Impact of lameness and claw lesions in cows on health and production

JN Huxley BVetMed, DCHP, DipECBHM, PhD, MRCVS

University of Nottingham, School of Veterinary Medicine and Science, Sutton Bonington Campus, Sutton Bonington, Leicestershire, LE12 5RD, United Kingdom. E-mail: jon.huxley@nottingham.ac.uk; Telephone: +44 (0)1159 516419; Fax +44 (0)1159 516440.

Abstract

Lameness is one of the most important endemic diseases of cattle, particularly in the dairy sector. It has a significant impact on health and welfare and leads to a range of production losses. This article reviews the English language peer reviewed literature on the impacts of lameness in cattle on measures of health and production.

There is a wealth of evidence from around the world demonstrating that lameness reduces milk yield. The extent of the reductions identified is difficult to summarise, however when losses have been calculated across a whole lactation, most are between 270 and 574kg. It is noteworthy that there is now strong evidence that lameness is a disease of high milk production i.e. high yielding animals are more likely to become lame. The impacts of lameness on nutrition and body condition appear complex. Overall the literature suggests that lameness leads to a reduction in the time spent feeding. A positive correlation between low body condition score and lameness has been demonstrated in a range of studies. Historically it was considered that lame cows lost weight as a consequence of the largely negative impacts of disease, on nutrition. Increasingly, evidence is appearing which suggests that the association between body condition score and lameness may in fact be the other way around i.e. high yielding cows which loose body condition during periods of negative energy balance become lame. The effect of lameness on fertility, measured in studies from around the world, is unequivocal. Lameness has substantial negative effects on fertility performance and reproductive parameters across a wide range of areas. Evidence on the association between lameness and culling is mixed. The majority of published work suggests that animals which suffer from lameness are more likely to be culled, although the converse has also been demonstrated.

A review of the literature in this area demonstrates just how substantial the negative effects of lameness are on cattle health and production. The impacts are wide ranging and significant from both a welfare and an economic performance perspective. Further work is urgently required to control this important and prevalent condition.

Key Words

Lameness, Cattle, Health, Production, Yield, Fertility

Introduction

Bovine lameness, particularly in dairy cattle, is one of the key endemic diseases causing health and welfare problems and production losses. This disease challenge is only likely to increase over the coming decades as the dairy sector attempts to increase production, to feed a growing world population. Quantifications of the impacts of the disease are vital to highlight its importance to the industry and to calculate the relative cost benefits of control interventions.

Lameness in cattle is not a single condition, rather it is a symptom of a wide range of different diseases. The aetiology and pathogenesis of many of these diseases remains relatively poorly understood (Huxley, 2012). This article reviews the English language peer reviewed literature on the impacts of lameness in cattle on measures of health and production. It is not the intention of this review to describe the individual disease conditions, the reader is referred to standard texts or review articles (e.g. Archer et al., 2010a) for further information in this area.

Impacts of Lameness on Milk Yield

There is now a wealth of evidence from around the world on the impacts of lameness on milk yield in dairy cows; peer reviewed studies from Bulgaria (Mitev et al., 2011), Chile (Green et al., 2010), Finland (Rajala-Schultz et al., 1999), France (Coulon et al., 1996), Hungary (Gudaj et al., 2012), Israel (Yeruham et al., 2000), Sweden (Pavlenko et al., 2011), the UK (Green et al., 2002; Amory et al., 2008; Onyiro et al., 2008; Archer et al., 2010b; Reader et al., 2011) and the USA (Faust et al., 2001; Hernandez et al., 2002; Juarez et al., 2003; Hernandez et al., 2005a; Bicalho et al., 2008) have all demonstrated that lameness has a negative impact on milk production.

