

## Rumen Function and Lameness in South Island Cows

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### ***Introduction***

A South Island farmer whose opinion on these matters I genuinely respect once explained to me why he was very careful with how his managers spent their working day:

*“Cows don’t make me money, grass does. If my manager is dragged into spending lots of time on the cows (feet, mastitis etc) then soon his eye is off the pastures and that will end up costing me money.”*

If grass is the goose that lays the golden egg in the NZ dairy industry, then the dairy rumen is the nesting box that holds that egg for collection and sale. That being so, the rumen should be the best researched cubic metre in NZ. But it isn’t, and there is a tremendous amount of really important knowledge on how the rumen operates - digestion, movement, microbes - that we just don’t have. As a ruminant nutrition colleague of mine once agreed: *“All we really know about the rumen is that it is dark inside there.”* To further complicate matters, a large part of the rumen research we use here in NZ is drawn from work done overseas, routinely on cows fed lots of grain, while there is perishingly little hard data on the high production, grass based rumens that are the direction NZ dairy appears to be heading in. Despite this, or maybe because of this, there are a considerable number of loud and strong opinions around rumen function here in NZ, and particularly about the high producing cow. Although it seems there are a lot of issues around rumen function spoken about, in reality there is just one central theme that is most commonly discussed, and this is just recycled in different forms. This paper will look at what we know, and don’t know, about rumen function in grass based dairy cows under NZ conditions, and outline the results of the three years of rumen research on the Lincoln University Dairy Farm (LUDF), and what this tells us about the role of the rumen in lameness here in the South Island.

### ***Houston, is this rocket fuel crashing the engine.....?***

The rumen issue most commonly discussed is the idea that poor or sub-optimal rumen function results from high intakes of high quality grass. The idea appears in various forms – the grass has too much sugar, too little fibre, too much protein etc – and is blamed for lots of problems – lameness, lost production, liver abscesses, infertility, loose faeces, thiamine deficiency, early season metabolics, poor feed conversion efficiency, milk protein/fat issues etc. However, the central theme is straightforward –

rumen 'acidosis' (ie. unsatisfactorily low pH), and because there is a lot of confusion around this, it is worth briefly outlining what is meant when people use this term.

The rumen transfers energy from the diet to the cow by producing acids from carbohydrates during digestion. These acids are then absorbed through the rumen wall, used by microbes, flushed through past the rumen, or 'buffered' by compounds in the rumen fluid – saliva from chewing, or components of the plants themselves. If the rate of acid production in the rumen exceeds their removal or buffering (diets with a high content of rapidly digestible carbohydrates like starch or sugar), the sinking pH that results ('acidosis') eventually impacts on the rumen microbes and therefore, digestion and production. If it sinks low enough, it causes health problems directly.

The broad understanding of the impact of low rumen pH has been understood for about a hundred years, developed initially from observing cattle dying after gorging on grain. They commonly died rapidly, but if they lived long enough, they often developed other problems – extreme lameness due to inflamed tissues in the feet ('laminitis'), rumen wall damage, liver abscesses, and others. With the rise of feedlot systems after the second world war, rumen acidosis was identified as the primary nutritional disorder requiring control in grain fed cattle. However, in the 1970's, these principles observed in 'grain overload/poisoning' began to be applied to another scenario – it was suggested lameness and poor production was being induced by ongoing, low grade ('sub-clinical' or 'sub-acute') 'acidosis' in otherwise healthy grain fed cattle. If you accepted it, the logic was simple – if lots of grain caused really low rumen pH (a major problem), and no grain meant 'normal' pH (no problem), then somewhere in between was a threshold where the problems began. Just over that threshold somewhere was called 'sub-acute ruminal acidosis' (SARA). Lameness was linked with this on the basis that cattle with SARA may have mild, recurring laminitis that only showed itself by increased rates of 'normal' (sole or white line) lameness because of the damage it caused to the tissues that grew the foot horn (Nocek 1997).

There are two obvious questions about SARA. Firstly, what is the pH 'threshold' for SARA, and secondly, how was it established? The early consensus of researchers suggested a pH threshold of < 6.0 to define 'acidosis' by simply measuring pH in normal rumens. It is interesting to note that as more is understood about rumen function, and improved pH measurements are done, the threshold keeps dropping! The most recent reviews of the research topic seem to broadly agree around 5.5 (Marie Krause and Oetzel 2006). The research in this field used numerous methods to establish this SARA pH threshold: for example, measuring rumen pH in normal and grain poisoned cattle; measuring fibre digestion at different pH; monitoring microbe growth; measuring rumination cycles. On that basis, it is important to recognise these two points about the currently accepted pH thresholds used to diagnose

SARA: they are drawn largely from research on grain based diets, and they are built from information from many sources – they are not crisp, clear physiological guidelines.

