase with initial prevalence, and appear highly dependent on the willingness of the decision maker to pay and hence the minimum return on investment that they would find acceptable.

8.9 Further research

Given the economic importance of high SCC1, its contribution to geometric mean SCC throughout the first lactation, and a paucity of knowledge around the efficacy of management changes for Irish herds, or indeed those elsewhere, defining 'cost effective' management interventions for heifer mastitis, targeted at the ppp period in different herd scenarios should be a priority for further research. Intervention studies are ultimately required to determine efficacy in terms of SCC1 (and clinical mastitis risk), and micro-simulation methods could then be applied (using models similar to those in chapters 3 to 6) to determine 'cost effectiveness' over the lifetime of cows, without having to wait a long period of time for results. However, an improved understanding of farmers' requirements in terms of their willingness to pay, or minimum expected return on investment is required to complete this work. This information could be collected at low cost in surveys, could be completed in a short time frame and would be an important next step. A more complete picture of the economic importance of SCC during the first lactation could be gained by considering the impact on clinical mastitis and the resultant costs. However, this would inevitably be based on a sub-set of herds with clinical mastitis records available that may make the results less generalisable. The contribution of clinical mastitis may be partially captured in this thesis as it is likely to reduce cow longevity. However with no information on discarded milk, the cumulative milk production of cows was all assumed to be saleable, which could potentially underestimate potential savings through the control of heifer mastitis, if clinical mastitis risk reduced alongside the prevalence of cows with high SCC. Following chapter 2, the importance of mastitis control

during spring and summer should be investigated further in terms of contribution to high bulk milk SCC during autumn and winter in Irish dairy herds, which remains a problem for the milk processing industry, particularly where herd expansion is planned.

8.10 Conclusions

8.10.1 Overview

Mastitis in dairy heifers appears to be a particular problem for the Irish dairy industry. High SCC during the first lactation was shown to have an economically important impact on the lifetime milk yield of cows. A herd specific approach to identifying the major risk period for heifer mastitis is required. Knowledge of decision maker characteristics is important to determine budgets for disease control.

8.10.2 Chapter 2

After correcting for stage of lactation and milk yield, SCC for cows in Irish, English and Welsh dairy herds was higher and more variable in spring and summer, than autumn and winter. For Irish dairy herds, monitoring individual cows is particularly important in spring and summer, despite low bulk milk SCC, and farmers should not be complacent about udder health at this time. Increasing herd size was associated with a non-linear increase in cow

SCC, highlighting an important area that may influence cost effective dairy herd expansion.

8.10.3 Chapter 3

For cows in Irish dairy herds, SCC1 was negatively associated with first lactation and lifetime milk yield. For the majority of Irish dairy herds with ≥ 10% prevalence of cows with SCC1 ≥ 400,000 cells/mL, there are likely to be large savings associated with improving udder health for *pre-* and *peri-partum* heifers.

8.10.4 Chapter 4

For cows in English and Welsh dairy herds, SCC1 was negatively associated with cumulative milk yield over approximately 2 years. For dairy herds in England and Wales with ≥ 10% prevalence of cows with SCC1 ≥ 400,000 cells/mL, there are likely to be financial savings associated with improving the udder health of *pre-* and *peri-partum* heifers.

8.10.5 Chapter 5

Despite a negative association between SCC1 and longevity for cows in Irish dairy herds, the apparent effect was small and was therefore unlikely to be economically important when considered in isolation in terms of change in replacement costs.

8.10.6 Chapter 6

For cows in Irish dairy herds, geometric mean and variance of first lactation SCC, and SCC1 were negatively associated with both first lactation and lifetime milk yield. However, the apparent legacy of SCC early in the first lactation on SCC for the remainder of the first lactation was highly herd dependent. Approximately 50% of Irish dairy herds have potential to make savings through reducing SCC throughout the first lactation. This could involve preferentially targeting mastitis control measures in a herd specific manner towards the *pre-* and *peri-partum* period, or the lactating period, depending on individual herd SCC patterns.

8.10.7 Chapter 7

Suggested budgets for specific management interventions to reduce the herd level prevalence of cows with SCC1 $\geq$ 200,000 cells/mL increase with initial prevalence, but appear more dependent on the expected minimum return on investment of decision makers, than the probability of achieving the desired outcome, and hence perceived 'cost effectiveness' to the decision maker.