In papers which have investigated the impacts of clinical cases of lameness, loss in production has been demonstrated for mixed causes of lameness (Coulon et al., 1996; Rajala-Schultz et al., 1999; Warnick et al., 2001; Green et al., 2002; Bicalho et al., 2008; Mitev et al., 2011) as well as for specific lesions including sole ulcers (SU) (Amory et al., 2008; Green et al., 2010), white line disease (WLD) (Amory et al., 2008), digital dermatitis (DD) (Yeruham et al., 2000; Faust et al., 2001; Pavlenko et al., 2011), interdigital necrobacillosis (Hernandez et al., 2002) and double sole (Green et al., 2010). Losses have also been demonstrated for animals identified as lame by elevated locomotion score (i.e. all animals which are identifiably lame but may not necessarily have been treated), using a range of different scoring systems (Juarez et al., 2003; Hernandez et al., 2005a; Onyiro et al., 2008; Archer et al., 2010b; Reader et al., 2011; Gudaj et al., 2012)

The extent of the losses identified are more difficult to summarise and compare because of the wide range of different definitions used to describe the lameness, the methodologies used for the analysis, the way the data are presented and the problems associated with predicting lost yield in animal which become lame. Many papers report the losses as a reduction in daily yield making total losses difficult to quantify. When authors reported milk losses over a whole lactation, with the exception of one outlier (857kg (Hernandez et al., 2002)) the total losses attributed throughout lactation are remarkably similar and in a range between 270 and 574kg (Table 1). A number of papers have described milk losses occurring after the lameness event e.g. (Rajala-Schultz et al., 1999; Warnick et al., 2001; Green et al., 2002; Amory et al., 2008). There are a growing number of papers which describe the loss of production begins before the case is identified and treated (Green et al., 2002; Amory et al., 2008; Reader et al., 2011), in some cases this can be many months before the lameness is identified.

Perhaps of note in this section is the now strong evidence that lameness in dairy cattle is a disease associated with high production. Over the last thirty years a range of papers have demonstrated that high producing animals are more likely to become lame (Rowlands and Lucey, 1986; Barkema et al., 1994; Green et al., 2002; Hultgren et al.,

2004; Sogstad et al., 2007b; Amory et al., 2008; Bicalho et al., 2008; Archer et al., 2010b). For example, in a Dutch study the odds of becoming lame was 1.06 times higher, per 100kg increase in cumulative 100 days in milk production in the preceding lactation (Barkema et al., 1994). In a later study, high yielding cows were more likely to become lame, animals which were ever lame produced 342kg of milk more (over 305 days) compared with cows which were never lame (Green et al., 2002). This effect seems particularly true for the claw horn lesions SU and WLD (Rowlands and Lucey, 1986; Barkema et al., 1994; Hultgren et al., 2004; Sogstad et al., 2007b; Amory et al., 2008). This finding is important as the true impacts of lameness on production may be masked in studies which do not take this effect into account. It is interesting to note that the increase in production of 342kg per 305 day lactation between ever lame and never lame cows (Green et al., 2002) is very similar to the *loss* of production caused by a lameness event (outlined above) i.e. it suggests that higher yielding cows which become lame return to more average production for the herd. That said, the loss of production associated with a case of lameness will be influenced by lesion severity, the speed with which a lame cow is identified and treated and the treatment protocol employed. It therefore seems likely that early and effective treatment of lesions may limit the associated loss in yield.

Impacts of Lameness on Body weight and Carcase Quality

There is limited published data on the impact of lameness caused by claw lesions on carcase quality. This may be because foot lameness is less common in growing cattle. A Norwegian study on 2,645 Norwegian Red cattle sampled from 112 herds investigated the association between lameness and carcase quality if the animals were subsequently culled. Lameness during the first lactation and in the third or greater lactation was associated with a poorer conformation score. Conversely SU in the second lactation were associated with a higher conformation class (Sogstad et al., 2007a).