The other aspects of research around rumen pH that are relevant to the discussion are the methods developed to control it. While there are several groups of additives used – adding buffers, antibiotics to reduce certain rumen microbes - the mainstay is manipulation of the diet by the supply of fibre. This is a particularly important topic in grass based cows because there is a lot of misinformation peddled about it. International research on grain fed cattle demonstrated that a certain proportion of fibre of a sufficient length ('physically effective fibre') in the diet was needed to stimulate chewing the cud, thought to be done by physically 'scratching' the rumen wall to initiate the rumination reflex (Mertens 1997). This improved rumen pH in grain fed cattle because saliva is added to the rumen when chewing occurs, and it contains several buffers. From this work, the concept that if pH values in the range categorised as SARA occurred the diets must be too low in fibre became very popular, and has even made its way into NZ grass based dairying.

However, there are a few reasons why we need to be careful in applying this work to grass based cows – which usually means recommending the feeding of straw. Firstly, there is no easy translation of physically effective fibre content from mixed rations to pasture diets. There are different types of fibre, so, for example, the fibre in cereal straw behaves differently in the rumen to that in high quality pastures. Fibre 'length' in pastures is also not adequately understood, so attempts to develop physically effective fibre systems for grass based systems or predict rumen pH from pasture diets have been difficult (Kolver and Veth 2002).

Secondly, rumen contents with grain fed rations with straw are very different to those with diets of high quality pastures. As anyone who has spent a lot of time with hands inside rumens will testify, there is no textbook fibrous 'mat' on top of a more fluid layer in high intake cows on quality pastures – it is just a large, homogenous bulk. In grain based diets, it is the fibrous 'mat' that is believed to initiate rumination. However, the research to date does not make it clear that rumen movements, including rumination, are driven only by physically effective fibre in grass based rumens. Certain rumen movements increase with greater intakes, and it may be that rumen fill, rather than fibre content, is the important driver of rumination in high intake grass based cows.

Thirdly, we need to be careful not to uncritically assume that cows ruminate only because they are responding to rumen signals to do so for rumen health. For example, cows with lower intakes are able to spend less time grazing, leaving more 'free' time, and this time may be spent resting or ruminating simply because it is a reflex to do so. In such cases attempting to explain the amount of rumination observed by the fibre content of the pasture is more difficult.

### ***Theories linking rumen function and lameness on grass***

So, when poor rumen function on high quality pastures is typically discussed in NZ, it is almost always around the theme of ‘acidosis’. The traditional idea was that ‘acidosis’ was only a grain fed disorder, not seen in pasture based cattle. But more recently it has been suggested that contemporary pastures on highly managed farms are producing the disorder on the same basis as grain - the diet being too high in energy and too low in fibre. The idea has been that the South Island produces very high quality pastures that are used in a grazing system of low residuals and short rotations that maintains high ME and low fibre content across the season. In addition, the latitude means long sunny days and cool nights, which promote high sugar content. In combination with a high per cow production (intake), it has been suggested that these factors have resulted in a diet that shares some features of grains.

The link to lameness is by an extension of this idea. There have been suggestions from various sectors of the industry that given the contemporary pastures, SARA may be widespread. In turn, this rumen dysfunction is postulated to induce sub-clinical laminitis, and increased lameness due to poor horn quality or growth is the suggested result. Although there are a large number of ideas about just how an abnormally low rumen pH might cause laminitis, it is important to note that rumen dysfunction is central to this theory. It is also worth noting that although sub-clinical laminitis receives all of the media attention, there are numerous other mechanisms apart from rumen dysfunction – for example nutrient deficiencies, partitioning of nutrients across the lactation cycle, endocrine influences, and others – by which nutrition could influence lameness in cows.

### ***Outline of LUDF Rumen Function Research***

In 2005 there was no existing research on rumen function in high production grass based cows in typical South Island systems, or of any association of this with lameness. In response to this need, the South Island Dairy Lameness Project, funded by SIDE, SFF and Dairy NZ, began a large series of experiments on the LUDF to investigate rumen function in a typical commercial enterprise.

