Rumen function and lameness in highly managed pasture based dairy systems

Jim Gibbs

Lincoln University, Canterbury, New Zealand
Jim.Gibbs@lincoln.ac.nz

Introduction

Lameness is routinely a top order cow health concern for dairy industries internationally (Bell et al., 2009; Harris et al., 1988; Leach et al., 2009). Physical causes of lameness are comprehensively described in the literature of the field (Chesterton et al., 1989; Choquette-Levy et al., 1985), but nutrition is often also suggested as an important cause of lameness, and contrary to popular reporting, the precise relationship between diet and lameness is comparatively poorly described. Despite this, some compromise of rumen function is almost universally suggested as the precipitating disorder when lameness is attributed to nutrition (Nocek, 1997). This discussion paper will give an overview of the development of the contemporary understanding of role of nutrition in cattle lameness, the relevance of these to pasture based systems, and recent New Zealand research on rumen function in grass based systems.

Nutrition and lameness in grain based systems

There is little doubt that rumen dysfunction as a consequence of certain diets—inappropriate excesses of cereal grains—can result in severe, readily identifiable lameness in cattle. This has been understood by livestock keepers from antiquity, and is a feature of the literature as case studies from the early history of veterinary science (Kos et al., 2006; Plaizier et al., 2008). In these clinical scenarios, commonly termed 'grain poisoning' or 'carbohydrate overload', the pathology of the affected rumen tissue and laminar tissue of the hoof are well described (Danscher et al., 2009). Case studies and research into this disorder from the 1940s also generated broad patterns of rumen environment and microbiota change (Nagaraja & Lechtenberg, 2007). As irrigation expanded the US Mid West grain crop after WWII, feedlot cattle numbers increased dramatically, and controlling rumen acidosis in grain fed cattle was arguably the single greatest management challenge for these systems (Brent, 1976). As a consequence, there is extensive literature on practical ration formulation strategies to achieve this in the period until the 1980s, as reviewed in Marie Krause & Oetzel (2006).

As part of that body of work, a sequence of metabolic and microbiological events in the rumen after severe carbohydrate overload was described (Marie Krause & Oetzel, 2006; Plaizier et al., 2008). In terms of the broad underlying understanding of the critical control factors—the provision of excess rapidly fermentable substrate and the uncompensated production of volatile fatty acids and lactic acid—contemporary research has added little to these accounts, although recent equine work has suggested some new approaches (Kyaw-Tanner et al., 2008; Milinovich et al., 2008). The actual mechanism by which rumen changes induced hoof changes in severe carbohydrate overload, however, has never been definitively settled, and there are numerous suggested alternatives. Early work looked at endotoxins released during and with the death of gram negative bacteria, postulating that these were exported to the circulation and were vasoactive at sites within or supplying the laminar tissue, thereby reducing the supply of either oxygen or nutrients (Gozho et al., 2005; Khafipour et al., 2009). Others suggested alternative vasoactive substances—particularly histamine, but including prostaglandins and leucotrienes—were produced in inappropriate amounts when rumen microbial populations were disturbed by excess dietary carbohydrates (Koers et al., 1976). More recent research in equines has suggested proteinase activation is

significant (Mungall & Pollitt, 2002). In each case, however, subsequent research to reproduce these effects by administration of these substances in otherwise healthy animals or in survey studies reported mixed results (Kos et al., 2006; Maclean, 1970; Motoi et al., 1982), so elucidation of the definitive pathway to laminar injury has remained elusive. Partly in response to this, more recent work has emphasised the specific anatomical differences between cattle and horses at the level of the bovine claw, particularly the central role of the connective tissue in weight bearing, and how this may explain difficulties in the current understanding of the aetiology (Kos et al., 2006).

From the 1970s to 1980s, acute laminitis was still considered an expression of acute rumen 'overload' in cattle, but another allied concept with subtle but important differences began to be considered. There began to be discussion of another expression of low rumen pH, where the tissues of the feet were affected in a manner that was not clinically evident—a 'subclinical laminitis'—that predisposed cattle to lameness at a later date. In this scenario, a less severe rumen acidosis that was not clinically evident—'subacute rumen acidosis' (SARA)—in otherwise healthy grain fed cattle was considered responsible for various health and production impacts (Nocek, 1997).