The association between lameness and body condition appears complex. Historically the received wisdoms appears to have been that lame cows lost weight as a

consequence of the largely negative impacts on nutrition outlined below. For example, in a US study the prevalence of clinical lameness was significantly higher in cows with a body condition score less than or equal to 2.5 compared with higher condition scores (Espejo et al., 2006), on five Hungarian farms sound cows had condition scores significantly higher than lame cows (Gudaj et al., 2012) and in one UK herd significantly more lame cows had a low body condition score (Walker et al., 2008b). Similarly, in studies conducted in Austria and Germany, cows in the lowest quartile for body condition score were at higher risk of being lame (Dippel et al., 2009b) and Simmental cattle with a condition score of 4 or higher had lower odds of lameness compared to animals in the lowest quartile for body condition score (Dippel et al., 2009a). However a small number of studies have found no association between body condition and lameness (Heuer et al., 1999; Webster, 2001) and in one Scottish study high body condition score was associated with an elevated locomotion score i.e. a more lame animal (Onyiro et al., 2008).

Increasingly, evidence is appearing which suggests that the association between body condition score and lameness may in fact be the converse of what had previously been assumed i.e. cows in low body condition go on to become lame. A study in Germany demonstrated that cows with a condition score less than 3 at calving and 4 – 10 weeks into lactation were at significantly higher risk of suffering from lameness (Hoedemaker et al., 2009) and a Hungarian study concluded that the body condition score recorded two months previous was higher in non-lame cows compared to lame animals (Gudaj et al., 2012). Poor body condition and lameness may share a common causal risk (e.g. high metabolic load). Alternatively, the association may be mediated through the effects of body weight loss on the size of the digital cushion; the prevalence of WLD and SU was significantly associated with the thickness of the digital cushion and the thickness of the cushion was positively associated with body condition score (Bicalho et al., 2009).

Thus there may in fact be a complex vicious cycle involving milk yield, body condition score and nutrition plus a whole host of associated management factors which

influence these areas. For example, many high yielding cows mobilise body fat in early lactation to support peak yield, which causes loss of body condition predisposing them to lameness, which then alters their feeding behaviour causing a reduction in milk yield. This may well be an overly simplistic (or incorrect) interpretation of the data but could in part explain some of the complexity and discrepancies in the findings from different studies in these areas.

Impacts of Lameness on Nutrition

A number of studies have investigated the impacts of lameness on nutrition and feeding behaviour. Overall it would appear that lameness leads to a reduction in the time spent feeding although the effects appear complex. In a Spanish study, the time spent eating (28 minutes less between locomotion score (LS) 1 and 5) and the number of meals per day decreased as locomotion score increased resulting in a significant reduction in overall dry matter intake in the most lame cows (Bach et al., 2007). In two US studies, a study of 205 cows in 16 herds, increased locomotion score led to a significant reduction in time spent eating (Gomez and Cook, 2010) and whilst Cook et al found a numerical difference in feeding time as locomotion score increased (LS1: 4.5hrs; LS2: 4.2hrs; LS3: 3.8 hrs/day) the difference was not significant (Cook et al., 2004). In a UK herd, acute locomotor disorders (predominantly but not exclusively foot lesions) led to a decrease in feeding time and the number of meals per day and an increase in feeding rate (Gonzalez et al., 2008). Conversely, a number of studies have identified no differences between lame and sound animals. Galindo and Broom compared 10 lame and 10 non-lame animals and identified no differences between groups in the time spent feeding (Galindo and Broom, 2002) and in a pasture based UK herd, no difference in grazing or ruminating time was identified between lame and non-lame animals although lame cows had a significantly lower bite rate compared to their sound herd mates (Walker et al., 2008b). A Swedish study did not identify any significant differences in eating behaviour between animals affected by DD or SU compared to healthy controls although animals affected by DD spent longer ruminating whilst standing (Pavlenko et al., 2011). Finally it

182 appears that the interactions between nutrition and lameness can also occur remotely, in
183 a Canadian study, cows diagnosed with a sole lesion in mid lactation ate at a faster rate
184 and had more frequent meals during the two week period before calving. In the 24 hours
185 after calving, cows which developed lesions consumed more feed in more frequent meals
186 and during the first week after calving they consumed more feed in larger meals
187 (Proudfoot et al., 2010).

