Ruminal Acidosis – aetiopathogenesis, prevention and treatment

A review for veterinarians and nutritional professionals

by the Reference Advisory Group on Fermentative Acidosis of Ruminants (RAGFAR)

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> *Note:*
> General information and information relevant to dairy cattle is contained in the body of the document. Information specific to beef cattle and sheep is highlighted in coloured panels. A more detailed document on acidosis management in sheep is to be developed.

Abbreviations used in this document	
ADF	acid detergent fibre
cm	centimetres
CP	crude protein
DM	dry matter
DMI	dry matter intake
eNDF	effective NDF
g	grams
kg	kilograms
meq	milliequivalents
ml	millilitres
mm	millimetres
mg	milligrams
NDF	neutral detergent fibre
NSC	non-structural carbohydrates
peNDF	physically effective neutral detergent fibre
PEF	physical effectiveness factors
ppm	parts per million
VFA	volatile fatty acid

Foreword

The AVA is indebted to the Reference Advisory Group on Fermentative Acidosis of Ruminants (RAGFAR), led by Dr Ian Lean, for the production of this valuable monograph. The Group was established by the AVA Therapeutics Advisory Committee to address the challenges presented by this condition.

The document complements the AVA codes and guidelines for the prudent use of antimicrobials (antibiotics) and the AVA strategy and efforts to lead the profession in this work. The Group has addressed the need for prevention as well as treatment of the disease, and taken a multidisciplinary approach involving nutrition and animal husbandry.

The work will contribute to the health and welfare of ruminants and is especially important at times when nutritional fibre resources are compromised by drought. It is also important for modern systems of ruminant production, where prudent husbandry requires recognition of the threat from acidosis. This work further develops the thread of veterinary achievement in rural industries. Veterinarians have worked with producers, agronomists and animal scientists to greatly reduce the impact of disease on health and production.

Dr Diane Sheehan
President, AVA

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Contributors

Chairman

IAN J. LEAN BVSc, PhD (Calif), MACVSc

Ian is a registered Veterinary Specialist, past president of the Australian Association of Cattle Veterinarians (AACV) and Cattle Chapter of the Australian College of Veterinary Scientists. Ian is director of Strategic Bovine Services and Global Dairy Consultancy Company, USA. He is an Adjunct Professor in Veterinary Science with the University of Sydney. He has been appointed to the scientific boards of several international companies and has more than 200 scientific articles. Ian instigated with Bruce Christie the pregnancy diagnosis scheme for the AACV and has mentored about 20 postgraduates. His interests are in improving profitability of ruminant production and in researching interactions between nutrition, health and reproduction in cattle.

Scientific editor

EMERITUS PROFESSOR FRANK ANNISON BSc, PhD (London), DSc (London), AM

Frank Annison completed his PhD in biochemistry at London University and was awarded a DSc from the same institution. He worked in industry in the UK, at the University of New England and the University of Sydney where he was head of the Department of Animal Science. Frank's scientific achievements in nutrition are profound. He was pivotal in providing understandings of the biochemistry of the rumen and contributed greatly to the field of quantitative nutrition of a number of species. He was awarded the Roche International Prize for nutrition and is a Fellow of the Nutrition Society. He was awarded an AM for his contributions to the understandings of nutrition.

ELIZABETH BRAMLEY BSc(Hons), BVMS, PhD (Syd)

Elizabeth graduated from Murdoch University with a Bachelor of Science (research honours) in 1995 and Bachelor of Veterinary Medicine and Surgery in 1997. She commenced work with Finley Veterinary Clinic, a predominantly dairy practice in the Riverina area of NSW in 1998. Between 2001 and 2005, she completed a PhD at the University of Sydney on ruminal acidosis in Southern Australian dairy cattle. The literature review was the basis for this document. Elizabeth is currently working in mixed veterinary practice and raises her own cattle.

GLENN BROWNING BVSc (Hons), DipVetClinStud (Syd), PhD (Melb)

Glenn Browning is a Professor in Veterinary Microbiology at The University of Melbourne, with interests in the pathogenesis, diagnosis, treatment and control of bacterial and viral diseases of a variety of domestic animals. He graduated with a BVSc from The University of Sydney in 1983, was an intern at the Rural Veterinary Centre at The University of Sydney in 1984 and completed a PhD at The University of Melbourne in 1988. After three years as a Veterinary Research Officer at the Moredun Research Institute, Scotland, he joined the staff at the Faculty of Veterinary Science, The University of Melbourne, in 1991.

Introduction

Ruminal acidosis is increasingly recognised as a significant disorder of ruminants. This condition increases the morbidity and mortality of stock, markedly reduces weight gains in the feedlot, complicates drought feeding strategies for sheep and cattle, and is increasingly recognised in pastoral and confined dairying. It may be the most significant health disorder of ruminants fed on high-quality pastures and grain.

The aims of this review are to evaluate the current knowledge of the patho-physiology of acidosis, and provide practical information on the diagnosis, prevention and treatment of the condition in beef and dairy cattle, and sheep.

Aspects of acidosis of relevance to the feed industry and veterinary practice are also examined, including the prudent use of antibiotics, both in the treatment and prevention of sequelae to the disorder.

Ruminal acidosis in cattle

Definition
pH is a scale from 0-14 that defines whether a material is acidic, neutral or basic (Acidic pH is < 7, basic pH is >7). pH is a logarithmic, not a linear scale.

Definition
Carbohydrates are the simple sugars, starches and structural sugars in the cell wall of plants which are fermented by the rumen microbes at different rates and to different extents.

Definition and clinical presentation of ruminal acidosis

Acidosis is a pathological condition associated with the accumulation of acid or depletion of alkaline reserves in blood and body tissues, and characterised by increased hydrogen ion concentrations (Blood and Studdert 1988). Ruminal acidosis refers to a series of conditions that reflect a decrease in pH in the rumen of cattle. Rumen lactic acidosis (grain overload, grain poisoning, acute indigestion) develops in sheep and cattle that have ingested large amounts of unaccustomed feeds rich in ruminally fermentable carbohydrates (Crichlow and Chaplin 1985; Nocek 1997).

The resulting production of large quantities of volatile fatty acids (VFA) and lactic acid decreases rumen pH to non-physiological levels, simultaneously weakening the buffering capacity of the rumen, and reduces the efficiency of rumen flora and fermentation. Lactic acidosis can cause ruminitis, metabolic acidosis, lameness, hepatic abscessation, pneumonia and death (Lean et al. 2001). Acidosis can be divided into two categories – clinical and sub-clinical.

Clinical acidosis

Prevalence

Clinical acidosis presents as a mild to severe form of the disease, depending on the type and amount of feed ingested. Bramley (2006 unpublished) found a herd prevalence of clinical acidosis of at least 3% in a survey of 100 Australian dairy herds.

Clinical signs

Cattle with mild clinical acidosis exhibit anorexia, decreased milk production and scouring (Underwood 1992). The severe form of the disease may progress to include metabolic acidosis, depression, dehydration, toxaemia and 'downer cow' syndrome (Bolton and Pass 1988). Peracute acidosis may result in recumbancy, coma and death in eight to 10 hours (Underwood 1992). Clinical signs in the acute form develop within eight hours and precedes the onset of metabolic clinical acidosis that peaks in 36 hours (Underwood 1992). Clinical acidosis generally affects one or more cattle in a herd and is often precipitated by sudden dietary changes.

Sequelae

Problems occurring as a sequel to acute ruminal acidosis include hypocalcaemia resulting from calcium malabsorption, laminitis from the release of histamine and endotoxins into the circulation, polioencephalomalacia from an induced thiamine deficiency, ruminitis and liver abscessation (Bolton and Pass 1988; Nagaraga and Chengappa 1998). Bacteria implicated in liver abscessation are *Fusobacterium necrophorum* and *Archanobacterium spp.* These reach the liver through the portal circulation via damaged ruminal epithelium (Bolton and Pass 1988).

Epistaxis cases – extensive bleeding from mouth and nose.

Very liquid faeces – poorly formed.

Acidotic cows showing poor rumen fill, arched backs (associated with lameness) and loose faeces.

Sub-clinical acidosis

Prevalence

Sub-clinical ruminal acidosis is usually of greater economic importance than clinical disease and can often affect a significant proportion of the herd. Bramley et al. (2006 unpublished) found a 10% prevalence in sub-clinical acidosis in dairy cows in NSW and Victoria.

Clinical signs

Signs often associated with sub-clinical acidosis include a reduction in milk fat content, feed conversion efficiency, feed intake and decreased digestion of fibre (Lean, Wade et al. 2000), laminitis causing lameness (Nocek 1997; Owens et al. 1998), liver abscessation (Owens et al. 1998), scouring (Nocek 1997), and a higher incidence of left and right displacements of the abomasum (Shaver 1997).

Sequelae

Sub-clinical acidosis often goes unrecognised and undiagnosed until significant herd involvement and obvious clinical signs are evident. At this stage, large financial losses and long-term health issues, such as a high prevalence of herd lameness, may be inevitable.

Diagnostic tests

The definition of sub-clinical acidosis is controversial, with some authors suggesting that a pH of 5.5 detected by rumenocentesis be used as a cutpoint for detecting the disorder (Garrett et al. 1999). However, *in vitro* fibre digestibility is reduced when pH drops below 6.2 (Grant and Mertens 1992; Grant 1994; Calsamiglia et al. 1999). Bramley et al. (2006 unpublished) used an approach of categorising cows on VFA, lactic acid and ammonia concentrations, and pH. One group of cows identified this way had signs that were consistent with acidosis, being characterised by high concentrations of valerate and propionate and low concentrations of ammonia. While the pH was low and the lactic acid concentrations high for this group, these were the least discriminatory variables. Using pH as the sole measure to detect acidotic cows was neither highly sensitive nor specific. Improved diagnostic measures to confirm diagnoses made on clinical signs need to be developed.

Conclusions

At present, a diagnosis of sub-clinical acidosis should be tentatively made when more than 50% of ruminocentesis samples taken 2-4 hours after grain feeding or 4 hours after pasture feeding fall below 6.2. There is a high likelihood of ruminal acidosis in a herd when more than 30% of the sampled cows have a ruminal pH of 5.5 or less (Olson 1997; Garrett et al. 1999). A ruminal pH of 5.6 to 5.8 suggests a marginal or developing problem of ruminal acidosis, while a pH of greater than 5.9 is considered 'normal' (Olson 1997). It is likely that more sensitive diagnostic methods will be identified in the future, based on other indicators.

pH test strips.

Ruminal acidosis in sheep

Clinical signs of clinical acidosis in sheep are similar to those in cattle, although the grinding of teeth and muscle fasciculation have been reported in clinical cases. Sheep differ from cattle in rumen function; feed tends to pass quicker through the rumen than with cattle and sheep tend to have a higher incidence of intestinal disturbances compared to cattle, e.g. enterotoxaemia (Pulina 2004).

When grazing, sheep tend to select the more digestible parts of the plants than cattle. Sheep also have less powerful jaws and need a longer time chewing their cud and they grind their food particles finer than cattle (van Soest 1994). Sheep spend a shorter time eating than cattle, but a longer time ruminating. On pasture sheep spend half the time eating compared to cattle, but almost four times longer chewing. It is unclear whether the pathophysiology of acidosis differs substantially between sheep and cattle, but it clinically appears to be similar.

The management of sheep differs from other ruminant species. Sheep tend to be extensively managed in large mobs of between 200 and 5,000. Therefore, individual attention is less likely and signs of ill health are difficult to detect and monitor. Unlike dairy cattle, there is no regular monitoring of performance and changes in growth rates may not be readily detected, unless they are very obvious.

While estimates of sheep fed in lots in Australia are not readily available, at least four million per annum are fed this way in preparation for live export. Feedlots vary greatly in overall size and usually hold between 200 and 400 sheep in each pen. Live export diets and those used in conditioning before export are higher in fibre than those in opportunity lots where lambs destined for slaughter are fed to achieve high growth rates. Ewe lambs are also fed on diets containing significant amounts of highly soluble carbohydrate to achieve adequate body weight for mating at seven months of age or in times of drought.

There can be significant risk of acidosis for sheep in the cropping regions, where sheep are often given access to grain stubble after harvest. These paddocks may have large amounts of grain available on the ground, either remaining after harvest or spilt during the handling of the grain. These sheep usually do not go through any adaptation stage. A similar situation can occur when sheep are introduced on to crops, particularly brassicas such as rapes, kales or turnips, that are highly digestible, and when sheep selectively graze the most digestible material.

Feeding systems used to supply sheep vary considerably. In drought, sheep may be fed a grain trail in the paddock at intervals of daily through to twice each week. They may be fed grain continuously in bin feeders, be confined to a feedlot with a feed trough supplying a formulated ration each day, or be fed grain supplied in feed bins with access to hay or straw. In paddocks sheep may be provided access to one bin feeder for approximately every 800 sheep.

Early introduction to grains or concentrates establishes an awareness of grains and encourages more consistent uptake of grain by the mob of sheep. The number of sheep managed in feedlots is increasing rapidly, however, many feedlotters are purchasing store sheep that may not have been imprinted and are being introduced to a feeding regime to which they are not accustomed. That increases the risk of differential feed intake and grain engorgement by some sheep. Where feed bins are used, most have an adjustable outlet to regulate the amount of grain ingested. Sheep lick the feed from under the adjustable bar used to limit intake. If the adjustment is not set correctly, some sheep have not been conditioned or the setting becomes altered, then larger intakes of grain can occur, leading to increased incidence of acidosis.

A common cause of severe clinical acidosis outbreaks is when sheep have had accidental access to large amounts of grain. These outbreaks result when large amounts of grain are spilled in harvested paddocks or during grazing in paddocks that contain grain silos. Access results either from spilled grain or if a weak hatch opens.

Proposed aetiology of ruminal acidosis in cattle

The aetiology (cause) effects and prevention of ruminal acidosis have been extensively researched and reviewed (Hungate et al. 1952; Dirksen 1970; Huber 1976; Slyter 1976; Underwood 1992; Nocek 1997; Owens et al. 1998). Both *in vitro* and *in vivo* trials have been conducted, however much of this research has concentrated on feeding large amounts of starch-based concentrate as the cause of acidosis (Crichlow and Chaplin 1985). These studies provide excellent information on acidosis in feedlot cattle and sheep on diets very high in starch. Ruminal pH in experimental cases was often less than 5 and caused severe acute clinical disease (Nocek 1997). This pH range appears to be lower than that of cattle solely fed on pasture where the mean daily pH has been reported between 5.6 and 6.4 (Van Vuuren et al. 1992; Stockdale 1994; Carruthers et al. 1996; O'Mara et al. 1997; Kolver et al. 1998), with optimal ruminal pH being above 5.8 (De Veth and Kolver, 1999) to 6.0 (Van Soest 1994) for fibre digestion.