To investigate rumen function satisfactorily, several parameters must be measured: the pH; the acids and ammonia; the microbes; and the movement. In this project, several new methods of measuring these parameters were specially developed for use. Specific molecular technology methods for assessing the rumen microbe populations were also imported here for this work. To be relevant, it is also important that these parameters are measured in conditions that mirror genuine working farms, rather than on research farms in highly controlled conditions. To do this, 15 cows in the LUDF herd were fitted with rubber rumen cannulae to allow open access to the rumen, and trained to wear a

backpack. The backpack held several small computers cabled to probes that were placed through the cannulae and weighted to sit at the bottom of the rumen. Once a month the backpacks and probes were fitted to 5 cows for 4 days and the rumen pH, temperature and pressure were continually recorded. The cows remained with the herd at all times. Samples of the rumen contents were collected and analysed twice weekly for acids, ammonia and microbe populations.

In addition, experiments were done to assess the effect of a number of specific diet manipulations (eg. straw feeding) on rumen function. In these, two groups of 5 cannulated cows were used, with all cows remaining in the herd. One group was fed the treatment for an appropriate warm up period, then both groups were fitted with the backpacks and probes, and the rumen parameters were recorded. The groups were then swapped over and the measurements repeated. For the straw treatment the experiments were repeated twice – in spring and in summer in different years.

The use of the continuously recorded pH enables very accurate measurements to be obtained, and also provides information on the daily patterns of pH change in response to grazing management and milking. The recording of pressure changes allows both rumination and general rumen movement to be counted. The use of new DNA technologies in ‘profiling’ the rumen microbe populations allows sensitive assessments of the effect of pH on these populations. This project was the first time these research methods to assess rumen function had been used in free grazing dairy cows.

The results obtained from the monthly recordings across the seasons and years showed that the rumen pH was significantly lower than had previously been accepted for grass based ruminants. For example, in some experimental periods the cows spent about 80% of their time under 6.0, 20% under 5.5 and even 10% under 5.0. It was common for cows to have pH values below 5.5 recorded each day. The pH values also showed great changes – for example from 5.0 to 6.5 – several times across the day, and these changes were closely associated with grazing and milking times.

However, despite these pH values the acid and ammonia concentrations were not unusual, and the DNA profiling showed that the rumen microbe populations were stable both across the experimental periods and also across the seasons. Rumination patterns were also unremarkable, and appeared broadly consistent with widely accepted rumination frequencies. There was no evidence to suggest these ruminants were dysfunctional or in any trouble – instead, they appeared to be very well adapted to the conditions. The cows showed no sign of any disease or production impediment as a result of these pH values, and were above average for the herd in production and reproduction figures.

Barley straw in the diet did not reduce the rumen pH, and contrary to the popular view, reduced the rumination count in the cows it was fed to. Overall rumen movement was lower in straw fed cows.

These results suggest that these high production, grass based ruminants operating in highly managed South Island farms have unusual features that have not been adequately researched to date. The results suggest the pH thresholds that define SARA in grain fed diets overseas do not satisfactorily apply to the observed rumen function in apparently healthy, high production cows on grass. The results suggest that 'normal' function in such ruminants has a more rapidly changing pH that falls to lower values for longer periods of the day than has been assumed, without the negative impact described for similar pH values in research on grain based diets. Taken together, these results suggest rumen function in these high production, grass based cows is atypical and have important differences from traditional descriptions of grass based rumen function.

These results are also consistent with other pasture based research here and in Australia looking at the effect of straw on rumen function or production in that they do not show any apparent advantage in the use of supplementary straw in pasture diets. In the one large split herd trial in NZ where straw feeding was assessed for an appropriately long period, production was reduced by 3.4% over the spring in the herd fed straw. If that result was achieved in the LUDF herd in 2008 at \$7.90/kg MS for an average MS production of 420 kg/cow/year, it would be an annual loss of \$113 a cow, or about \$75 500 for the 670 cow herd, excluding the costs associated with buying and providing the straw.

### ***Summary and Conclusions***

Much of the research used to describe 'normal' and 'sub-optimal' rumen function in pasture based systems here in NZ, and particularly around appropriate fibre and rumen pH, has been done abroad on diets based around grain. The relative lack of specific 'local' research means there are robust reasons to exercise caution in directly applying these results to contemporary NZ high production systems based on grass. This includes defining SARA pH thresholds and any direct link suggested with laminitis, and how appropriate fibre levels are established in pastures.

The results obtained in the research at LUDF do not support the conclusion that there is widespread rumen dysfunction in these high production cows on quality pastures on a typical South Island farm, and therefore it would seem unlikely that primary rumen function is a direct cause of increased lameness in these systems.

## **References**

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