188 The impacts of lameness on nutrition and feeding behaviour appear complex.
189 Whilst alterations in behaviour at the time the animal is lame can be attributed to the
190 degree of discomfort (dependant on the diagnosis, lesion severity and treatment), the
191 temporally remote effects are more interesting and currently more difficult to explain.
192 Claw horn lesions take many months to develop. Periparturient feeding behaviour may
193 directly increase the risk of a future lameness event by increasing the pressure on the
194 support structures of the foot by altering standing times around calving (the period when
195 the pedal bone is most mobile within the hoof capsule (Tarlton et al., 2002)).
196 Alternatively periparturient feeding behaviours may increase lameness risk indirectly by
197 affecting the rate and extent of body condition score loss in early lactation and therefore
198 the association can be explained by the impacts on the digital cushion (as discussed
199 previously). Finally periparturient feeding behaviour and lameness may share an as yet
200 unidentified causal risk common to both. Further work is needed in this area to help us
201 better understand the aetiology and control of this important and painful condition.

202

203 **Impacts of Lameness on Reproduction**

204 The impact of lameness on fertility is unequivocal; data from around the world including
205 the UK (Lucey et al., 1986; Collick et al., 1989; Peeler et al., 1994; Faust et al., 2001;
206 Walker et al., 2008a; Morris et al., 2009; Walker et al., 2010), other countries in Europe
207 (Barkema et al., 1994; Hultgren et al., 2004; Sogstad et al., 2006; Kilic et al., 2007;
208 Vacek et al., 2007), the USA (Lee et al., 1989; Sprecher et al., 1997; Hernandez et al.,
209 2001; Melendez et al., 2003; Garbarino et al., 2004; Hernandez et al., 2005b; Bicalho et
210 al., 2007; Machado et al., 2010), Mexico (Argaez-Rodriquez et al., 1997), India (Sood

and Nanda, 2006; Sood et al., 2009) and New Zealand (Alawneh et al., 2011) have demonstrated that lameness negatively impacts on a wide range of measures of reproductive performance (Table 2). These effects have been demonstrated over a prolonged period of time and in cows managed in a wide range of different production systems.

The reasons for the effects on reproductive performance remain unclear, although lameness has been demonstrated to negatively impact on cyclicity and expression of oestrus. In the pre-service period, lame cows had an increased odds of delayed cyclicity (Garbarino et al., 2004), a higher incidence of ovarian cysts (Melendez et al., 2003), receive more hormonal reproductive treatments (Sogstad et al., 2006); a higher odds of receiving a treatment for anoestrus (Hultgren et al., 2004) and were less likely to ovulate (Morris et al., 2009). In three UK studies, lame cows had shorter periods when herd mates attempted to mount them (Walker et al., 2010), oestrus was less likely to be observed (Peeler et al., 1994) and severely lame cows had significantly lower oestrus intensity score and a lower frequency of total mounting activity (Walker et al., 2008a). A study conducted in India demonstrated that the frequency of standing to be mounted was significantly lower in lame animals (2.4 vs 8.0 events (Sood and Nanda, 2006)).

The physiological mechanisms behind this demonstrably strong association remain unclear. The consequences may be mediated through disturbances in reproductive hormone profiles and follicular dynamics which have been demonstrated to be disturbed in lame cows compared to sound animals (Walker et al., 2008a; Sood et al., 2009; Morris et al., 2011). This could be due to the link between lameness and nutrition. If the described changes in feeding behaviour lead to an overall reduction in dry matter intake, the resulting effects on energy status could cause infertility through impaired folliculogenesis and ovulation. Finally, it is increasingly recognised that immune cells play a role in ovarian function (Shirasuna et al., 2013). It is possible that inflammation caused by disease at remote sites, in this example the foot, may directly impact on reproductive function.