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Appendix

Example code for a linear regression model including predictions and micro-simulation

The example is based on the WinBUGS 1.4.3 code for Model 3.1 with the outcome; lifetime milk yield. Coefficients (beta[k]) are estimated from the data at each simulation and used in predictions to check within-model fit (using samp_1), for cross validation (using samp_2), and for micro-simulation.

```
model
{
for (k in 1:17) { beta[k] ~ dflat() }              # Prior distribution for beta[k]
tau ~ dgamma(0.001000,0.001000)          # Prior distribution for cow level precision (1/variance)
tau.u2 ~ dgamma(0.001000,0.001000)    # Prior distribution for herd level precision (1/variance)

for(i in 1:N) {
lifetime_milk[i] ~ dnorm(mu[i],tau)
mu[i]<- beta[1]                          # Intercept
+ beta[2] * lnscc1_gm_[i]          # ln SCC1 centred on mean
+ beta[3] * calv1_mo_1[i]          # First calving in January
+ beta[4] * calv1_mo_2[i]          # First calving in February
+ beta[5] * calv1_mo_3[i]          #  First calving in March
+ beta[6] * calv1_mo_4[i]          #  First calving in April
+ beta[7] * calv1_mo_5[i]          #  First calving in May
+ beta[8] * calv1_mo_7[i]          #  First calving in July
+ beta[9] * calv1_mo_8[i]          #  First calving in August
+ beta[10] * calv1_mo_9[i]        #  First calving in September
+ beta[11] * calv1_mo_10[i]      #  First calving in October
+ beta[12] * calv1_mo_11[i]      #  First calving in November
+ beta[13] * calv1_mo_12[i]      #  First calving in December
+ beta[14] * calv1_yr_2004[i]    #  First calving in 2004
+ beta[15] * calv1_yr_2005[i]    #  First calving in 2005
+ beta[16] * calv1_yr_2006[i]    #  First calving in 2006
+ beta[17] * ln_AFC_gm_[i]       # ln AFC centred on mean
+ u2[herd_id[i]]

pred.milk[i] ~ dnorm(mu[i], tau)

 resid[i] <- pred.milk[i] - lifetime_milk[i]    # Residual

milk_gp1[i] <- lnscc1_gp1[i] * pred.milk[i]
milk_gp2[i] <- lnscc1_gp2[i] * pred.milk[i]
milk_gp3[i] <- lnscc1_gp3[i] * pred.milk[i]
milk_gp4[i] <- lnscc1_gp4[i] * pred.milk[i]


}

mean_resid <- sum(resid[])/ N                    # Mean residual

mean_milk_gp1<- sum(milk_gp1[]) / denom1
mean_milk_gp2<- sum(milk_gp2[]) / denom2
mean_milk_gp3 <- sum(milk_gp3[]) / denom3
mean_milk_gp4 <- sum(milk_gp4[]) / denom4

for (j in 1:n2) {                                # Herd level random effect
u2[j] ~ dnorm(0,tau.u2)}
for(m in 1:N2) {
```

Estimation of 'beta[k]' from data in samp_1

Prediction of mean residual and mean lifetime milk yield for cows in samp_1 grouped by ln SCC1.

```
xmu[m]<- beta[1]
+ beta[2] * (xlnscc1_gm_[m])
+ beta[3] * xcalv1_mo1[m]
+ beta[4] * xcalv1_mo2[m]
+ beta[5] * xcalv1_mo3[m]
+ beta[6] * xcalv1_mo4[m]
+ beta[7] * xcalv1_mo5[m]
+ beta[8] * xcalv1_mo7[m]
+ beta[9] * xcalv1_mo8[m]
+ beta[10] * xcalv1_mo9[m]
+ beta[11] * xcalv1_mo10[m]
+ beta[12] * xcalv1_mo11[m]
+ beta[13] * xcalv1_mo12[m]
+ beta[14] * xcalv1_yr2004[m]
+ beta[15] * xcalv1_yr2005[m]
+ beta[16] * xcalv1_yr2006[m]
+ beta[17] * ln_AFC_gm_[i]

xpred.milk[m] ~ dnorm(xmu[m], tau)
```

Prediction of lifetime milk yield of cows in samp_2

```
xmilk_gp1[m] <- xlnscc1_gp1[m] * xpred.milk[m]
xmilk_gp2[m] <- xlnscc1_gp2[m] * xpred.milk[m]
xmilk_gp3[m] <- xlnscc1_gp3[m] * xpred.milk[m]
xmilk_gp4[m] <- xlnscc1_gp4[m] * xpred.milk[m]
```

Prediction of mean residual and mean lifetime milk yield for cows in samp_2 grouped by ln SCC1

```
}
xmean_milk_gp1<- sum(xmilk_gp1[]) / xdenom1
xmean_milk_gp2<- sum(xmilk_gp2[]) / xdenom2
xmean_milk_gp3 <- sum(xmilk_gp3[]) / xdenom3
xmean_milk_gp4 <- sum(xmilk_gp4[]) / xdenom4
```