Table 1: Rumen microbial populations					
Bacteria and archaea	Primary substrate	Optimum rumen pH	Primary requirement	Main fermentation products	Microbial doubling times
Bacteria	About 630 different bacteria (50% of microbial mass)				
	Fibre and pectin	6.3 to 6.8	NH_3, isoacids	Acetate	8-10 h
C. aminophilum	Protein	6 to 7	Protein, peptides, NH_3	NH_3, Isoacids	4-8 h
Allisonella histaminiformans	Histidine	4.5 to 6.5	Histidine, peptides from silage	Histamine	Rapid
S. bovis	Starch and sugars	5.5 to 6.5	Peptides, AA, NH_3	Propionate, lactate	15-30 m
Secondary – *M. elsdenii, Methanogens*	Lactic, H_2	6 to 6.8	Peptides, AA, malic	Propionate, CH_4	2-4 h
Protozoa	About 30 different protozoa (40-45% microbial mass)				
	Starch, sugars	6.3 to 7.0	Peptides, AA, bacteria	Propionate, H_2	15-24 h
Fungi	About 14-15 types of fungi (3-8% microbial mass)				
	Fibre	6 to 7	NH_3, AA, sugars	lactate, acetate, H_2	15-24 h
Bacterial viruses	(5-7 types and .0000001% TMM) Yeasts (0.1-0.2% TMM)				

There has been little research on the aetiology of acidosis induced primarily by pasture feeding (Wales et al. 2001), but more on the feeding of starch-rich concentrates to cows on pasture (Opatpatanakit et al. 1994; Clayton et al. 1999; Lean, Wade et al. 2001; Wales et al. 2001). Wales et al. (2001) found that cows fed solely on pasture or pasture supplemented with grain had a rumen pH lower than 5.5 during the day. Traditionally, in Australia and parts of New Zealand, the supply of starch-rich concentrates is associated with 'slug feeding' practices in dairy herds, where large quantities of concentrates are consumed without adequate buffering by cows fed in the bail twice daily at or around milking. Increasingly, beef cattle are being fed supplements while they are on pasture.

Lean et al. (2000) proposed that the following mechanisms could lower ruminal pH:

- access to preformed acids in feeds, such as some silages;
- a failure to produce buffering with endogenously derived buffers such as salivary bicarbonate;
- production of lactic acid in the rumen; and
- production of large amounts of weak volatile fatty acids, acetic acid, butyric acid and propionic acid.

Pastures low in Neutral Detergent Fibre (NDF) and high in non-structural carbohydrates (NSC), such as lucerne, clover-dominant pastures and very lush temperate and subtropical grasses, and supplementation of pastures with silages and grain, expose cattle that are predominantly pasture-fed to the risk of acidosis.

Definition

Concentrates refers to all grain-based feeds fed as supplements, whether they be whole or processed grains, grain mixes or grain-based pelletised feeds.

Definition

Neutral Detergent Fibre (NDF) is a chemical laboratory estimate of the plant cell wall.

The aetiology of ruminal acidosis on carbohydrate-rich diets

Figure 1 indicates the proposed sequence of events involved in the aetiology of acute ruminal acidosis induced by the sudden introduction of high levels of starch.

Increase in fermentable CHO

Polymer → cellubiose
maltose
xylobiose
glucuronides

Sugar → xylose
hexoses → organic acids
organic acids

VFA
Methane
CO_2

ADP ⇌ ATP

1 microbe
CH_2O
NH^3
AA
→ 2 microbes

Increase in growth rate and increase in VFA (all bacteria)

Decrease in pH

Increase S. bovis growth rate

Increase in lactic acid

Decrease in S. bovis
Increase in lactobacilli

pH<5.0

Decrease in growth rate many bacteria

Decrease in pH

Stasis in fermentation

D & L lactate absorbed

Metabolic acidosis

Figure 1: Proposed sequence of events associated with the induction of acute ruminal acidosis. CHO = carbohydrate **(adapted from Baldwin and Allison 1983; Nocek 1997).**

Changes in rumen bacteria

Initially, this metabolic insult increases the growth rates of all bacteria in the rumen, resulting in an increase in total volatile fatty acid production and a decrease in ruminal pH. It is likely that the provision of increased substrates for microbial production, e.g. ammonia and peptides, will favour bacterial growth rather than production of VFA. When large amounts of starch are added to the diet, the growth of *Streptococcus bovis* is no longer restricted by a lack of this energy source and this population grows faster than other species of rumen bacteria (Russell and Hino 1985). *S. bovis* produces lactic acid, an acid 10 times stronger than acetic, propionic or butyric acid, the accumulation of which eventually exceeds the buffering capacity of rumen fluid. Glucose produced from the breakdown of starch and other carbohydrates is converted to fructose 1,6-diphosphate (See figure 2). Russell and Hino (1985) found that fructose 1,6-diphosphate had a positive feedback on the conversion of pyruvate to lactate by activating lactate dehydrogenase. Fructose 1,6-diphosphate is also converted to triose phosphate in increasing concentrations. Triose phosphate acts to inhibit pyruvate formate lyase. The net effect of these changes is a switch from predominantly acetate and formate production to lactate production (Russell and Hino 1985).

Definition

Streptococcus bovis *is a gram positive, non-mobile coccoid bacterium normally present in the rumen microbial population. The major fermentation product of* S. bovis *is lactic acid.*

Figure 2: Fermentation of carbohydrates in the rumen **(adapted from Hungate 1966;** Baldwin and Allison 1983; Lean **1987; van Houtert 1993).**

Changes to rumen organ motility

The increase in VFA concentrations may also initially decrease reticulo-ruminal motility by acting on receptors in the rumen wall. These receptors, in sheep, are activated when non-dissociated VFA concentrations exceed 3.0 mM (Crichlow and Chaplin 1985). Crichlow and Chaplin (1985) found that the decrease in ruminal motility was independent of a decrease in rumen or systemic pH, and occurred when venous blood concentrations were normal. A decrease in motility results in a decrease in rumination and less production of saliva. Saliva contains high concentrations of bicarbonate ions and is an important buffering mechanism for the rumen. Saliva is produced through stimulation of the rumen and mastication.

Changes in rumen pH and lactic acid absorption

A decrease in bicarbonate and increase in lactic acid concentrations in the rumen further decreases ruminal pH. When ruminal pH is maintained above 5.5, an equilibrium exists between producers and utilisers of lactic acid, such that lactic acid does not accumulate in the rumen (Nocek 1997). When pH is less than 5.5, no cellulolytic and relatively few saccharolytic bacteria, including *P. ruminicola*, a significant producer of VFAs, survive. In contrast, *S. bovis* multiplies until ruminal pH is less than 5.0, a pH that allows an increase in *Lactobacillus* growth. Both of these bacterial species produce D and L-lactic acid (Figure 3). D-lactate and L-lactate are absorbed across the rumen wall and depress blood pH. As L-lactate is metabolised more rapidly than D-lactate, the metabolic acidosis is due in large part to the accumulation of the latter (Bolton and Pass 1988). The lowered pH creates a ruminal environment hostile to protozoa and fungi and populations of both these fall precipitously. Chemical damage of the surface epithelium of the rumen mucosa occurs and results in the adherence of debris and penetration by particulate matter from the rumen. Bacterial and mycotic organisms begin to invade the rumen wall causing ruminitis. Rumen papillae are damaged and can slough from the rumen wall (Ahrens 1967; Lee et al. 1982). Absorption patterns change and endotoxins and histamine are released in the acute stages of the disease process (Mullenax et al. 1966).
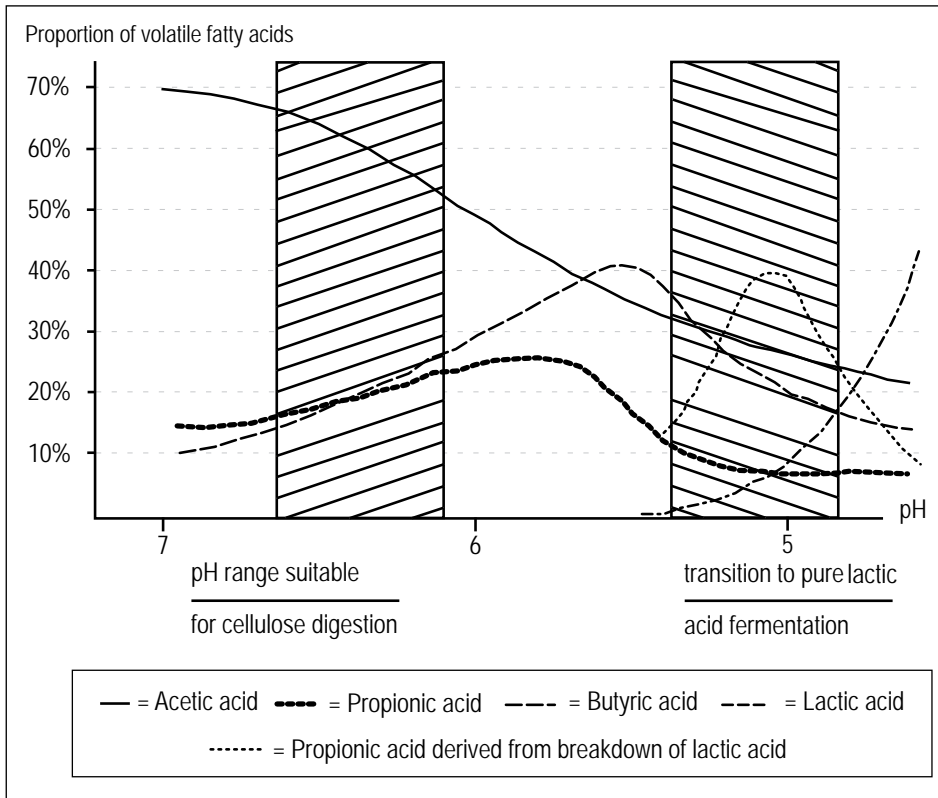
Figure 3: Proportions of acids produced in the rumen when pH **falls from 7.0 (after Kaufmann** and Ruhr 1967, reprinted from: Rosenberger, Clinical Examination **of Cattle, 1979, Paul Parey Scientific Publishers, Berlin and Hamburg).**

Acidosis and diarrhoea

As lactate passes from the abomasum into the intestinal tract, this creates an osmotic gradient. The resultant increase in fluid in the lumen is responsible for the profuse diarrhoea and subsequent dehydration seen in clinical acidosis.

pH and sub-clinical acidosis

In sub-clinical acidosis, the pH will often fluctuate in the rumen during the 24-hour period, with a nadir (lowest point reached) recorded two to four hours after feeding concentrate in the bail (Harrison et al. 1989). Krajcarski-Hunt et al. (2002) found that induction of sub-clinical ruminal acidosis significantly reduced the mean daily rumen pH from 6.36 to 5.72, increased daily time below pH 6.0 from 155 to 938 minutes per day and increased the time where the rumen pH was <5.6 from 15 to 594 minutes per day. The risk of acidosis is increased when cattle are fed forages high in non-structural carbohydrates (sugars and starches) with low effective fibre content, such as clovers and young lucerne (alfalfa) and possibly ryegrass that may not stimulate adequate rumination and salivation, especially when fed in combination with concentrate feeding.

Cow scouring.

Rumen pH and fermentation

Importance of pH in rumen function

The optimal pH for cellulolysis, proteolysis and deamination is between 6 and 7 (Lewis and Emery 1962; Mould et al. 1983). Ruminal cellulolysis is totally inhibited at a pH less than 6.0 (Mould et al. 1983) and dry matter digestibility decreases with decreasing pH (Tilley et al. 1964). Kolver and de Veth (2002) reviewed 23 studies in dairy cattle that examined the rumen pH of cows on pasture-based diets. A lower ruminal pH was correlated with higher microbial nitrogen flow from the rumen, higher total and individual volatile fatty acid concentrations, milk and milk component yields and dry matter intake, but lower concentrations of fat and protein in milk and a lower milk fat to protein ratio.

There is increased efficiency of production with increased concentrations of carbohydrates in the diet, but cattle may partition more precursors towards body fat than milk fat.

pH and diet type

Kolver and de Veth (2002b) also reported that lactating cows fed diets containing a mean of 80% pasture had a mean daily ruminal pH of 6.2 (range of 5.6 to 6.7). These results were based on cows grazing a number of pasture species and direct extrapolation to Australasian conditions, where many herds graze predominantly ryegrass and clover pastures may not be appropriate. Few studies have been conducted on this pasture combination, but one study reported a mean daily rumen pH of 5.9 when cows ate an estimated 15.6 kg DM of perennial ryegrass (*Lolium perenne* L.)- white clover (*Trifolium repens* L.) per day (Wales et al. 2001).

Rumen pH fluctuates throughout the day depending on diet, time of feeding of concentrates and the supplementation of fibre sources such as hay following milking in many Australasian systems. Variation due to biphasic feeding patterns induced by twice daily feeding at or after milking may produce a substantial, but relatively short-lived depression of ruminal pH. Daily mean ruminal pH will not adequately represent the highly variable characteristics of ruminal pH.

The physical form of the diet, e.g. a reduction in the forage particle size or the processing of grain, decreases ruminal pH (Krause et al. 2002b). This response reflects less saliva production and more rapid breakdown of carbohydrates in the rumen. The amount of rumen fermentable carbohydrate present in the rumen or the digestibility of starch in the rumen can be negatively correlated to rumen pH (Yang et al. 2001; Krause et al. 2002a). Feeds high in pre-formed acids, such as some silages, will also reduce rumen pH (Lean 1987).