It is important to note here that the rationale of this approach was based on the understanding of rumen pH response to starch seen in 'grain poisoning' cases. It assumed there was a generally robust and broadly linear progression in rumen responses from 'normal' to 'unsatisfactory' pH, so a number of the systemic impacts identifiable at severe acidosis pH thresholds (4.0 – 5.0) would be similarly present at higher pH, albeit proportionately reduced. For example, microbial death and the production or release of vasoactive compounds within the rumen were postulated to occur at some level with pH between 5.5 and 6.0 (Marie Krause & Oetzel, 2006), although by the time clinical lameness is apparent in these severe cases the rumen pH is routinely well below that.

There have been, however, some clear demarcations between severe acidosis and SARA suggested, and one example is the lack of appreciable lactic acid in typical SARA (Dohme et al., 2008). The original threshold values described for SARA were then assumed to be at some point between the very low pH (5.0) seen with severe acidosis and the 'normal' pH (>6.0) seen with standard diets, but gradually came to be defined by functions of certain microbial sub-populations. For example, Russell & Wilson (1996) demonstrated cellulolytic bacterial populations functioned better above pH 6.0, and Veth & Kolver (2001) showed a slight but significant reduction in fibre digestion below pH 5.8. There is a broad consensus in the TMR literature of a SARA threshold between pH 5.5 and 5.8 (Marie Krause & Oetzel, 2006; Nocek, 1997; Yang & Beauchemin, 2006).

There are numerous impacts of SARA on systemic physiology beyond the rumen that have been suggested; liver abscessation, reproductive performance, milk production and milk fat yield are some prominent examples (Marie Krause & Oetzel, 2006). However, the effect of SARA on lameness in particular has been emphasised. One reason for this could be the assumption that laminitis could be proportionately induced to a greater or lesser severity by the relative amounts of active compounds released from the rumen. In this paradigm, when the laminitic insult was mild, an increase in clinical lameness was thought to result by some undescribed negative effect of these compounds on the subsequent horn growth rate or quality which predisposed the sole, wall or white line to lesions, rather than by typically identifiable laminitis (Nocek, 1997).

From the late 1980s forward there was a series of studies reported investigating the link between lameness and the presence of SARA, and the one common feature was the proposition that white line disease was the lameness category most associated with SARA (reviewed by Nocek 1997). An extension of this work was the investigation of supplementary biotin in dairy cattle, as some studies had suggested post rumenal biotin supply may be reduced in SARA cows (Midla et al., 1998; Santschi et al., 2005). One widely cited British study reported an improvement in white line disease incidence with biotin supplementation (Hedges et al., 2001), and an Australian study

suggested a slight improvement in herd lameness with supplementary biotin (Fitzgerald et al., 2000).

As contemporary research has more closely defined SARA thresholds in terms of microbial activity and function, SARA has come to be a central driver in ration formulation in dairy total mixed ration (TMR) systems internationally (Yang & Beauchemin, 2006).

There are a number of strategies employed to prevent SARA in TMR systems, but the dietary supply of structural carbohydrate in a physical form that promotes rumination is topical because it is directly relevant to the contemporary dialogue around rumen function in pasture based systems. This approach was a result of the work of Mertens (1997) who demonstrated in TMR fed cows a significant increase in chewing minutes per day (including eating and rumination) with increased NDF supply in a physical form of diet particle size large enough to be retained on a sieve screen of a defined pore size. On the basis that increased rumination led to increased rumen pH due to salivary buffering, this 'physically effective fibre' supply was widely adopted to prevent SARA in TMR systems (Yang & Beauchemin, 2006).

Rumen pH in pasture based systems

Given that research from grain based systems dominates the field of nutritional influences on lameness, it is reasonable to ask what direct connection this might have with lameness in pasture based systems. Pasture based dairy systems were historically not considered to be at risk of laminitis, as rumen acidosis was understood as a grain based disorder. However, as many Australian pasture based herds use supplementary grain, there was a concern that SARA may be induced this way (Fitzgerald et al., 2000). In addition, there have been significant advances in improving energy density of perennial ryegrass cultivars, protein and water soluble sugar content have been rising (Woodfield & Easton, 2004), and highly regimented pasture management systems have been adopted, particularly in New Zealand, that result in pasture dry matter digestibility above 80% (Lambert et al., 2004). In addition, the highly managed pastures are of low structural carbohydrate content relative to pastures from decades before.

It was argued that these fibre levels could be regarded as sub-optimal by extrapolation of the TMR research on rumination drivers (Tacoma et al., 2004). It was extrapolated that such a diet shared characteristics – energy density, proportions of rapidly fermentable and structural carbohydrates and rapidly degradable protein – of the TMR diets associated internationally with SARA, so by extension the diet energy content and physical structure may predispose some pasture based cows to SARA (Westwood et al., 2003). At the same time, there were several studies on rumen pH at grazing which suggested lower values than those historically associated with pasture based cows (Wales & Doyle, 2003; Williams et al., 2005). Taken together, these studies gave impetus to the application of the SARA and laminitis idea for pasture based cows.