Micro-simulation

```
for(m in 1:1000){

margin[m] ~ dnorm(0.17, 1111.1111)    # Distribution of milk margin (€)

# For heifers in herds with a presumence of cows with high SCC1 >= 20%

 lnscc1_sim1[m] ~ dnorm(0.47, 0.55)       ## increase in mean lnscc1 centred on 0 from observed data

milk_20hi[m] <- beta[1] + ( lnscc1_sim1[m] * beta[2]) + beta[4] + beta[15]

 milk_20hi_cost[m] <- milk_20hi[m]  * margin[m]

# For heifers in herds with a presumence of cows with high SCC1 >= 10%

lnscc1_sim2[m] ~ dnorm(0.15, 0.65)

milk_10hi[m] <- beta[1] + (lnscc1_sim2[m] * beta[2]) + beta[4] + beta[15]

 milk_10hi_cost[m] <- milk_10hi[m]  * margin[m]


# For heifers in herds with a presumence of SCC1_hi < 5%

lnscc1_sim3[m] ~ dnorm(-0.38, 1.26)

milk_5lo[m] <- beta[1]  + (lnscc1_sim3[m] * beta[2]) + beta[4] + beta[15]

 milk_5lo_cost[m] <- milk_5lo[m]  * margin[m]

# For heifers in herds with a presumence of SCC1_hi < 10%

 lnscc1_sim4[m] ~ dnorm(-0.25, 0.96)

milk_10lo[m] <-  beta[1]  + (lnscc1_sim2[m] * beta[2]) + beta[4] + beta[15]

 milk_10lo_cost[m] <- milk_10lo[m]  * margin[m] }
```

```
# Mean cost per heifer calved into the herd

milk_20hi_sum <- sum(milk_20hi_cost[])/1000

milk_5lo_sum <- sum(milk_5lo_cost[])/1000

 milk_10lo_sum <- sum(milk_10lo_cost[])/1000

milk_10hi_sum <- sum(milk_10hi_cost[])/1000

# Differences in marginal costs for herd scenarios

diff_milk_20_5 <- milk_5lo_sum - milk_20hi_sum       # >= 20% to <5%
diff_milk_20_10 <- milk_10lo_sum - milk_20hi_sum    # >= 20% to <10%
diff_milk_10_5 <- milk_5lo_sum - milk_10hi_sum       # >= 10% to < 5%
```

Example code for a logistic regression model

The example is based on the WinBUGS 1.4.3 code for Model 5.1 for which the outcome is the binary occurrence of cow disposal in any 50 day interval from first calving.

```
model{


 for (k in 1:33)
{beta[k] ~ dflat() }    # Prior distribution for beta[k]
tau.u2 ~ dgamma(0.001000,0.001000)  # Prior distribution for cow level precision (1/variance)
tau.u3 ~ dgamma(0.001000,0.001000)  # Prior distribution for herd level precision (1/variance)

for(i in 1:N) {
disposed[i] ~ dbern(p[i])

logit(p[i])  <- beta[1]                  # Intercept
+ beta[2] * lnscc1_gm_[i]          # ln SCC1 centred on mean
+ beta[3] * milk1_gm_[i]           # ln TDY1 centred on mean
+ beta[4] * fat1_gm_[i]            # ln fat1 centred on mean
+ beta[5] * calv1_mo_1[i]          # First calving in January
+ beta[6] * calv1_mo_3[i]          # First calving in March
+ beta[7] * calv1_mo_4[i]          # First calving in April
+ beta[8] * calv1_mo_5[i]          # First calving in May
+ beta[9] * calv1_mo_6[i]          #  First calving in June
+ beta[10] * calv1_mo_7[i]         # First calving in July
+ beta[11] * calv1_mo_8[i]         # First calving in August
+ beta[12] * calv1_mo_9[i]         # First calving in September
+ beta[13] * calv1_mo_10[i]        # First calving in October
+ beta[14] * calv1_mo_11[i]        # First calving in November
+ beta[15] * calv1_mo_12[i]        # First calving in December
+ beta[16] * cull_mo_1[i]          # Disposal in January
+ beta[17] * cull_mo_2[i]          # Disposal in February
+ beta[18] * cull_mo_3[i]          # Disposal in March
+ beta[19] * cull_mo_4[i]          # Disposal in April
+ beta[20] * cull_mo_5[i]          # Disposal in May
+ beta[21] * cull_mo_6[i]          # Disposal in June
+ beta[22] * cull_mo_7[i]          # Disposal in July
+ beta[23] * cull_mo_8[i]          # Disposal in August
+ beta[24] * cull_mo_9[i]          # Disposal in September
+ beta[25] * cull_mo_10[i]         # Disposal in October
+ beta[26] * cull_mo_11[i]         # Disposal in November
+ beta[27] * ln_afc_gm_[i]         # ln AFC Centred on mean
+ beta[28] * ln_interval_gm[i]     # ln int centred on mean (polynomial)
+ beta[29] * pow(ln_interval_gm[i], 2)
```

```
+ beta[30] * pow(ln_interval_gm[i], 3)
+ beta[31] * DIM1_cat_2[i]                # DIM 100 to 199 days
+ beta[32] *  DIM1_cat_3[i]               # DIM 200 to 304 days
 + beta[33] * DIM1_cat_999[i]             # Missing data
+ u2[cow_id[i]] * cons[i]
}


for (j in 1:n2) {
u2[j] ~ dnorm(0,tau.u2)                  # Cow level random effect
}
for (j in 1:n3) {                         # Herd level random effect
u3[j] ~ dnorm(0,tau.u3)
}

}
```

Examiners

This thesis was examined by Prof. Sarne De Vliegher and Dr. Andrew Bradley.