Rumen pH starts to decline immediately after feeding concentrates or silage. Concentrates cause a more rapid decline in rumen pH than silages. The nadir of rumen pH when feeding concentrates separately from forages is between two and four hours after feeding. Krause et al. (2002a) found that the nadir of rumen pH in cows given coarse silage was five hours after feeding and for cows fed finely chopped silage there was a less-pronounced nadir nine hours after feeding. Therefore, to provide an overview of herd status, samples of rumen fluid should be collected from a minimum of six to 12 cows (Olson 1997; Garrett et al. 1999). When concentrates are fed separately from forages, samples should be taken two to four hours following the feeding of concentrates. For grass only or total mixed ration herds, samples should be collected four to eight hours after feeding. A portable pH meter capable of measuring pH from a very small volume of rumen fluid yields very similar results to a standard meter with probe (Garrett et al. 1999). Consequently, portable pH meters provide a valuable tool for practitioners wishing to measure rumen pH.

The physiological buffering capacity of the rumen

Buffering capacity is defined as the number of moles of H+ per litre of test system required to cause a given change in pH (Counotte et al. 1979). The physiological buffering capacity of the rumen allows rumen pH to be maintained between 6 and 7 on most diets in most conditions. However, when highly fermentable carbohydrates are fed to stock, particularly without prior exposure, rapid fermentation occurs in the rumen, resulting in a decrease in rumen pH from between 6 and 7 to 5.5 or lower. The amount of decrease in pH after an increase in fermentation rate depends on the buffering capacity of the rumen (Counotte et al. 1979). If there is a continual increase in the concentrates fed, the carbohydrate challenge may result in the buffering capacity being depleted with resultant acidosis. Typical beef feedlot finisher diets provide a substantial challenge to buffering capacity. It is now common practice to use dietary buffers and modifiers to work in conjunction with the physiological buffering capacity of the rumen.

The best substances for buffering in a given pH range are those whose pKa values are such that pKa ± 1 lies within this range. The primary rumen buffers are bicarbonate from saliva production and volatile fatty acids present in the rumen (Counotte et al. 1979). Secondary buffers include phosphorus buffers, sometimes classified as neutralising agents (Counotte et al. 1979) and carbonate buffers, also sourced from saliva (Lean 1987). The pKa of bicarbonate and dihydrogen phosphate are within the range 6 to 7 (Czerkawski 1986).

In the normal pH range of the rumen, the most important buffers are those originating from saliva, with the weak volatile fatty acids more important in lower pH ranges. Saliva in cattle is produced from a number of glands; parotid, mandibular, sublingual, labial, ventral, medial and dorsal buccal and pharyngeal (Kay 1960). The parotid, buccal and pharyngeal salivary glands are responsible for the production of most HCO_3 and HPO_4. The parotid gland contributes 40% to 50% of total 70 and 180 litres of saliva produced per day. Concentrations of HCO_3 and HPO_4 are approximately 125 and 25 meq/L, respectively (Lean 1987).

Buffering capacity is normally influenced by factors altering the amount or quality of saliva production, the concentration of ruminal acids and CO_2, and the rate of absorption or passage of digesta through the rumen (Lean 1987). The average pH of saliva is about 8.0 (Counotte et al. 1979). At this pH, 86% of the phosphate is in the HPO_4^{2-} form. Upon entering the rumen, the following reaction occurs: $HPO_4^{2-} + H_3O^+ \leftrightarrow H_2PO_4^- + H_2O$, until 10% is in the form of HPO_4^{2-} and 90% as $H_2PO_4^-$. This reaction raises the rumen pH, but reduces the buffering capacity of the phosphate system (Counotte et al. 1979). Bicarbonate acts similarly, but is more important when the rumen pH is 6.25 or lower because the pH value of the equilibrium reaction ($HCO_3^- + H_3O^+ \leftrightarrow H_2CO_3 + H_2O \leftrightarrow CO_2 + 2H_2O$) is 6.25 (Counotte et al. 1979).

When the rumen pH is less than 6.0, the decrease in bicarbonate concentration and therefore buffer capacity poses a potential threat to the animal, especially when re-feeding concentrates may occur before buffering capacity recovers. Below pH 5.5, lactate-fermenting bacteria are unable to grow, partly because of their need for bicarbonate, which is not present in these conditions, allowing lactic acid to build up in the rumen, further depressing pH.

Techniques for rumen fluid collection

Three techniques for rumen fluid collection may be used to determine ruminal pH.
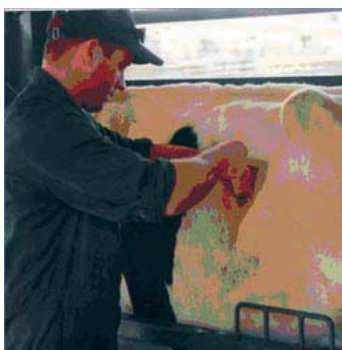
Rumen cannula

Collection of ruminal contents from cattle with an indwelling ruminal cannula remains the preferred method for determining ruminal pH. However, practical limitations, including the need to regularly clean and monitor rumen cannula sites, limits their use in commercial situations. Some large dairy herds have rumen cannulated cows to provide a ready monitor of rumen function and provide fluid for transfaunation, although this is not common practice in Australia.

Rumenocentesis

The collection of ruminal fluid using percutaneous needle aspiration is a more practical method for determining ruminal pH. Rumen fluid is collected using a 14-gauge 1½ inch (about 4 cm) disposable needle attached to a 30 mL eccentric syringe. A 2 cm by 2 cm area is shaved on the left paralumbar fossa approximately one hand length ventral to the lumbar spinal transverse processes and one hand width caudal to the last rib, avoiding the major muscle masses. This area is surgically prepared, local anaesthetic injected, and the sampling needle is inserted firmly through into the rumen. A minimum of 4 mL should normally be obtained from most animals using this procedure. This method may not be completely benign in healthy cattle, because cattle had reduced milk yields for 24-48 hours post-sampling (Opatpatanakit et al., unpublished) in one trial. A trial involving 800 cows in Australia reported only one incident involving a mild abscess forming around the surgical site (Bramley 2004). Garrett et al. (1999) found that the pH of rumen fluid collected via rumenocentesis was 0.28 units lower than that of fluid collected through a ruminal cannula. Filtration or aspiration of the rumen fluid had no effect on pH.

The rumenocentesis method for **collecting rumen fluid.**

Figure 4: The relationship between stomach tube and rumenocentesis **pH (Bramley, 2006, unpublished).**
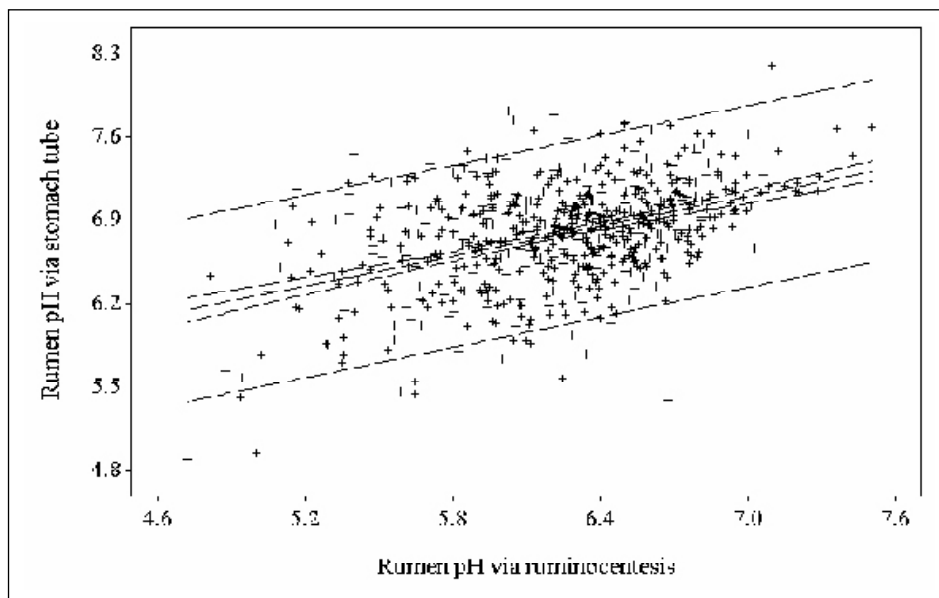
Rumen stomach tube

A stomach tube suited to collecting ruminal fluid can be constructed using a 3½ m long, 19 mm diameter plastic reinforced garden hose attached to an equine stomach pump. The other end should be riveted to a 10 cm to 15 cm length of copper pipe with multiple holes drilled along all sides. This reduces the risk of blockages with ingesta.

Samples of ruminal fluid taken by stomach tube have a higher pH than those obtained through a rumen cannula (Erdman 1988), either as a result of salivary contamination or sampling in the fibre mat of the rumen (Figure 4). Therefore, ruminal pH samples collected by stomach tube should be interpreted with caution. This method will be more specific, but less sensitive, for the detection of acidosis. The rumen fluid collected from the stomach tube should be examined for evidence of saliva contamination before measuring pH and samples discarded if contaminated.

Duffield et al. (2000) compared samples obtained from the cranial ventral rumen, with rumenocentesis and stomach tube samples. Rumenocentesis samples were sensitive for detecting pH of the cranial ventral rumen, while stomach tube samples were poorly sensitive. Both tests had similar specificities. The most accurate field test for ruminal pH was rumenocentesis.

The stomach tube method for **collecting rumen fluid.**

Indirect indicators of ruminal acidosis

Chewing activity

Chewing activity can indicate the presence or lack of adequate fibre in the diet and, therefore, is also an indication of sub-clinical acidosis in a herd. Practical guidelines for assessing the proportion of cows chewing within a herd have been proposed (Lesch and Sawyer 1981). When cattle are not actively grazing, walking, drinking or sleeping, 50% or more of the herd should be ruminating and/or actively chewing. Where fewer than 50% of cattle are identified with chewing activity, the potential for sub-optimal ruminal pH and rumen function should be considered.

Chewing activity, when assessed together with other animal and feed measures, can be a useful, non-invasive measure of rumen function in a dairy herd.

Rule of thumb

50% of cows at rest in the paddock should be actively chewing at any one time.

Faecal changes

The faeces of stock can provide indirect evidence of clinical and sub-clinical acidosis and lack of fibre in the diet. There are other factors such as parasites and diseases that cause scouring. However, herds with a large percentage of dung heaps that are soft and un-formed, a high prevalence of cows scouring or faecal smearing around the perineum can indicate acidosis. This evidence needs to be supported by other indicators, such as feed evaluation and analysis, milk fat concentrations, the prevalence of lameness, rumen sampling and reduced chewing activity for confidence in a diagnosis.

Typically, the faeces of acidotic stock are more liquid, contain undigested fibre and grain, are often lighter in colour and may contain gas bubbles. The smell of the faeces may be bitter-sweet, rather than the typical herbaceous character found from stock fed on grass. Faecal moisture content is increased as a result of a lactate-based osmotic overloading of the large intestine, the lactate coming from the rumen (Bolton and Pass 1988).

Lactate also causes sequestration of large quantities of water in the rumen, inducing dehydration. Increased proportions of undigested feed resulting from reduced microbial cellulolytic activity at a lower ruminal pH also contribute to diarrhoea (Bolton and Pass 1988). Notwithstanding this, faecal pH is poorly correlated with ruminal pH due to fermentation and buffering in the hind gut in dairy cows (Clayton et al. 1999) and should be interpreted with caution.

Faeces can be scored on a 1 to 5 scale, ranging from scouring with no form and a large proportion of water from cows on a diet of minimal fibre (score 1) to dry with excessive amounts of fibre from cows on a predominantly hay diet (score 5).

Score 1: Faeces extremely liquid, bubbling and containing grain.

Score 2: Runny – stools do not form up – little evidence of fibre.

Score 3: Porridge-like consistency, forms 'chocolate cake' stools. Well-digested fibre is evident.

Score 4: Excessive fibre evident throughout stool, forms piles more than 50 mm high.

Feed characteristics

Assessment of the chemical and physical characteristics of a diet is an important diagnostic tool when assessing nutritional problems in the herd. The examination of chemical properties such as percentage dry matter, digestibility, energy, non-structural carbohydrate (NSC) content, crude protein (CP), neutral detergent fibre (NDF) and acid detergent fibre (ADF) will provide an overview of feed quality. This information, when combined with an evaluation of the physical properties of quality, including particle size and stem to leaf ratios of forages, will indicate the possibility of sub-optimal rumen function and acidosis. Risk of ruminal acidosis will be greater when pasture is lush, leafy with a high leaf to stem ratio and growing rapidly (Westwood and Lean 2001). Pasture quality can be increased by use of fertiliser, especially nitrogenous fertilisers that increase growth and nitrogen concentrations. Grazing interval can also influence pasture quality. Ryegrass grazed at the two-leaf stage will have a lower NDF and higher crude protein than ryegrass grazed at the three-leaf stage. The harder cows graze into pasture the higher the overall NDF and the lower the protein as cows eat more stem and less leaf. Lax grazing increases the risk of subacute acidosis as cows select the best-quality pasture, that is pasture lower in NDF and with higher CP.

The low pH of silages (pH less than 4.0) resulting from pre-formed lactic acid can contribute to lower ruminal pH and, therefore, acidosis. This is particularly relevant when considering maize and whole crop cereal silages. The chop length of forages used for silage or hay will further influence ruminal pH, with short chop silage and hay reducing chewing and rumination time and, therefore, saliva production (Sudweeks et al. 1975; Grant et al. 1990a; Grant et al. 1990b; de Boever et al. 1993). Particle size is negatively correlated with chewing activity per kilogram of DM (Allen 1997; Mertens 1997) and positively with ruminal pH (Allen 1997). For practical purposes, greater than 25% of the forage component of the diet should contain forage with a length comparible with the muzzle width of the animal grazing.

Particle size and processing also applies to the concentrates being fed. Starchy cereal grains, particularly wheat, can be rapidly rumen fermentable, produce lactic acid and increase the risk of acidosis (Opatpatanakit et al. 1994). In terms of the risk of acidosis, the ranking is wheat>triticale>barley>oats>sorghum>maize. This ranking reflects the amount, rumen degradability and cellular structure of starch in each grain. More finely ground grains also increase the risk of acidosis, as does heat-treated grain i.e. pelleted grains that have been both finely ground and heated. Herds that had a high prevalence of acidotic dairy cows also had significantly higher ratios of NSC to NDF in the overall diet (Bramley 2006, unpublished).