However, the application of rumen pH research generated from TMR systems to pasture based systems has been demonstrated to have several important limitations. Kolver et al. (1998) showed that the widely used Cornell model did not reliably predict rumen pH in pasture based cows, and also demonstrated that establishing optimal fibre content was not possible by uncritically using TMR system data and approaches (Kolver & Veth, 2002).

Importantly, several studies on supplementary fibre (straw) to pasture based dairy cows in Australia (Wales & Doyle, 2003) and New Zealand (Tacoma et al., 2004) have failed to either demonstrate any increase in rumen pH or any reduction in lameness, and both groups observed a reduction in milk yields in straw supplemented cows. Nevertheless, in both Australia and New Zealand there has been interest from the veterinary profession and the dairy industry in the role of nutrition broadly, and SARA particularly, in dairy lameness (Tacoma et al., 2004; Westwood et al., 2003).

High quality pastures and lameness in the South Island of New Zealand

In the South Island of New Zealand dairy industry, there has been a view that lameness levels were increasing, and were above those of the traditional dairy areas of the North Island. A nutritional cause was not uncommonly suggested for lameness in these systems, as it was in some quarters attributed to the higher quality pastures of the region, and particularly the high water soluble carbohydrate content of the grass given the long daylight latitudes. There was a strong veterinary voice that fitted the clinical findings to the idea that SARA was common and the high lameness rates were a result (Tacoma et al., 2004).

Although this concept of a nutritional cause for South Island lameness rested on three readily identifiable factors—higher recorded lameness rates; high quality pastures on commercial farms; and compromised rumen function (often summarized as the effect of pH on the microbes) in these scenarios—by 2004 none of the three had been systematically assessed in the Australia or New Zealand dairy industries.

A research group of dairy farmers, consultants, and Lincoln University veterinarians and scientists undertook a project to gather information in each of these three areas in the South Island. The work was funded by the New Zealand Ministry of Agriculture Sustainable Farming Fund and the national dairy research body 'Dairy NZ'. The research had two 'arms': an on-farm recording system for lame cases treated and pasture sampling, and an experiment series using rumen fistulate cows to determine rumen function parameters in the Lincoln University Dairy Farm (LUDF) herd. In this paper only the latter will be discussed.

The rumen function experiments (2006-2009) were undertaken to provide data to answer several simple questions: what is the diurnal pattern of rumen pH in cows grazing high quality, highly managed pastures in the typical South Island large herd; and was this feed management system and subsequent rumen environment associated with rumen dysfunction (as assessed by rumen metabolite measurement and microbial community profiling) similar to that suggested as a cause of herd lameness in TMR systems?

The work used a novel system of datalogging actual rumen pH and temperatures in fifteen fistulate cows that were part of the LUDF herd of 670 cows. Small backpacks were used to hold the dataloggers that were connected to probes on the floor of the rumen, and for several days each month these were continuously recorded to the dataloggers. Direct rumen, faecal and urine sampling was also done across these periods. Specific rumen metabolites (volatile fatty acids, ammonia) were assessed and microbial communities were also analysed using specific molecular biology techniques of polymerase chain reaction based denaturant gel gradient electrophoresis.

The rumen pH and metabolite diurnal patterns and the microbial community profiling obtained in these experiments suggested there are several important rumen function differences between grass based and TMR systems that should be considered when applying international research to grazing cattle. First, there are consistently intervals of relatively low pH (5.0-5.5) and significant periods of the total day could be recorded as having rumen pH at this level.

However, the grazing intake patterns across the day are very different to the distribution of intake with TMR, and the observed influence of multiple daily grazing bouts on pH, which shifted rapidly and frequently across the diurnal range, suggested grass based rumen pH was more dynamic. Rumen metabolite analysis supported the marked variation in rumen function diurnally. This 'dynamism' of rumen pH appeared stable and consistent across cows and seasons, which did not suggest any compromised rumen pH homeostasis.

Second, the measured concentrations of volatile fatty acids and ammonia across the experiments were closely associated with these rumen pH shifts; were consistent with values expected from high production cows; and did not suggest any rumen dysfunction.

Third, in these dynamic rumens the microbial communities in individual cows appeared very stable across the diurnal cycle, the annual cycle, and even between years. There was no evidence of any significant microbial community fluctuation as a consequence of the recorded rumen pH.