Impacts of Lameness on Culling

The majority of published work on the association between lameness and culling suggests that animals which suffer from lameness are more likely to be culled, although the converse has also been demonstrated. Studies on small numbers of herds in New York, USA demonstrated that cows with claw horn lesions were 1.7 times more likely to die or be culled (Machado et al., 2010) and the hazard ratio for culling for lame cows diagnosed in the first half of lactation was two times that of non-lame cows (Booth et al., 2004). In a much larger US study, data from 953 farms in 21 states demonstrated that the odds of a herd being in a higher category of dairy cow mortality was higher on farms classified as having a high and moderate level of lameness (McConnel et al., 2008). In a large Canadian study (6500 cows in 157 herds) the median time to culling was 188 days for cows without hoof lesions and 157 days for cows with a lesion. After modelling the data, the culling hazard ratios were significantly higher for animals diagnosed with WLD (1.72), SU (1.26) and SH (1.36) at trimming; infectious foot lesions were not associated with culling (Cramer et al., 2009). Data from 1800 cows in five herds in New York, concluded that being identified as lame in the first 70 days after calving increased the hazard ratio of culling or death, for lame and severely lame cows by 1.45 and 1.74 times respectively, compared to non-lame cows (Bicalho et al., 2007). A smaller study demonstrated that lame cows were 8.4 times more likely to be culled than non-lame herd mates (Sprecher et al., 1997). Two large Scandinavian studies have demonstrated similar findings. In a study which sampled 2645 cows from 112 Norwegian herds, lameness in the first lactation and higher SH scores in second and higher lactation animals was associated with earlier culling (Sogstad et al., 2007a). Data from nearly 40 thousand Ayrshire dairy cows in 2338 herds in Finland demonstrated that lameness made it significantly more likely that animal would be culled (Rajala-Schultz and Grohn, 1999).

A number of studies have not found any significant relationship between lameness and the risk of culling. For example, data from 2368 cows in 102 Swedish herds demonstrated no significant association between the presence of SU at trimming

and culling (Hultgren et al., 2004) and data from 13 commercial herds in the Netherlands demonstrated that the proportion of cows culled amongst animals which had a case of lameness was significantly lower than amongst cows which remained sound. The authors postulated that this may be because animals which went lame were higher yielding and thus the owner was less willing to cull them (Barkema et al., 1994).

The associations between lameness and culling may at first appear straightforward i.e. animals are culled because they are lame. However, in addition to the fact that owners may be less willing to cull lame animals, the converse could also be true i.e. animals which are scheduled to be culled are more likely to become or remain lame. There are a whole host of possible reasons why this could be the case. For example, animals which are scheduled to be culled may be considered of low value to the farm hence they receive poorer quality management or are housed in the poorest quality accommodation, making them more likely to become lame. Once lame, they may not be prioritised for treatment or they may be treated suboptimally, making them less likely to recover. These interactions could in part explain some of the inconsistencies in the literature in this area. Further work, including more social science type methodologies are required to better understand the culling and management decisions made by farmers.

Overall Economic Impact of Lameness

Whilst a number of papers have estimated the costs of various aspects of financial losses attributable to lameness (e.g. veterinary services & therapeutics (New, 1991)), over the last 20 years, only a relatively small number of peer reviewed publications have attempted to calculate the total costs. The total costs include production losses, expenses associated with culling, treatment costs, additional management time and the costs of discarded milk. The published papers all considered that the financial consequence of lameness on milk production, infertility and culling were the most significant. A UK paper published in 1997 based on 1995 prices, calculated the average total cost per affected cow was £273 (~€345). A case of digital lameness, interdigital