Low milk fat concentration

Ruminal pH is positively associated with milk fat concentration (Kolver and de Veth 2002). A milk fat:milk protein ratio of <1.15:1 may indicate a risk of acidosis. Therefore, monitoring bulk vat milk fat percentage may be a useful indicator of acidosis in dairy herds. The relationship between ruminal pH and milk fat concentration is not absolute and is confounded by stage of lactation, dietary fat content and body fat mobilisation (Westwood and Lean 2001). A reduction in the concentration of milk fat should be considered to indicate the possibility of low ruminal pH and initiate investigations using other means of evaluating rumen function.

Heifers, generally being of a smaller body size than cows, are at greater risk of acidosis, especially if 'slug-fed' concentrate in the dairy in the same amounts as mature cows. This method of feeding may decrease the forage:concentrate ratio of heifers because they may also eat less pasture than older cows. Check the milk fat and protein of heifers separately if herd test data is available.

Laminitis/lameness

Lameness is common in the Australian dairy industry, with a reported incidence of 7.5% during lactation (Harris et al. 1988). Many factors have been implicated. Environmental factors including mud and moisture, lane and yard design, and temperature may combine with nutrition, animal management and individual animal characteristics to cause lameness (Chesterton et al. 1989). In most cases, a high prevalence of foot lameness will reflect the combined effects of several predisposing factors.

Laminitis (*pododermatitis aseptica diffusa*) is regarded internationally as an important predisposing factor to lesions of the claw, yet the role of laminitis in the pathogenesis of lameness in the Australian dairy industry is largely unquantified.

Acidosis has been implicated as a common cause of laminitis in dairy herds. Lesions associated with laminitis include solar haemorrhage and abscessation (Dewes 1978; Tranter and Morris 1991; Bergsten 2000); bruising of the solar corium (Dewes 1978; Harris et al. 1988); under-run sole (Tranter and Morris 1991; Bergsten 2000); and white line lesions . Claw lesions consistent with laminitic changes have been described for Australian dairy cattle (Jubb and Malmo 1991; Vermunt 1992). Many studies have identified nutritional factors that increase the risk of laminitis, including feeding diets high in rapidly fermentable carbohydrates (Nocek 1997; Shaver 1997). Bramley (2006, unpublished) in a study of rumen conditions in 100 Australian dairy herds found that herds with a high prevalence of acidotic cows had a high prevalence of lame cows. Laminitis can be defined as acute, sub-acute, chronic and sub-clinical, depending on the severity and duration of the condition.

The aetiology of laminitis has been extensively researched and reviewed in the past (Vermunt 1992; Nocek 1997; Ossent and Lischer 1998). Acute laminitis occurs after a metabolic insult, such as excessive feeding of a highly fermentable carbohydrate source, results in decreased ruminal and subsequently systemic pH. However, ruminal pH may have returned to normal when laminitic lesions are first diagnosed, as a result of the four to eight week delay between nutritional insult and the onset of laminitis.

The reduction of systemic pH activates a vasoactive mechanism, increasing digital pulse and total blood flow (Nocek 1997). Engorgement of digital veins and dilation of arterio-venous anastomoses of the lower limb result, and divert blood from other peripheral tissues (Ossent and Lischer 1998). Vasoactive substances that may cause vascular constriction and dilation include histamine, lactic acid, serotonins or endotoxins (Westwood and Lean 2001). Increased blood pressure results in damage to the blood vessels and oedema. Ischaemia and hyperaemia of the corium, causing tissue anoxia, may become evident clinically four to eight weeks after the initial insult occurs (Westwood and Lean 2001).

Signs include sole haemorrhages, discolouration of the horn from white to yellow and softening of the horn texture. Sub-clinical and chronic laminitis may also develop following a similar aetiology, but are not associated with systemic symptoms of illness. Claw lesions are associated with the production of inferior quality hoof horn, changing the conformation of the hoof. The dorsal wall surface may appear concave, with horizontal (growth arrest) lines extending around the circumference of the wall (Westwood and Lean 2001). The sole may appear flattened with overgrowth of the abaxial wall. Chronic laminitis probably occurs following repeated nutritional insults to the claw.

A practical assessment of lameness in a herd was developed (Sprecher et al. 1997) using a simple scoring system. A number of individual cows from the herd are scored for locomotion on a scale of 1 to 5, where 1 = clinically normal, 3 = moderately lame with the cow standing and walking with an arched back and 5= severely lame, showing pronounced arching of back and refusing to weight bear on one limb.

Similar changes to those outlined in dairy cattle occur in beef cattle and sheep.

> **Rule of thumb**
>
> *More than 5% prevalence of lameness score 2 cows is cause to investigate acidosis as a possible factor in a lameness problem. However, track condition, moisture and other disorders must also be assessed.*

A herd with a high prevalence of lameness. The cows are standing with arched backs and lowered heads, especially to take the weight off their hind limbs.
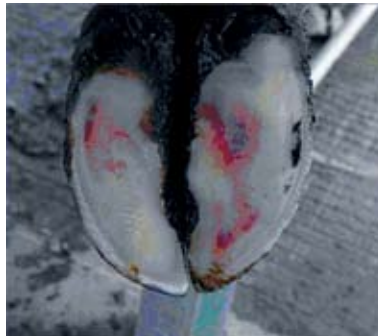
Paint brush sole haemorrhages and white line disease.

Lamintic rings – these are the result of **an outbreak of acute laminitis** approximately two months previously.

Abnormal horn formation due to production of inferior quality hoof horn.

Indirect indicators of ruminal acidosis in sheep

Chewing time: Sheep with sub-clinical acidosis tend to become inappetant and stop chewing their cud.

Faecal changes: Affected sheep pass faeces of a lighter colour which may have an acidic smell. Whole undigested cereal grains are seen in the faeces.

Laminitis/lameness: Laminitis is seen in sub-clinical cases, with sheep being reluctant to move. The first signs are seen about 24 to 48 hours after the first ruminal changes develop.

Lactating ewes fed a diet comprised mainly of cereal grain and affected with sub-clinical acidosis will produce lower milk production in relation to the feed on offer and the animals may develop a sub-clinical mastitis. This can be detected by an increased somatic cell count.

Young sheep in feedlots will have a reduced daily weight gain that can be detected if sheep are regularly weighed.

It is essential that shy feeders are identified early in the adaptation phase of a change to a diet containing a high content of soluble carbohydrate. These sheep should be removed and fed in a separate enclosure of similarly affected sheep at a lower stocking rate, or be returned to pasture.

Indirect indicators of ruminal acidosis in feedlot cattle

- Decline in pen feed consumption of more than 10% for two or more consecutive days.

- A pen incidence of bubbly scours of more than 3% on any given pen inspection.

- Evidence of laminitis in any *Bos taurus* cattle and more than 3% of *Bos indicus* cattle.

Bubbly scours
– a clinical sign of
ruminal acidosis.

Carbohydrate metabolism and requirements and rumen ecology of carbohydrate metabolism

Rumen bacteria can be divided into several groups based on carbohydrate substrate affinity; cellulose, starch, hemicellulose and sugar digesters, and acid utilisers. There is considerable recent work using microbial meta-genomics methods to examine the population of rumen micro-organisms. It can be anticipated that current understandings will improve with better characterisation of the organisms present.

Hydrolysis of carbohydrates

The majority of carbohydrates in ruminant feed are polymers; cellulose, hemicellulose, pectin and starch. The first step in the fermentation of dietary carbohydrates is the breakdown of polysaccharides to oligosaccharides and disaccharides (Baldwin and Allison 1983). Bacteria achieve this hydrolysis of polysaccharides by secreting extracellular enzymes.

Cellulose and hemicellulose are the most abundant of the plant carbohydrates. Hydrolysis of these to the disaccharide cellobiose occurs by the secretion of an extracellular cellulase. *Ruminococcus albus* and *R. flavefaciens* both have the ability of producing extracellular cellulase in response to the presence of cellulose (Baldwin and Allison 1983). Both cellulose and hemicellulose are fermented slowly in the rumen.

Pectins are an important group of structural carbohydrates found in the plant cell wall and are fermented much more rapidly than cellulose or hemicellulose. Pectins are hydrolysed by *F. succinogenes*, *P. ruminicola*, *B. fibrisolvens* and *S. bovis* by secreting methylesterase and polygalacturonidase.

Starch content of grains versus some fibrous feeds (% of DM):

Grains	55-70%
Rye/clover pasture	4-7%
Oaten hay	5%
Rye/clover hay	2%

Starch (amylose and amylopectin) is hydrolysed by microbial amylases to maltotriose, maltose and some glucose (Baldwin and Allison 1983; van Houtert 1993). Starches and other storage carbohydrates are not present in high concentrations in fibrous feeds. These are more prevalent in grains and other concentrated feed sources. The fermentation rate of starch depends on the type of starch being degraded and on physical form. For example, starch from barley grain is fermented more rapidly than that from corn or sorghum grain (Waldo 1973). Heating cereal grains is known to break the crystalline structure of cereal starches and increase rates of hydrolysis and fermentation (Theurer 1986; Zinn 1993). Zinn (1993) found that steam rolling barley, as opposed to dry rolling, increased the net energy for the maintenance value of barley by 2.8% to 7%. This process is probably uneconomic in many situations, however the routine process of cracking, rolling and crushing cereal grains is a common practice to increase availability and rumen degradability, resulting in increased milk yields for cows (Yang et al. 2001).

The disaccharides, i.e. cellobiose, maltose, xylobiose and glucuronides, are broken down into their constituent monosaccharides; xylose and hexoses. Most microbes ferment monosaccharides (sugars) through anaerobic glycolysis.

The biochemical pathways involved in carbohydrate fermentation in the rumen have been extensively reviewed (Hungate 1966; Baldwin and Allison 1983; Czerkawski 1986; van Houtert 1993). The primary pathway (Figure 2) fermenting fructose, maltose and cellobiose to hexose, glucose and then pyruvate is the Emden-Meyerhof-Parnas pathway of anaerobic glycolysis (Baldwin and Allison 1983). The net products from this pathway are 2 ATP/mole of hexose fermented to pyruvate, 2 pyruvate and 2 NAD^+. There is a need for an electron sink product in this pathway to regenerate NAD^+ from NADH in order to maintain ATP production. Electron sink products are critical for the formation of ATP, which is used by bacteria for maintenance and growth. Electron sink products include propionate, methane butyrate, lactate, ethanol and hydrogen. Acetate is not an electron sink product. The other pathway is the pentose pathway converting xylobiose and uronic acids. This pathway feeds into the pathway of anaerobic glycolysis with the net ATP yield being 1.67/mole of pentose used when forming an electron sink product.

The fate of pyruvate and NADH formed in the rumen depend greatly on the substrates available in the rumen and, therefore, the microbes involved. The most common pathway of pyruvate conversion to propionate is the succinate pathway, where pyruvate is converted to oxaloacetate, succinate and then propionate. This pathway is cobalt and vitamin B12 dependent. The minor of the two pathways is the acrylate pathway converting pyruvate to lactate and then propionate via a number of intermediary steps. The acrylate pathway is used by *M. elsdenii* and *P. ruminicola* (Baldwin and Allison 1983). Pyruvate can also be converted to acetate through acetyl CoA, with the ATP yield being 1 ATP/mole, and is used by *Clostridia*, *M. elsdenii* and *V. parvula*. Butyrate is converted from pyruvate through an additional step to aceto-acetyl CoA, regenerating NAD^+ from NADH, therefore resulting in an electron sink product. Formate is effectively a by-product of the conversion of pyruvate to acetyl-CoA when producing acetate. The formate released is converted to CO_2 and H_2 which are converted to methane.

In ruminants fed predominantly roughage diets high in cellulose, intermediate in soluble sugars and low in starch, cellulolytic and saccharolytic bacteria predominate. Cellulose is hydrolysed primarily to acetate and methane via anaerobic glycolysis, methane is the electron sink product. Propionate amounts produced on cellulose predominant diets are 15% to 20% of the total VFA pool. When cereal grains containing starch are fed in increasing concentrations, amylolytic bacteria fermenting this substrate produce a higher proportion of propionate (35% to 45% of total VFA pool) (Orskov 1986). This is not only due to high starch fermentation, but also because the fermentation products formed from other carbohydrates are altered to favor propionate (Baldwin and Allison 1983). In comparison to propionate and acetate, butyrate is quantitatively the least important of the three major VFAs in the rumen fluid of animals fed roughages, usually contributing only 0.05 M to 0.1 M to the total VFA pool (van Houtert 1993). The longer-chain volatile fatty acids are present in smaller, insignificant quantities.

Cellulolytic bacteria

Two gram negative, cellulolytic bacteria are prevalent when cellulose is fed to cattle; *Fibrobacter (Bacteroides) succinogenes* and *Butyrivibrio fibrisolvens*. *Clostridium lochheadii* a gram positive bacteria, is also relatively common (Hungate 1966). *Ruminococcus albans* and *Ruminococcus flavefaciens* also present on cellulose-dominant diets. Latham, Sharpe et al. (1971) found that the genera *Butyrivibrio* and *Ruminococcus* both decreased in concentration as the percentage of grain (barley or maize) increased in the ration. The ruminococci are extremely sensitive both to the feeding of monensin and a decrease in rumen pH. *R. flavefaciens* did not survive a pH of less than 6.1 in continuous culture (Russell and Dombrowski 1980).

Amylolytic bacteria

Amylolytic bacteria digest starch, i.e. amylose and amylopectin, and include a number of cellulolytic bacteria, e.g. *C. lochheadii*, and some strains of *F. succinogenes* and *B. fibrisolvens* (Hungate 1966). Other starch digesters include strains of *Prevotella (Bacteroides) ruminicola, Streptococcus bovis*, *Bacteroides amylophilus*, *Succinomonas amylolytica* and *Selenomonas ruminantium* (Hungate 1966). These bacteria are found in greater concentrations in the rumen of animals fed diets high in carbohydrates. Cows fed a diet of 20% hay, 80% rolled barley or flaked maize showed increases in *Selenomonas*, *Prevotella* and *Streptococcus* genera compared with cows fed a 100% hay diet (Latham, Sharpe et al. 1971).