The results of the research over this period when considered together with the high milk solids production and excellent reproductive performance of the experimental cows over the duration of the research, do not provide evidence to indicate the recorded rumen pH were associated with compromised rumen function. The rumen environments of these grazing cows appeared highly adapted to the typical large herd, highly managed pasture based system in place at LUDF.

Conclusions

There is strong interest in the role of rumen function in cow production and health in TMR systems internationally, and much of the current approach is linked to the prevention of rumen acidosis, using the concept of SARA. While the literature around rumen pH measurement is steadily increasing for TMR systems, and the association of SARA with reductions in herd production or health is increasingly being explored, significant uncertainty remains about any definitive mechanism by which lameness may be induced as a consequence of SARA.

For pasture based systems in Australia and New Zealand, there are also additional difficulties with the extrapolation and use of the international TMR research here. The early research in pasture based rumen pH indicates there may be significant differences in rumen function compared with TMR systems, which suggests caution should be used in the uncritical application of TMR derived pH targets, thresholds and ration formulation principles in pasture based systems. It also highlights the value of system specific research in rumen science, and the ongoing need for 'local' research in rumen function.

References

Bell, NJ, Bell, MJ, Knowles, TG, Whay, HR, Main, DJ & Webster, AJF 2009, The development, implementation and testing of a lameness control programme based on HACCP principles and designed for heifers on dairy farms, *Veterinary Journal* 180, 178-188.

Brent, BE 1976, Relationship of acidosis to other feedlot ailments, *Journal of Animal Science* 43, 930-935.

Chesterton, RN, Pfeiffer, DU, Morris, RS & Tanner, CM 1989, Environmental and behavioural factors affecting the prevalence of foot lameness in New Zealand dairy herds - a case-control study, *New Zealand Veterinary Journal* 37, 135-142.

Choquette-Levy, L, Baril, J, Levy, M & St-Pierre, H 1985, A study of foot disease of dairy cattle in Quebec, *Canadian Veterinary Journal* 26, 278-281.

Danscher, AM, Enemark, JMD, Telezhenko, E, Capion, N, Ekstrm, CT & Thoefner, MB 2009, Oligofructose overload induces lameness in cattle, *Journal of Dairy Science* 92, 607-616.

Dohme, F, DeVries, TJ & Beauchemin, KA 2008, Repeated ruminal acidosis challenges in lactating dairy cows at high and low risk for developing acidosis: ruminal pH, *Journal of Dairy Science* 91, 3554-3567.

Fitzgerald, T, Norton, B, Elliott, R, Podlich, H & Svendsen, O 2000, The influence of long-term supplementation with biotin on the prevention of lameness in pasture fed dairy cows, *Journal of Dairy Science* 83(2), 338-344.

Gozho, GN, Plaizier, JC, Krause, DO, Kennedy, AD & Wittenberg, KM 2005, Subacute ruminal acidosis induces ruminal lipopolysaccharide endotoxin release and triggers an inflammatory response, *Journal of Dairy Science* 88, 1399-1403.

Harris, DJ, Hibburt, CD, Anderson, GA, Younis, PJ, Fitspatrick, DH, Dunn, AC, Parsons, IW & McBeath, NR 1988, The incidence, cost and factors associated with foot lameness in dairy cattle in south-western Victoria, *Australian Veterinary Journal* 65, 171-176.

Hedges, V, Blowey, R, O'Callaghan, C, Packington, A & Green, L 2001, A longitudinal field trial of the effect of biotin on lameness in dairy cows, *Journal of Dairy Science* 84, 1969.

Khafipour, E, Krause, DO & Plaizier, JC 2009, A grain-based subacute ruminal acidosis challenge causes translocation of lipopolysaccharide and triggers inflammation, *Journal of Dairy Science* 92, 1060-1070.

Koers, WC, Britton, R, Klopfenstein, TJ, & Woods, WR 1976, Ruminal Histamine, Lactate and Animal Performance, *Journal of Animal Science* 43, 684-691.

Kolver, ES, Muller, LD, Barry, MC & Penno, JW 1998, Evaluation and application of the Cornell Net Carbohydrate and Protein System for dairy cows fed diets based on pasture, *Journal of Dairy Science* 81, 2029-2039.

Kolver, ES & Veth, MJ d 2002, Prediction of ruminal pH from pasture-based diets, *Journal of Dairy Science* 85, 1255-1266.

Kos, J, Babic, T, Vnuk, D, Dzaja, P & Smolec, O 2006, Bovine laminitis, *Hrvatski Veterinarski Vjesnik* 29, 5-15.