lameness and a SU were estimated as £213 (~€269), £113 (~€143) and £392 (~€496) respectively and increased to £240 (~€304), £131 (~€166) and £425 (~€538) if calculated as the average total cost per affected cow (Kossaibati and Esslemont, 1997). Using a partial budgeting model based on the data from 21 Dutch farms published in 1997, the total costs were calculated as 230 NLG (~€104) per affected cow and 50 NLG (~€23) per animal in the herd (average incidence 21%) (Enting et al., 1997). More recently (papers published in 2010 and 2012), using a dynamic simulation model the total costs due to foot disorders for dairy cows in the Netherlands were estimated as €53 per cow per year (Bruijnis et al., 2012) and $75 (~€60) per cow per year (Bruijnis et al., 2010). The average clinical case was estimated to cost $95 (~€76) and a subclinical case $18 (~€14) (Bruijnis et al., 2010).

Conclusions

A review of the literature in this area demonstrates just how substantial the negative effects of lameness are on cattle health and production. The impacts are wide ranging and significant from both a welfare and an economic performance perspective. Further work is urgently required to control this important and prevalent condition.

Acknowledgements

The work reported here was in part based on a projected funded by DairyCo (www.dairyco.org.uk / www.ahdb.org.uk) a levy funded, not for profit organisation working on behalf of British dairy farmers and part of the Agricultural Horticultural Development Body.

Table 1: Reported losses in milk production over a lactation, associated with a case of lameness

Report milk loss (kg)
270-440kg (Coulon et al., 1996)
314-424kg (Bicalho et al., 2008)
350kg (Archer et al., 2010b)
357kg (Green et al., 2002)
369kg (Amory et al., 2008)
372kg (Gudaj et al., 2012)
574kg (Amory et al., 2008)
857kg (Hernandez et al., 2002)

Table 2: Reported impact of lameness in cattle on measures of reproductive performance

Measure of Reproductive Performance	Reported Impact of Lameness
Calving to first service interval	8 days longer (Collick et al., 1989); 2.9 days longer (Barkema et al., 1994); 2.8 times more likely to require an interval greater than the mean (Sprecher et al., 1997); 92 vs 82 days (Kilic et al., 2007); 89 vs 82 days (Vacek et al., 2007)
Calving to conception interval	11 days longer (Lucey et al., 1986); Significantly increased (Collick et al., 1989); 113 vs 93 days (Argaez-Rodriquez et al., 1997); 140 vs 100 days (Hernandez et al., 2001); 180 vs 130 days (Hernandez et al., 2005b); 134 vs 104 days (Kilic et al., 2007); 163 vs 119 days (Machado et al., 2010); 12 days longer (Alawneh et al., 2011)
First service to conception interval	3.4 days longer (Barkema et al., 1994)
Calving interval	2% longer (Hultgren et al., 2004); Significant extended (Sogstad et al., 2006)
Days open	Significantly higher (Argaez-Rodriquez et al., 1997); 15.6 times more likely to require an interval greater than the mean (Sprecher et al., 1997); 162 vs 130 (Vacek et al., 2007); 28 days more (Lee et al., 1989)
Measures of conception	Lower conception rates (41 vs 55% (Kilic et al., 2007)); lower hazard ratio for conception (Lee et al., 1989); 0.52 times as likely to conceive (Hernandez et al., 2001); lower first service conception rate (18 vs 43% (Melendez et al., 2003); lower first service conception risk (Hultgren et al.,

	2004); a lower hazard ratio of being detected pregnant (Bicalho et al., 2007); less likely to conceive (Machado et al., 2010); a lower conception hazard (Alawneh et al., 2011)
Number of services per conception	9 times more likely to require a number greater than the mean (Sprecher et al., 1997); median 5 vs 3 (Hernandez et al., 2001); 2.45 vs 2.15 (Vacek et al., 2007); 1.35 times higher risk of conception failure (Hernandez et al., 2005b)

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