P. ruminicola, a gram negative rod or coccobacillus is found in many different diets and has accounted for about 60% of the isolates in cattle fed on silage (Van Gylswyk 1990; Stewart et al. 1997). There is wide genetic divergence between strains in this group (Hudman and Gregg 1989). *P. ruminicola* cannot degrade cellulose, but probably utilises starch and plant cell wall polysaccharides, e.g. xylans and pectins (Stewart et al. 1997).

S. bovis, a gram positive, non-motile coccoid is capable of very rapid growth when the diet changes suddenly from hay to cereal grain (McAllister et al. 1990). Normally, when rumen pH declines, bacteria can accumulate toxic concentrations of anions such as acetate, however, as extracellular pH declines, the intracellular pH of *S. bovis* is also lowered, thereby avoiding toxicity (Russell 1991a; Russell 1991b). Although *S. bovis* is predominantly amylolytic it is present in forage-based diets (Hungate 1966; Russell and Hino 1985). The major fermentation product of *S. bovis* is L-lactate, although some strains also produce formate and acetate (Stewart et al. 1997; Al Jassim and Rowe 1999). *S. bovis* isolates are very sensitive to virginiamycin (Al Jassim and Rowe 1999).

S. ruminantium, a gram negative rod, is also found in high numbers in the rumen of animals fed cereal grains. *S. ruminantium* provided 22% to 51% of the total bacteria in steers fed cracked corn and urea (Caldwell and Bryant 1966; Hungate 1966). Some strains also utilise D-L lactate (Henderson 1975). During growth in high glucose environments, D and L – lactate is the major fermentation product, but this is replaced by acetate and propionate in low glucose environments (Melville et al. 1988a; Al Jassim and Rowe 1999). Strains able to utilise lactate and grown on glucose can convert accumulated lactate to acetate and propionate. Virginiamycin does not affect growth of *S. ruminantium* in the rumen, because being gram negative, *S. ruminantium* isolates are instrinsically resistant to virginiamycin, even at concentrations of 8 µg/ml *in vitro* (Al Jassim and Rowe 1999).

Hemicellulose digesters

Hemicellulose is digested in the rumen to the same extent as cellulose (Heald 1953) by a number of bacteria, including *P. ruminicola*, *B. fibrisolvens, R. flavefaciens and R. albus.* All cellulolytic bacteria can digest hemicellulose. Optimal pH for hemicellulose digestion is between 6.0 and 7.0.

Fermentation of sugars in the rumen

Fermentation of sugars is the primary source of energy for the formation of the high-energy phosphate bonds of ATP that are utilised by rumen protozoa, bacteria and fungi for maintenance and growth (Baldwin and Allison 1983). The main end products from this fermentation process are methane gas, carbon dioxide and volatile fatty acids (Figure 2). The volatile fatty acids present in highest concentration in the healthy rumen are the three volatile fatty acids associated with carbohydrate fermentation – acetic acid, propionic acid and butyric acid – although concentrations vary depending on the nature of the carbohydrate source in the diet (Hussein et al. 1991). Annison (1954a) demonstrated the presence of isobutyric, isovaleric and 2-methylbutyric acid in the rumen of sheep maintained on various diets. These iso-acids derived from amino acids are essential nutrients for cellulolytic bacteria. Valeric acid is an important indicator of risk of acidosis because lactic acid is converted to this by lactic acid utilising bacteria and protozoa. Traces of formic acid were occasionally observed (Annison 1954b), but constituted a maximum of only 5% of the total volatile fatty acids present in the rumen (Gray et al. 1951). D and L lactic acid can also be present in the healthy rumen in small quantities (Kuncharapu et al. 2000), however concentrations increase with the increase in concentration of starch in the diet (Lee et al. 1982; Al Jassim and Rowe 1999). Kuncharapu et al. (2000) reported that sheep maintained on lucerne pellets had concentrations of 1.54 +/- 0.59 mM D-lactate and 0.78 +/- 0.15 mM L-lactate present in the rumen.

> **Definition**
>
> *Fermentation is the anaerobic, enzymatic conversion of organic compounds, especially carbohydrates, to simplier compounds.*

Sugar fermenters

Sugar-fermenting bacteria are very important because many forage plants contain appreciable concentrations of sugars. Genetic gain to produce higher-quality grasses has been effective, with seed companies improving growth rates and the nutritive value of pastures. Thomas (1960) found that ryegrass, for example, contains 1% glucose, 1% fructose, 9% sucrose and 19% fructan. New tetraploid ryegrasses can contain approximately 20% sugar (unpublished information).

A number of species of *Lactobacillus* present in the rumen are important sugar fermenters. *Lactobacilli* are common in the rumen of young animals on milk rations and also when high grain diets are fed to older animals. *Lactobacilli* present in the rumen as commensals include oxygen-tolerant species such as *L. acidophilus*, *L. casei*, *L. fermentum*, *L. plantarum*, *L. buchneri*, *L. cellobiose*, *L. helveticus* and *L. salivarius* (Hungate 1966). Two anaerobic gram positive rods, *L. ruminus* and *L. vitulinus*, were first isolated from the rumen and produce predominantly L-lactic acid and D-lactic acid, respectively (Stewart et al. 1997; Al Jassim and Rowe 1999).

Acid-utilising bacteria

Acid-utilising bacteria are also found in the rumen on grain diets where there is a high production of lactic acid, e.g. *Selenomonas ruminantium*, *Veillonella parvula*, *Megasphaera elsdenii* and *Anaerovibrio lipolytica*. *M. elsdenii*, a gram negative coccus, is by far the most significant of this group of bacteria. *Megasphaera elsdenii* is found primarily in calves and cattle receiving rations high in grain (Hungate 1966; Stewart *et al.* 1997). *Megasphaera elsdenii* utilises 60% to 95% of the lactate available in the rumen (Counotte *et al.* 1981) and valerate is a major product from this substrate (Stewart et al. 1997). *Megasphaera elsdenii* takes five to seven days to establish rumen populations of 5.5×10^{11} per ml in concentration after the introduction of grain to the diet (Klieve et al. 2003). In feed-lot cattle, it appears in the rumen in conjunction with *S. bovis* and other lactic acid-producing bacteria. However, as pH decreases below 5.5, the growth of *M. elsdenii* is inhibited and lactate production exceeds its utilisation in the rumen, depressing pH even further (Russell and Allen 1983). Other users of lactic acid include *Selenomonas ruminantium ss lactilytica* and entodidiomorph protozoa. The key electron sink products for the hydrogen from lactic acid are valerate and propionate.

Anaerovibrio lipolytica has two key roles in the rumen; the hydrolysis of lipids and utilisation of lactate. This gram negative rod has been isolated from the rumen of animals fed both predominantly concentrates and roughages, with major substrates being lactate, glycerol and fructose.

Veillonella parvula (previously *alcalescens*), a gram negative micrococcus is very similar to *Selenomonas* based on 16S rRNA sequencing, and ferments D and L-lactate to acetate and propionate. *V. parvula* also has the ability to ferment glycerol. Numbers of this bacterium in the rumen appear small and it is probably not of great significance, even on starch-based diets (Hungate 1966; Stewart et al. 1997).

Fibre definition and requirements

Fibre definition

Fibre is the slowly digestible or indigestible fraction of feeds that occupies space in the gastro-intestinal tract of animals (Mertens 1997). These are the lignin, cellulose and hemicellulose fractions of the plant. There have been numerous attempts to classify nutritional components of feed, thereby establishing quantities in various feeds and requirements by ruminants for those various components. The system most recently embraced for describing the energy content of plant material, the Neutral Detergent Fibre (NDF) method was initially developed by Van Soest and Wine (1967).

Fibre requirements in the diet

Ruminants require fibre in the diet to maximise production and maintain health by sustaining a stable environment in the rumen (Allen 1997; Grant 1997a). An adequate intake of neutral detergent fibre (NDF) and acid detergent fibre (ADF) is necessary for maintaining ruminal pH within the normal range (Jung and Allen 1995; Mertens 1997).

The minimum requirement for fibre in the diet remains poorly defined. Fibre must be of high quality and sufficient particle size to ensure maximum dry matter intake (DMI) (Journet and Remond 1976; Spahr 1977; Wangsness and Muller 1981) and optimal chewing activity (Grant et al. 1990a), rumen fermentation and milk fat percentage (Van Soest 1963; Woodford, Jorgensen et al. 1986). Inadequate dietary fibre intake has been associated with low milk fat, rumen acidosis and dietary inefficiency in dairy cattle (Van Soest 1994). The ability of roughages to physically stimulate chewing time is particularly important due to the increased production of salivary buffers during chewing, aiding in the maintenance of rumen pH above 6.0 and preserving optimal rumen function.

NDF

The US National Research Council (NRC) for Dairy Cattle (2001) recommends a minimum of 25% NDF in the total dietary DM, 75% of which is supplied by coarse forage to maintain rumen function and health.

This sets the limit for the use of non-forage fibre sources that are less effective in stimulating chewing than forage fibre. Diets contributing less NDF from forage than 75% of total NDF or if they are not fed as a total mixed ration, require higher minimum concentrations of NDF (National Research Council 2001). However, because the effectiveness of fibre within by-product feeds and forages is variable, due to rumen fermentation being variable, chemical and physical properties alone should not be used in isolation. Measurements of NDF, being based purely on chemical analysis, are therefore not sufficient to define effective fibre.

eNDF and peNDF

Recent research has aimed at defining fibre requirements using a combination of both chemical and physical factors. Two factors have been suggested in this equation; physically effective NDF (peNDF) and effective NDF (eNDF). Physically effective NDF is related to the physical characteristics of fibre that influence chewing activity (Mertens 1997) and would be expressed as a product of NDF concentration and physical effectiveness factor determined by total chewing response (Grant 1997). Physical effectiveness is determined by attributes such as chewing time and the biphasic nature of ruminal contents (floating mat of large particles on a pool of liquid and small particles) (Armentano and Pereira 1997; Mertens 1997).

Chewing time is strongly related to forage content ($p<0.05$) (Woodford, Jorgensen et al. 1986) and particle size (Grant et al. 1990b) and is a reasonably practical variable to measure. Mat consistency, although a good outcome, is difficult to measure (Armentano and Pereira 1997). Feed characteristics also determine peNDF; fibrousness, roughage value, physical structure and particle size of the feed all contribute to this.

Effective NDF is related to the total ability of a feed to replace forage or roughage in a ration, so that the percentage of milk fat produced by cows eating the ration is effectively maintained (Mertens 1997). The animal response that is associated with eNDF, therefore, is milk fat percentage; the extent of milk fat depression associated with a feed. Effectiveness factors for NDF can vary between zero when a feed has no ability to maintain milk fat percentage to greater than one when a feed maintains milk fat percentage more effectively than maintaining chewing activity (Mertens 1997).

Mertens (1997) found that a minimum peNDF intake of 22.3% of the ration DM, was required to maintain a ruminal pH of 6.0. Kolver (1998) proposed that high-quality pasture had an eNDF between 40% and 50% of total NDF. Currently, there is no accurate estimate of eNDF and peNDF for perennial ryegrass or white clover pastures, however the eNDF of similar Australian pastures has been estimated to be 65% to 75% of total NDF (Porter et al. 2001). A study incorporating results from 14 data sets from cows grazing pure pasture indicated that 41.1% NDF or 11.5% effective NDF (of DM per cent) was required to maintain rumen pH at 5.8 to 6.0. This figure corresponded to 29% eNDF of total NDF (Kolver and de Veth 2002). Under some conditions the diet of pasture-fed dairy cows may contain less than 20% eNDF and cows could be at greater risk of ruminal acidosis. It is important to note that even limited supplementation of low NDF pastures with concentrates will produce diets that have extremely low NDF concentrations. Many Australian pastures are low in NDF in late autumn through to later spring.

Conclusions

Many pasture-based diets, especially those supplemented with concentrates, will be near the lower limit of effective fibre required in the diet. Stating a single requirement for eNDF does not ensure that this will be sufficient to meet the needs of cattle because of the difficulty in determining the true eNDF of pastures. Estimates of diet eNDF should be matched with observations from the field of cud-chewing, faecal formation, milk fat and protein content, and rumen pH to ensure that cows are receiving a diet that will keep rumen function optimal and safe.

Definition

Physical effectiveness factors (PEF) are assigned to different feeds based on the ability of a feed to stimulate chewing activity.

Long-stem hay is given a value of 1.0, rolled barley has a PEF of 0.69. The peNDF is calculated by multiplying the PEF for a feed by the chemical NDF value.

Rule of thumb

The target peNDF for cattle is 22% of DM.

Physiology of indicators of adequate fibre in the field

Bulk milk fat concentration

Effective NDF accounts for factors that affect peNDF; factors that affect ruminal acid production and milk fat production. A change in ruminal acid production affects ruminal pH, affecting the buffering capacity of the rumen and, therefore, the quantities and ratios of volatile fatty acids produced, directly impacting on milk fat percentage (Mertens 1997).

Maintaining milk fat percentage has been the focus of much of the recent research and field application of effective fibre (Beauchemin and Buchanan-Smith 1989; Grant et al. 1990 a&b; Beauchemin 1991; Armentano and Pereira 1997; Krause et al. 2002 a&b); hence the interest in measuring eNDF. Milk fat percentage can have significant economic impacts, is easy to measure and reflects animal performance. It is, however, a reasonably insensitive measure of effective fibre, with some studies documenting no change in milk fat percentage associated with improved rumen function when roughage in the diet was increased (Beauchemin and Buchanan-Smith 1989). This indicates that other factors, such as chewing activity, ruminal pH and saliva production, measured in collaboration with milk fat percentage, may be useful in assessing more accurately effective fibre. Certainly, the trans 10 conjugated fat content in the rumen and amounts of precursors, e.g. linoleic acid and linolenic acid, in the diet may also have profound effects on milk fat content (National Research Council 2001). Thomson et al. (2001) reported that high-quality pasture contains 4-5% total lipid, 60% of which was linolenic acid and 13% linoleic acid. Changes in rumen biohydrogenation increase the molar proportions of trans fatty acids that inhibit milk fat synthesis (Kolver and de Veth 2002).