Kyaw-Tanner, MT, Wattle, O, Eps, AW v & Pollitt, CC 2008, Equine laminitis: membrane type matrix metalloproteinase-1 (MMP-14) is involved in acute phase onset, *Equine Veterinary Journal* 40, 482-487.

Lambert, MG, Clark, DA & Litherland, AJ 2004 Advances in pasture management for animal productivity and health, *New Zealand Veterinary Journal* 52, 311-319.

Leach, KA, Dippel, S, Huber, J, March, S, Winckler, C & Whay, HR 2009, Assessing lameness in cows kept in tie-stalls, *Journal of Dairy Science* 92, 1567-1574.

Maclean, CW 1970, The haematology of bovine laminitis, *Veterinary Record* 86, 710-714.

Marie Krause, K & Oetzel, GR 2006, Understanding and preventing subacute ruminal acidosis in dairy herds: A review, *Animal Feed Science and Technology* 126, 215–236.

Mertens, DR 1997, Creating a system for meeting the fiber requirements of dairy cows, *Journal of Dairy Science* 80, 1463-1481.

Midla, L, Hoblet, K, Weiss, W & Moeschberger, M 1998, Supplemental dietary biotin for prevention of lesions associated with aseptic subclinical laminitis (pododermatitis aseptica diffusa) in primiparous cows, *American Journal of Veterinary Research* 59, 733.

Milinovich, GJ, Burrell, PC, Pollitt, CC, Klieve, AV, Blackall, LL, Ouwerkerk, D, Woodland, E & Trott, DJ 2008, Microbial ecology of the equine hindgut during oligofructose-induced laminitis, *ISME Journal* 2, 1089-1100.

Motoi, Y, Obara, Y & Hayashi, M 1982, Histamine concentration in blood and contents of digestive tract in cattle overfed concentrate, *Japanese Journal of Veterinary Science* 44, 9-14.

Mungall, BA, & Pollitt, CC 2002, Thermolysin activates equine lamellar hoof matrix metalloproteinases, *Journal of Comparative Pathology* 126, 9-16.

Nagaraja, TG & Lechtenberg, KF 2007, Acidosis in feedlot cattle, *Veterinary Clinics of North America, Food Animal Practice* 23, 333-350.

Nocek, JE 1997, Bovine acidosis: implications on laminitis, *Journal of Dairy Science* 80, 1005-1028.

Plaizier, JC, Krause, DO, Gozho, GN & McBride, BW 2008, Subacute ruminal acidosis in dairy cows: the physiological causes, incidence and consequences, *Veterinary Journal* 176, 21-31.

Russell, JB & Wilson, DB 1996, Why are ruminal cellulolytic bacteria unable to digest cellulose at low pH? *Journal of Dairy Science* 79, 1503-1509.

Santschi, D, Berthiaume, R, Matte, J, Mustafa, A & Girard, C 2005, Fate of supplementary B-vitamins in the gastrointestinal tract of dairy cows, *Journal of Dairy Science* 88(6), 2043.

Tacoma, H, Kuperus, W, Hockstra, F & Troost, M 2004, Sole Horn Quality and Dietary Fibre Intake: A New Zealand Perspective, *In* "Dairy Vets Newsletter", Vol. 2 (22), pp. 4-13.

Veth, MJ d & Kolver, ES 2001, Digestion of ryegrass pasture in response to change in pH in continuous culture, *Journal of Dairy Science* 84, 1449-1457.

Wales, WJ & Doyle, PT 2003 Effect of grain and straw supplementation on marginal milk-production responses and rumen fermentation of cows grazing highly digestible subterranean clover pasture, *Australian Journal of Experimental Agriculture* 43, 467-474.

Westwood, CT, Bramley, E & Lean, IJ 2003, Review of the relationship between nutrition and lameness in pasture-fed dairy cattle, *New Zealand Veterinary Journal* 51, 208-218.

Williams, YJ, Doyle, PT, Egan, AR & Stockdale, CR 2005, Increasing the intake of highly digestible Persian clover herbage reduces rumen fluid pH and the rate of degradation of neutral detergent fibre in grazing dairy cows. *Australian Journal of Experimental Agriculture* 45, 1529-1537.

Woodfield, DR & Easton, HS 2004, Advances in pasture plant breeding for animal productivity and health, *New Zealand Veterinary Journal* 52, 300-310.

Yang, WZ & Beauchemin, KA 2006, Effects of physically effective fiber on chewing activity and ruminal pH of dairy cows fed diets based on barley silage, *Journal of Dairy Science* 89, 217-228.