> **Definition**
>
> *Biohydrogenation is the process of taking unsaturated fats (containing double bonds) and saturating (breaking) them by adding a hydrogen ion where the carbon bond used to be.*
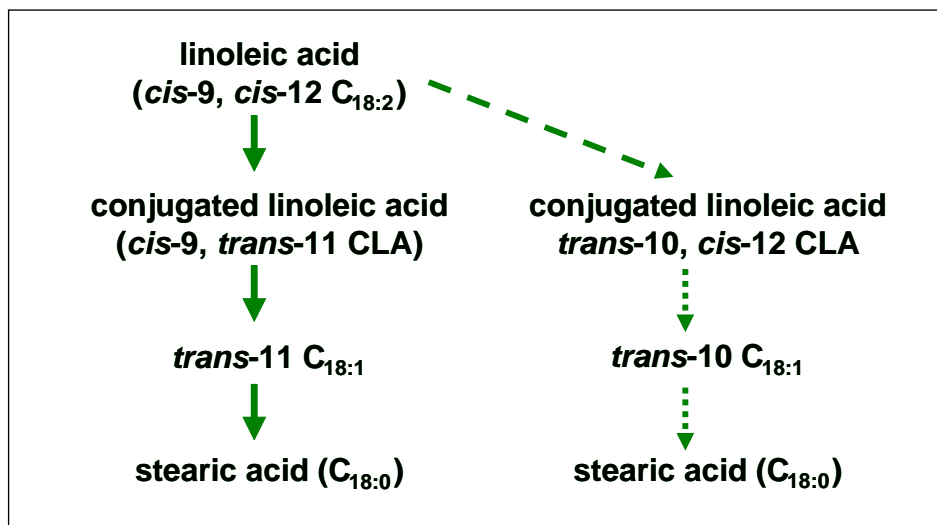
Pathway of biohydrogenation
(From Griinari and Bauman 1999).

To enable these changes to occur, it is also proposed that rumen pH must be low (National Research Council 2001). Low fibre concentrations in the diet may be one contributing cause to this low pH. Kolver and de Veth (2002) found that although data reported on analysed diets fulfilled both requirements, large decreases in milk yield and fat percentage were not observed.

Optimal chewing time per kilogram dry matter has been estimated at between 24 minutes chewing activity (Woodford and Murphy 1988) to maintain milk fat percentage at 2.9% and 30 minutes to maintain milk fat percentage and optimal rumen function (Mertens 1997). Mertens (1997) analysed data from 36 studies to establish chewing requirements to maintain milk fat percentage (Table 2).

Table 2: Summary of chewing requirements in dairy cattle (adapted from Mertens 1997)		
Requirement	Chewing time/day (min)	Chewing time/kg DM (min)
3.4% milk fat	589	27.7
3.6% milk fat	744	36.1

Saliva production, associated with chewing time, may also be an important physical measurement for effective fibre (Balch 1971; Allen 1997). Saliva acts to buffer acids produced during ruminal fermentation of feeds, thereby promoting fibre digestion. Eating and ruminating stimulate the flow of saliva, although some flow continues constantly. Saliva secretion is estimated to be 1.5 to two times higher during chewing compared with resting (Cassida and Stokes 1986). The rate of eating is important in determining the buffering of the feed-saliva mixture (Van Soest 1994). High concentrate and pelleted forage diets are characterised by less net saliva flow than diets higher in roughage (Van Soest 1994). Beauchemin et al. (1989) estimated that total saliva production for cows increased by 7 litres per day, or 2.6%, when corn silage diets were supplemented with hay producing one hour's increased chewing time. These 7 litres provide an increase in HCO_3^- of approximately 875 meq and HPO_4^{2-} of 175 meq (Lean 1987).

Prevention of ruminal acidosis

'Because of fluctuations in pasture availability and quality, and inefficiencies in concentrate delivery systems, dairy cattle on many farms will be exposed to a high level of risk of acidosis several times over a lactation and regularly during a lifetime.'

Adequate fibre

Fibre requirements and indicators of adequate fibre in the diet, including milk fat percentage, chewing activity and faecal examination, are discussed in this document. There is some disagreement on the most appropriate way to define the optimum amounts of fibre in the diet and recommended minimum concentrations of fibre. The current system of definition using NDF may not be adequate to describe the effectiveness of fibre in the diet to stimulate salivation and rumen stability. Notwithstanding this, there is agreement that a combination of chemical and physical properties of fibre in the diet should be described.

Chewing time is particularly important in the prevention of ruminal acidosis. This action stimulates the flow of saliva and buffers the rumen in a pH range between 6 and 7. Chewing time is increased when long-stem forages are fed and reduced when particle size is decreased, either by reducing forage particle size (Krause et al. 2002b) or increasing the processing of concentrates. Fibre is often an option to aid in the prevention of acidosis, depending on supply in the region. Many dairy farmers choose to feed a poor-quality fibre source, including low-quality hay or straw. There are two potential problems with this approach. First, stock may eat good-quality lush pastures and concentrate supplements in preference to the poor-quality fibre sources offered. Second, when stock eat poor-quality fibre sources with low crude protein percentages and metabolisable energy values, the need increases for higher-quality forages or concentrate supplements to meet energy and protein requirements.

Shepherd (2002) showed by using simulation modeling that many cows within large dairy herds have limited access to grazed forage. This problem arises under circumstances where cows that are milked early in the milking process are given access to pasture before those milked later, who, consequently, have inadequate intakes of fibre.

It is recommended that under circumstances when pastures are lush or limited, good-quality cereal hays, additional pasture or legume hay should be offered to assist in prevention of acidosis. It is important to ensure there is adequate distribution to ensure that all cattle get access. The amounts of forage in the diet should be calculated to provide more than 32% NDF, with greater than 80% being sources from long forage.

Two different methods for providing additional fibre to cows.

Beef cattle fibre requirements

The roughage requirements for lot-fed beef cattle are inadequately defined. De Campeneere et al. (2002) investigated the effects of decreasing the roughage:concentrate ratio of a diet. They determined the proportion of roughage at which changes in rumen parameters indicated that further reduction in the proportion of roughage could negatively affect production. This study measured rumen changes in the study group over time as the proportion of roughage was decreased and was, therefore, not structured to make production comparisons for different proportions of roughage in the diet. However, significant effects on *in sacco* degradability of dietary components indicated minimum roughage inclusions for maize silage and straw when fed with a range of rapidly fermented by-products.

De Campeneere et al. (2002) found that a minimum of 8.1% straw (DM basis), or 14.7% maize silage (DM basis), was necessary to prevent a decrease in *in sacco* degradability of maize silage, grass silage and wheat grain when fed with a concentrate diet. From these diets it was calculated that the minimum NDF requirement was 41% for the straw diet and 31.5% for the maize silage diet. Conventional Australian feedlot diets frequently have only approximately 13% NDF. There are several differences between the experiment diet of De Campeneere et al. (2002) and the diets commonly used in Australian feedlots. The trial diet had extremely rapidly fermented concentrates such as citrus pulp, tapioca, sugarbeet pulp, beet molasses and beet molasses solubles that were high in sugars. Only the maize silage-based diet contained cereal grain; wheat at the very low inclusion of 5%. Further, the roughage length in the experiment diet was only 16 mm for the straw and 8 mm for the maize silage, and no rumen modifiers were included. Most Australian feedlot diets, by contrast, have high inclusions of cereal grains that, in most, cases are either tempered, steam rolled/flaked or reconstituted; generally, only small feedlots dry roll grain. The currently accepted rule of thumb for target roughage length in the Australian feedlot industry is 50 mm to 100 mm. Commercial feedlots in Australia almost always include a rumen modifier to prevent or assist in the control of ruminal acidosis. Therefore, De Campaneere et al. (2002) have demonstrated, using *in sacco* degradabilities, that NDF concentrations of 30% to 40% are necessary with extremely rapidly fermented diets, where the roughage is approximately one order of magnitude shorter than the target length, and in the absence of rumen modifiers. Further, De Campeneere et al. (2002) showed that rumen pH, and more importantly, gross clinical signs, are poor indicators of the effects of inadequate roughage on the efficiency of rumen fermentation as measured by *in sacco* degradability of silage and grain.

Further research is required to define roughage/fibre requirements for Australian lot-fed cattle. Experiments measuring rumen changes presumably correlated to production and economic outcomes, such as *in sacco* degradabilities of major diet ingredients, and using diets common to the Australian feedlot industry, could generate a range of NDF concentrations at defined roughage lengths for investigation in commercial settings. The commercial experiments would allow the relationship between NDF and *in sacco* degradabilities to be correlated with production responses and therefore profitability over large numbers of cattle.

Similar to lot-fed cattle, the effects of inadequate dietary roughage/fibre concentration on the production and profitability of grazing beef cattle have not been quantified. However, the experiments described above with intensively fed cattle could be used as a starting point for investigation of the production effects of pasture fibre concentrations on grazing cattle. If inadequate pasture fibre concentrations were found to have negative effects in grazing cattle, further research would be warranted to determine cost-effective measures to correct this problem.

Gradual adaptation to starch-rich feeds

The majority of dairy cows in Australasia are fed some concentrate supplements during lactation, usually twice daily during milking. The potential for problems to arise from this system reflects the amount and type of concentrate fed. Further, cows are often fed poorer-quality pastures and higher dietary NDF concentrations during the dry period than during lactation. A sudden dietary shift from high-fibre diets to lower fibre/higher concentrate diets, combined with lower feed intake during the week before calving (Grant and Albright 1996), can disrupt rumen function and place cattle at increased risk from metabolic disorders, including acidosis.

Therefore, it is advisable to feed a transition diet for several weeks before calving. During the transition period, fibre levels in the diet are ideally progressively reduced and concentrates are introduced (Lean et al. 1998). Transition diets often include smaller amounts of concentrates than are fed after calving to allow rumen microbial populations to adjust to a change in diet. Some dairy feeding systems also allow for individual cow identification in the lactating herd and adjustments in concentrate proportions and amount can be made depending on the number of days in milk. Both strategies help minimise the disruption in rumen function, therefore reducing the risk of acidosis.

Transition diets are also often used to introduce other dietary controls for ruminal acidosis used frequently in lactating cow rations, including neutralising agents and rumen modifiers. Buffers and neutralising agents should be carefully assessed to ensure that these do not increase the dietary cation anion difference of the diet before calving. It is recommended that, whenever feasible, concentrate feeding should be distributed through a day, rather than solely at milking. Transition rations should be fed to allow a gradual introduction to concentrate. The NSC content of the lactating diet should not routinely exceed 38%; that of the transition diet 32%.

> **Definition**
>
> *A transition diet is fed for the final 2-3 weeks before calving when the dairy cow makes the transition from being pregnant and dry to being a lactating animal.*

Rumen buffers and neutralising agents

Dietary buffers have been commonly used in dairy cattle diets for a number of years for their perceived positive effect on production and prevention of acidosis. For a compound to act as a buffer in the rumen, it must be water soluble, a weak acid, base or salt thereof and have a pKa near the physiological pH of the rumen. A true buffer should lessen the decrease in pH without causing a pH increase compared with a neutralising agent that elevates pH (Staples and Lough 1989). Buffers used in the dairy industry include sodium bicarbonate, sodium sesquicarbonate, potassium bicarbonate, magnesium carbonate and calcium carbonate, of which sodium bicarbonate is the most common (Erdman 1988). Sodium bentonite is used as a buffer but does not have properties consistent with a buffer.

Neutralising (alkalinising) agents are distinctly different to buffers in that they always cause a pH increase in the rumen. Neutralising agents include sodium carbonate, potassium carbonate, magnesium oxide, sodium hydroxide and calcium hydride (Staples and Lough 1989).

Sodium bicarbonate

Sodium bicarbonate has been well researched in dairy cattle, with the product having been on the market for the past 40 years. Staples and Lough (1989) reviewed 40 studies with typical responses, including an increase in milk production and milk fat content. Fat content was probably increased because of an associated decrease in the production of trans-C18:1 fatty acids through ruminal biohydrogenation (Kalscheur et al. 1997; Kennelly et al. 1999). Staples and Lough (1989) also found an increase in acid detergent fibre digestion in nine of 12 studies reviewed with some articles also reporting increases in ruminal liquid dilution rates. Other responses included increased rumen pH particularly on high concentrate diets in some studies (Kalscheur et al. 1997), increased total volatile fatty acid production and increased acetate to propionate ratios (Kennelly et al. 1999).

Dietary sodium bicarbonate acts as a buffer in the same way as endogenous sodium bicarbonate found in saliva. Sodium bicarbonate works in an optimal pH range of 6.2 to 6.5 with a pKa value of 6.25. Not all studies have shown an increase in rumen pH in sodium bicarbonate treatment groups. There may be two reasons for this finding. Firstly, if the initial rumen pH was less than 6.0, the buffering capacity of sodium bicarbonate is less. Secondly, studies with diets containing greater than 30% dry matter from forage show less-pronounced effects by dietary buffers on rumen pH and milk fat percentage (Erdman 1988). Those studies also averaged 6-7% more ADF in the diet, possibly contributing to higher endogenous bicarbonate buffering in the both control and treatment groups. Good-quality ryegrass/sub-clover pastures in Australia, which are low in NDF and high in soluble sugars, have the potential to induce sub-clinical acidosis (Lean et al. 2000; Bramley 2006), particularly when cattle are supplemented with concentrates. The effect of sodium bicarbonate feeding in this situation needs further research. Clayton et al. (1999) found that feeding 200 g of sodium bicarbonate per day had limited effects on measures of gastro-intestinal tract acidity, including rumen pH. This finding may be associated with the high solubility and short period of effectiveness for sodium bicarbonate in the rumen. The use of buffers should, therefore, not replace the requirement for effective dietary fibre.

Sodium bicarbonate has value in two ways; as a source of sodium to meet sodium requirements and help provide a positive dietary cation-anion balance for lactating cows, and as a buffer in maize silage-based diets (200-300 g/head/day). Further, there are significant milk production benefits for increasing lactating diets to a dietary cation anion difference of 350 to 400 meq per kg.

Rule of thumb

Sodium bicarbonate should not be included in a transition diet.

Magnesium oxide

Magnesium oxide is classified as a slow-releasing neutralising agent because of its undefined pKa and relative insolubility in water (Erdman 1988). However, Erdman (1988) reported the effectiveness of magnesium oxide in raising rumen pH and milk fat percentage. Staples and Lough (1989) reviewed responses to magnesium oxide on corn silage and pasture-based diets with increases in both milk production and fat recorded when magnesium oxide was included at 0.4% to 0.8% of dry matter. The acid-consuming capacity of magnesium oxide is between 41.9 and 49 meq per day, significantly higher than other buffers and neutralising agents such as sodium bicarbonate with 11.9 meq per day (Schaefer, Wheeler et al. 1982). Water solubility, however, is variable and dependent on the particle size of the magnesium oxide product (Erdman 1988). Processing to a minimum size of 425 mm appears to enhance the effect (Staples and Lough 1989). Kalscheur et al. (1997) found the addition of 0.5% magnesium oxide and 1.5% sodium bicarbonate to both low and high-forage diets increased rumen pH. In the same study, buffering had a negative effect on the presence of $trans\text{-}C_{18:1}$ fatty acids in the milk on low-forage/high-concentrate diets, increasing milk fat percentage.

Magnesium is a vital requirement for dairy cattle, especially those on lush pasture. Magnesium oxide (30-45 g/head/day) is a logical means to supply this requirement. Responses to supplementation are very positive in trials conducted on pasture.

Sodium bentonite

Sodium bentonite is a colloidal, hydrated, aluminium silicate clay, consisting principally of montmorillonite. It is not a buffer, but has a high ion exchange and moisture-absorbing capacity (Bringe and Schultz 1969). A number of northern American studies with feed lot dairy cattle indicate bentonite increased milk fat concentrate and yield on high-concentrate, low-fibre diets (Bringe and Schultz 1969). Studies in cows grazing pasture supplemented with up to 10 kg per head per day of grain-based concentrates found no significant effects on milk yield or composition (Hamilton et al. 1988; Ehrlich and Davison 1997). However, Erlich and Davison (1997) found that feeding 4% bentonite did increase rumen pH, lower rumen ammonia, increase faecal protein and decrease faecal starch. The lack of consistent evidence of effect coupled with necessary high feeding rates (0.5 kg to 1 kg per head per day) suggests a limited role for bentonite in controlling acidosis.

Calcium carbonate (limestone)

Limestone has limited buffering capacity in the rumen at physiological pH because of low solubility, despite a high potential to consume acids (Erdman 1988). Haaland et al. (1982) found that limestone had greatest effect on buffering capacity between pH 5.0 and 4.5. However, it may regulate pH in the intestines (Kellaway and Porta 1993), increasing faecal pH when added at 1.8% to 2.5% of the diet (Russell et al. 1980; Haaland and Tyrrell 1982; Haaland et al. 1982). Rumen ammonia and total VFA concentrations do not appear affected with the addition of limestone to the diet (Haaland and Tyrrell 1982; Haaland et al. 1982).

> **Rule of thumb**
>
> *Magnesium oxide has a distinct role as a source of magnesium in the diet. Cows do not have the ability to store magnesium and therefore require a daily intake to maintain normal body functions and prevent grass tetany during times of stress, e.g. winter.*

Rumen modifiers

It is important to consider the regulatory status of veterinary medicines when selecting a rumen modifier.

To find out the current regulatory status of a veterinary medicine or to view product labels, veterinarians and other animal health advisers should consult the approved product database at www.apvma.gov.au.

Any use of a veterinary medicine that is not consistent with labelled directions is considered off-label use.

An appropriate approach to off-label use by veterinarians that has been developed by the AVA is summarised in the APPENDIX on page 55 and should be reviewed as part of the process of rumen modifier selection.

Ionophore rumen modifiers

Ionophore rumen modifiers prevent or aid in the prevention of digestive and metabolic disturbances caused by erratic feed intake or specific feed problems including bloat and acidosis. Ionophores have the potential to aid in the control of acidosis by two distinct mechanisms. The first mechanism is to reduce lactic acid-producing strains of bacteria such as *Streptococcus bovis* and *Lactobacillus spp.* The other mechanism by which ionophores reduce acidosis is through changes in eating dynamics. Subacute acidosis increases variation in dry matter intake (DMI) and decreases total DMI.

Ionophores restore consistent eating behaviour in beef cattle, which contributes to a reduction in digestive conditions including acidosis, feedlot bloat and death. Ionophores available in Australia include monensin, lasalocid, narasin and salinomycin.

Monensin

Rumen modifiers and feed antibiotics play an important and significant role in the Australian dairy industry, with the positive effects of supplementation increasingly well recognised. Sodium monensin is an ionophore antibiotic produced by *Streptomyces cinnamonensis* which selectively modifies the rumen microflora and improves the digestive efficiency of cattle (Lean et al. 2000). Modes of action of monensin include modification of rumen total VFAs and increasing propionate percentage (Burrin and Britton 1986), reduction of feed intake (Thonney et al. 1981; Fox et al. 1988; Abe et al. 1994) and changes to rumen gas production including reduced methane output (Stanier and Davies 1981). Monensin also modifies digestibility of feed (Potter et al. 1976; Raun, Cooley et al. 1976), reduces rumen liquid and solid turnover rates (Lemenager et al. 1978), changes protein utilisation (Horn et al. 1981), increases retention of zinc and selenium and increases milk production (Lean et al. 2000). Milk fat percentage is significantly lower in cows treated with monensin (Abe et al. 1994), however, such results are not consistent. Importantly, monensin has been found to reduce risk of acidosis *in vitro* (Nagaraja et al. 1987) and prevent clinical acidosis in cattle experimentally induced to exhibit acidosis (Avery et al. 1980; Nagaraja et al. 1981) with rumen pH increasing in treated animals (Horn et al. 1981; Nagaraja et al. 1981).

Monensin acts by inhibiting the growth of *S. bovis*, as well as other major lactate-producing rumen bacteria such as *Lactobacillus*, *Butyrivibrio* and *Lachnospira in vitro* (Dennis et al. 1981). D- and L-lactate concentrations in the rumen were lower, rumen pH higher and blood D- and L-lactate, pH and pCO2 did not change in cattle treated with monensin when acidosis was experimentally induced with finely ground corn or glucose (Avery et al. 1980; Nagaraja, Avery et al. 1982). Importantly, ionophore antibiotics must be present for at least two days before carbohydrate challenge in order for effective control of acidosis (Nagaraja et al. 1981).

Table 3 summarises the effects of the ionophore antibiotics compared with non-ionophore antibiotics on the inhibition of lactic acid production *in vitro*. Although monensin on its own only has a maximum inhibition of lactic acid production of 76% compared with the control, the combination of monensin and tylosin can achieve 93% *in vitro* equalling that of virginiamycin. Caution must be exercised in extrapolating from controlled in vitro experiments to recommendations for use in animals under field conditions. The diets used in the studies of Avery et al. (1980) and Nagaraja et al. (1981) were consistent with those used in beef cattle feedlots, but may not necessarily reflect dairy diets which are higher in fibre.

Table 3: In vitro lactic acid inhibition by antimicrobial compounds (adapted from Nagaraja et al. (1987)).						
Compound	Total lactic acid		L(+) lactic acid		D(-) lactic acid	
	[1]IC_{50} (µg/mL)	[2]I_{max} (%)	[1]IC_{50} (µg/mL)	[2]I_{max} (%)	[1]IC_{50} (µg/mL)	[2]I_{max} (%)
Monensin	0.54	76	0.26	80	>24.0	49
Lasalocid	0.57	83	0.30	89	9.5	64
Virginiamycin	0.13	93	0.13	96	0.64	83
Tylosin	0.45	92	0.35	97	1.64	69
Monensin + Tylosin[3]	0.39	93	0.30	98	1.8	69

[1] Concentration of antimicrobial providing 50% inhibition of lactic acid concentration compared with control fermentation.

[2] Maximum inhibition of lactic acid production compared with control.

[3] Monensin and tylosin mixed at a ratio of 3:1 (w:w).

Rule of thumb

Underdosing with monensin significantly reduces efficacy. Monensin toxicity can occur at 2-3 times the recommended daily per cow dose.

Milk production responses indicate that the combined benefits of monensin fed to dairy cattle at 250-300 mg/head/day are reflected in increased milk production (Beckett et al. 1998). Given that monensin has an effect in controlling acidosis, the product is recommended to control disease, increase milk and meat production. Note that monensin is not approved by the APVMA for the prevention of acidosis.

Lasalocid

Lasalocid is also an ionophore with similar activity to monensin, although probably not used as commonly as a feed additive in the Australian cattle industries. Lasalocid treatments of cattle with experimentally induced acidosis have higher rumen pH and lower L- and D-lactate concentrations than control cattle or cattle treated with monensin (Nagaraja, Avery et al. 1982; Nagaraja, Avery et al. 1985). Table 3 indicates maximum inhibition of lactic acid production *in vitro* compared with the control is approximately 83% in comparison to monensin of 76% (Nagaraja, Taylor et al. 1987). Dennis et al. (1981) found that lasalocid delayed the growth of *S. ruminantium in vitro* at concentrations of 6-12 µg per mL for 19 to 25 hours, respectively. Monensin also delayed growth at these concentrations, but only for 10 hours. Increased total VFA concentrations in combination with increased molar concentrations of propionate were also found in lasalocid-treated cattle compared with controls when experimentally induced with acidosis, a similar finding to monensin (Nagaraja, Avery et al. 1981). Thonney et al. (1981) found that lasalocid-treated cattle had higher dry matter intakes (*p*<0.01) and greater weight gains (*p*<0.005) than monensin-treated cattle.

It does appear likely that this product will have efficacy in modifying the risk of acidosis. There is less information available on responses to lasalocid in pasture-fed cattle than on monensin. Lasalocid is an alternative ionophore to monensin and use can be recommended under similar circumstances. Note that lasalocid is not approved by the APVMA for the prevention of acidosis.

Other rumen modifiers

Virginiamycin

Virginiamycin is an antibiotic with primarily gram positive activity. It is effective in reducing lactic acid production *in vitro* (Nagaraja et al. 1987) by removing *S. bovis* (Hedde et al. 1980), the organism primarily responsible for lactic acid production on starch-based diets. Al Jassim and Rowe (1999) found *S. bovis* which produced only L-lactate to be very sensitive to virginiamycin *in vitro* at concentrations of 2 μg/mL. Effects seen on addition of virginiamycin include higher rumen pH, lower *Lactobacillus* and *S. bovis* counts causing lower L-lactate concentrations (Clayton et al. 1999; Coe et al. 1999), higher faecal pH (Clayton et al. 1999) and higher propionate as a percentage of total VFAs on a 22% concentrate diet (Hedde et al. 1980). Other lactic acid-producing bacteria include *L. vitulinus and S. ruminantium.* Sensitivity of *L vitulinus* isolates, which produced D- and L-lactate, to virginiamycin varied from being highly sensitive to resistant at 8 μg/mL virginiamycin *in vitro* (Al Jassim and Rowe 1999). *S. ruminantium*, a gram negative bacterium, also produced D- and L-lactate, and was intrinsically resistant to virginiamycin, as expected, even at 8 μg/mL (Al Jassim and Rowe 1999).

The combination of monensin and tylosin as outlined in Table 3 needs further investigation on pasture-based diets as an alternative to virginiamycin. Other effects of supplementing with virginiamycin include increased average daily weight gain and/or feed conversion efficiency, a reduction in the incidence and severity of liver abscesses (Rogers et al. 1995), and reduced *in vitro* digestibility depressing feed intake in sheep trials when abruptly introduced (Thorniley et al. 1996).

Virginiamycin is an effective agent for controlling the risk of acidosis in dairy herds. It should be considered in circumstances where other means of controlling acidosis are difficult to manage. The product has been used in sheep to control the risk of acidosis in drought feeding and in beef feedlots. Note that currently (July 2007), the only rumen modifier approved for use by the APVMA in the prevention of acidosis is virginiamycin (as Eskalin 500, Eskalin Feed Premix and Eskalin Wettable Powder).

Tylosin

Tylosin, a macrolide antibiotic, has only recently been registered for use as a feed additive in the Australian dairy industry, with a claim for reduction in the incidence of liver absesses in cattle. Tylosin, like virginiamycin, has been shown to be effective in reducing lactic acid production *in vitro* (Nagaraja et al. 1987). Tylosin decreased total VFAs, and reduced D- and L-lactate concentrations in the rumen (Lean et al. 2000). Feeding tylosin reduces liver abscesses, a potential sequel to acidosis, by 40% to 70% (Nagaraja and Chengappa 1998). Note: This is the registered claim for the product in Australia.

The combined response of tylosin and monensin has also been examined (Coe et al. 1999; Lean et al. 2000). In Coe's study, following carbohydrate challenge, rumen lactate concentrations in cattle receiving monensin and tylosin followed the rise observed in untreated cattle for the first 36 hours, then remained low until day four. However, F. necrophorum numbers were kept low. This combination produced significantly lower rumen pH, higher volatile fatty acid and blood urea nitrogen concentrations and lower mean glucose concentrations than animals treated with virginiamycin or tylosin (Lean et al. 2000). The lower pH was probably due to the higher concentrations of total VFAs in the rumen rather than lactic acid which was lower in the plasma than in other treatments not containing tylosin. When tylosin is mixed with monensin at a rate of 1:3, the maximum inhibition of lactic acid production *in vitro* compared with the control is 93%, a rate equaling that of virginiamycin (Table 3). The tylosin-monensin combination also sustained a higher level of milk production, greater weight gain over the trial period than other treatments in combination with significant changes in rumen and blood metabolites (Lean et al. 2000).

Use of tylosin (150 mg/head/day) in combination with monensin or possibly other ionophores should be considered in a similar context to virginiamycin. Certainly more field information on the efficacy of tylosin in preventing acidosis, rather than the sequelae to acidosis, would be useful. Note that tylosin is not approved by the APVMA for the prevention of acidosis.

It should be emphasised that neither virginiamycin nor monensin/tylosin combinations are a tool to support poor management, rather to support herds with short-term or structural feeding problems that increase the risk of acidosis. Virginiamycin is highly effective in controlling rumen fermentation to reduce lactic acid production and is a useful means of reducing health risks from acidosis. It is important to introduce herds slowly to virginiamycin or monensin/tylosin combinations, as a rapid introduction can cause ruminal disruption and in some herds a marked change in urine colour has been noted, with cows producing bright red urine. The colour may arise from alterations in plant pigment metabolism and does not appear to be associated with any adverse physiological effects per se.

Antimicrobial agents and ruminal acidosis

The use of antimicrobial drugs, such as ionophores and other rumen modifiers in food-producing animals has come under increasing scrutiny. It is clearly apparent that the use of antimicrobials in human medicine has led to substantial problems with acquired antimicrobial resistance in the pathogens of humans. This finding has considerable potential to lead to antimicrobial treatment failure and less-effective health delivery in the future.

It has been argued by some that antibiotic use in food-producing animals is not essential. Any putative reduction in the risk of antibiotic resistance in bacteria in food animals that might be achieved by a complete ban or much more stringent regulation is therefore justifiable. There is circumstantial evidence associating agricultural use of avoparcin with the development of resistance to vancomycin in enterococci. It is not possible to argue that there is no risk to humans from antibiotic use in animals, however, despite intensive study there are no studies demonstrating treatment failure in man due to antimicrobial resistance in animals.

There are obvious economic arguments for the continued use of antimicrobials in agriculture, given their positive impact on health and production, but the animal welfare arguments are often overlooked. Several aspects of this debate are highlighted in this review.

Defining the risk

Most disease control, e.g. animal or plant importation, is evaluated using a technique called 'risk analysis'. This discipline is used to identify and quantify the likelihood of events occurring using the best available information on these and calculating the probability of certain outcomes resulting. While it is difficult to exactly quantify all the aspects of risk to humans involved in antimicrobial usage in food-producing animals, risk analysis provides an appropriate means of estimating the risk posed.

Bacteria can rapidly acquire resistance *in vitro* after exposure to antibiotics and there is also evidence showing selection *in vivo* in cattle. The likelihood of persistence of that resistance *in vivo*, the transfer of resistance to pathogens for humans carried by cattle or sheep, or transfer of resistance in commensals of animals to pathogens of humans are harder to define. Even once a human pathogen has acquired resistance, the likelihood that it will cause disease is also not well defined. However, risk assessments conducted on virginiamycin and macrolide antibiotics in cattle indicate that the upper-end risks of continued use of these causing human treatment failure are extraordinarily low (perhaps one failure in a population of 250 million in 200 years).

The need for appropriate stewardship and husbandry of the animals that we care for requires appropriate management methods (as outlined in regard to acidosis in this document), including environmental manipulation, vaccination, genetic selection and therapeutic treatments to ensure the animals do not suffer in the process of food production. It is critical to note that in the case of acidosis, antibiotic treatments are required to care for affected stock and the prevalence of the condition in some populations is high. The use of therapeutic antibiotics may pose a similar risk, however extraordinarily low, to prophylactic treatments of producing antibacterial resistance. These risks have also not been quantified. Therefore, there are competing risks in regard to antibiotic use in this condition that should be considered and provide support for the appropriate prophylactic use of antibiotics.

The need for further data to support efficacy

Any use of antimicrobials to treat animals' needs must be supported by strong evidence of efficacy. Many of the antimicrobials that are used for the control of ruminal acidosis were registered many years ago. There is a relative lack of recent studies, limited studies of efficacy in new areas of application and a lack of data on interactions with other rumen modifiers. More data are needed to confirm the safety and efficacy of long-term administration. The AVA encourages companies selling antimicrobials for production animals to sponsor suitable trials and publish the results of these trials. We also note the challenges facing the sponsoring companies with regard to the high cost of research in comparison to market scale and encourage industry funding bodies to be active in co-sponsoring such studies.

The need for better surveillance

There is a need for a structured approach to provide more effective surveillance of bacterial populations in cattle and sheep. Such surveillance needs to examine not only the presence of bacteria resistant to single drugs, but also the potential for the co-selection of genes for resistance, particularly through changes in the prevalence of multiple resistance to antibiotics. These data would also be valuable in the context of studying the effect of withdrawing antibiotics on enteric bacterial populations.

Yeasts

Live yeast (*Saccharomyces cerevisiae*) has been fed to cattle in varying amounts as part of the by-products of the baking and brewing industries in the form of bakers' yeast and brewers' grains. Both of these products contain high levels of yeast. More refined products based on superior yeast strains and carefully selected yeast media have been developed that have improved fermentation of rumen material *in vitro*. There have been many studies evaluating the use of yeast cultures as rumen modifiers. Many types of yeast supplements are commercially available for dairy producers. The two main types are Yea-Sacc[1026®] (manufactured and marketed by Alltech) and Diamond V XP® (produced by Diamond V Mills).

Yeasacc is a whole yeast preparation and claims to be beneficial through the actions of the living yeast organism in the rumen. It is proposed that the contents of the yeast cell wall aid in the binding of pathogenic bacteria within the rumen, reducing the risk of colonisation of detrimental bacteria and, consequently, ruminal upsets.

Diamond V XP is a yeast culture and relies on the actions of metabolites produced by the yeasts in culture to stimulate the rumen microflora and increase the digestive efficiency of the animal. Biomate Yeast® product (Hansen Biotechnology Industries) is a whole yeast culture, but is not as widely used as the former two products in Australia.

Prevention of ruminal acidosis in feedlot cattle

- Cattle should have access to hay on arrival at the feedlot before induction.
- The starter ration should not contain any more than about 50% grain (more concentrated starter rations are used but the inherent risk should be recognised).
- Cattle should be adapted to the final diet slowly (no less than 10-14 days on a starter diet, 21 days may be used).
- Virginiamycin may be added to the diet at 20 ppm (can be used in combination with ionophores, e.g. monensin 20 ppm/virginiamycin 20 ppm).
- Feed changes must be implemented gradually (e.g. change in rations and grain).
- Avoid fluctuating feed intake:
  - i) the feed trough should not be empty during peak feeding periods (sunrise/sunset);
  - ii) ensure sufficient trough space (guidelines = 25-30 cm/head, 30-38 cm/head and 38-46 cm/head for yearlings, 15 mo – 2 yo, and bullocks, respectively);
  - iii) do not overcrowd pens;
  - iv) feed more often during cold, wet or windy weather, and when barometric pressure drops dramatically; and
  - v) do not increase feed allocation by more than 10% a day once intake has plateaued.
- Ensure rations are consistently mixed.
- Monitor level of fines in ration and ensure grain processing targets are met (dry rolling = two fragments, tempered barley = squash and crimp with maximum two fragments, tempered wheat or sorghum = maximum four fragments, steam flaking = 28-38 kg/hL depending on grain).
- Ensure roughage is of sufficient chop length (mean 5-10 cm long).

Processing target for tempered barley – squash and crimp with maximum two fragments.

Ensure that roughage is not excessively milled – it needs to act as effective roughage. The hay in the photo is not over-processed.

Prevention of ruminal acidosis in sheep

Adequate fibre

Sheep fed a diet consisting of highly digestible, very lush pasture, legume pastures or cereal grain require continuous access to other sources of fibre. This may be included in the diet if a mixing wagon is used, or can be supplied as bales of hay or straw in close proximity to the water supply. Sheep will eat the hay or straw providing that access is sufficient. Productive sheep, whether growing or lactating, require 30% of fibre in their diet.

Many sheep are fed on monocultures of highly digestible crops such as lucerne, brassicas or chicory. It is interesting to note that when given access to a source of fibre, the average daily weight gain of the sheep increases.

Imprinting

On most properties lambs that are likely to be fed cereal grains at some time in their lives are imprinted to grain feeding before weaning. Lambs become imprinted to eating grain by learning from their dams. A grain trail allocating about 50 g of grain per ewe is fed out on four separate occasions in the two weeks before weaning.

This is important because of the need to adapt weaned lambs to a grain feeding system. Typically, the amount of grain on offer is increased by about 50 g per head per day until the target amount is fed. If lambs are reluctant to eat grain these may not do so for the first 10 to 14 days. After this time they become very hungry and may eat a full grain ration to which the rumen has not adapted and acidosis can result. This is often the lighter lambs in the mob that may become subordinate at the competition at the feed source.

Rumen modifiers

Where there is a high risk of acidosis – irregular feeding systems with limited availability of forage or fibre, virginiamycin should be considered.

Risk management guidelines for stockfeed manufacture and provision to animals on farm

There are risks associated with each step in the process, from a stockfeed manufacturer initially taking a feed order from the farmer to providing the feed for consumption by animals on the farm. These guidelines are intended to assist veterinarians and nutritional professionals to identify and minimise these risks.

Step 1: Taking the farmer's feed order (if feed purchased from a stockfeed manufacturer)

- Was the correct feed product ordered for the class of stock?

- Was the inclusion rate of rumen buffers, neutralising agents or rumen modifiers ordered per tonne of feed appropriate for the feed product's intended feeding rate / animal / day?

To minimise these risks:

- <u>Always</u> ensure all verbal feed orders taken from the farmer are confirmed in writing.

- Refer to directions for use for all feed additives to be included, to confirm that the feed inclusion rates are appropriate for the intended feed use before the order is submitted to the stockfeed mill for manufacture.

Step 2: Manufacturing the feed (either at a stockfeed mill or on farm)

- Was the correct feed product manufactured (e.g. grain mix versus grain-balancer concentrate)?

- Were ingredients and additives in the feed product uniformly mixed?

- Were the grain components of the feed over-processed or the feed over-mixed? This can reduce feed particle size, thereby, both increasing the rate of breakdown in the rumen and reducing the physically effective NDF value of the feed.

- Can feed medications carry over from one feed batch to the next?

To minimise these risks:

- Ensure clear identification of all ingredients, feed product names and their ration formulations is maintained through all steps of feed manufacture.

- Ensure the feed mixer is fit for the purpose and is working efficiently at all times.

- Do not include rumen modifiers in powder forms in loose-mix feeds.

Step 3: Transportation of feed from stockfeed mill to farm and transfer into farm silo (if feed purchased from a stockfeed manufacturer)

- Can the feed be accidentally loaded into an incorrect compartment in the delivery truck?

- Can the feed be accidentally delivered to an incorrect farm silo?

- Was there significant feed particle separation during feed transport or as the feed was augured or blown from the delivery truck into a farm silo?

To minimise these risks:

• Ensure stockfeed mill procedures are in place to maintain the identity of feed products during loading and delivery.

• Do not include rumen modifiers in powder forms in loose-mix feeds.

• Consider feed delivery procedures and augurs and farm silo designs that minimise feed particle separation.

• Ensure that there are feed sheets that clearly state the recommended directions for use.

• Ensure farmers are aware that their feed order is designed for a specific feeding rate / animal / day, and that if they wish to change their feeding rate they should order the delivery of a new feed order from the stockfeed manufacturer to a different formulation.

Step 4: Provision of feed to animals on farm for consumption

• Was there adequate animal adaptation to the amount and type of feed product fed by the farmer to maintain rumen function and health?

• Has the farmer changed the daily feeding rate / animal / day of the feed from that which was formulated to be fed?

• Was the feed fed by the farmer without providing an adequate quality / quantity fibre sources as it was formulated to be fed with? This will result in inadequate daily intakes of eNDF by animals to maintain rumen function and health.

• Was there significant feed particle separation as feed was augured from the farm silo to feed troughs?

• Are animals able to sort the feed presented to them into its finer and coarser feed particles and selectively consume this?

• Does the feeding system ensure even daily feed intakes by animals? (be particularly wary of rotary dairies where cows can stay on the platform)

To minimise these risks:

• Educate farmers about the importance of maintaining rumen function and health, and the need to change their animals' diets gradually, while always satisfing their animals' dietary fibre requirements.

• Provide farmers with a delivery docket every time an order of feed is delivered, plus detailed product information.

Ruminal acidosis problems are most likely to occur on farms <u>when</u>:

- The total diet lacks sufficient fibre to stimulate chewing and the production of adequate amounts of saliva to buffer the rumen.

- Animals are grazing lush, leafy, rapidly growing pastures that are low in NDF and high in non-structural carbohydrates. Risk may be exacerbated by:

  – fertiliser application; and

  – lax grazing that allows more opportunity for the selection of high-quality swards.

- Animals are fed high levels of starch-based concentrates:

  – separately from forages, e.g. as Australian dairy cows are commonly fed in the dairy bail twice daily at milking time;

  – in a flat-rate feeding system rather than a system that allows for individual cow ID and feeding variable amounts of grain to suit the stage of lactation or intake;

  – without the inclusion of rumen buffers, neutralising agents and rumen modifiers;

  – containing higher starch cereal grains that are rapidly rumen fermentable (wheat > triticale > barley > oats > soghum > maize); and

  – containing finely ground cereal grains (including pelleted grains).

- Animals are fed rapidly rumen fermentable by-products such as bakery waste, wet brewers grain, apples, grapes and other fruits, and confectionery waste.

- Animals are fed forage-based diets with small particle sizes (i.e. diets containing hays and silages with short chop lengths).

- Animals are fed low pH silages (especially maize, high moisture grains, earledge and whole grain silages).

- Animals are able to sort the feed offered to them, resulting in uneven intakes of feed ingredients and additives, e.g. excessive intake of high starch grains, inadequate intakes of rumen buffers.

- Animals within the group / herd have uneven opportunity to consume forages and starch-based concentrates. For example, in a dairy herd:

  – later milked cows may have less access to grazed forage than cows milked early; and

  – heifers fed in a flat rate bail feeding system will consume more grain-based concentrate relative to grazed forage and their liveweight, than larger, mature cows.

- Animals are not given adequate time to adapt to a change in diet and are placed on finisher diets, e.g. dairy cows that are not fed a transition diet for several weeks before calving or beef cattle that are not provided with a feedlot starter diet.

- Animals get unrestricted access to rapidly rumen fermentable feedstuffs and gorge themselves, e.g. when a gate is left open to a grain storage area – common in wheat/ sheep belt areas.

Treatment of ruminal acidosis

Treatment of clinical acidosis

Treatment of clinical acidosis can be difficult and the chances of success depend on the severity of the case. Individual cattle can be treated successfully, however if a significant proportion of the herd is involved, the logistical challenge of providing treatment is substantial and the prioritisation of cases to be treated based on severity, labour availability and expertise, and the value of the cattle is critical. In a herd outbreak, clinicians should spend some time identifying individuals of the highest value and assessing these first. Treatment of severe cases (dehydration >8%, collapsed and subnormal temperature, static rumen and evidence of scouring) can be unrewarding, time consuming and expensive. These cases should only be treated when economic value determines that the attempt is warranted.

Treatment of mild cases of acidosis includes withholding concentrates and feeding hay to stimulate saliva flow. Additional therapy includes oral antacids such as magnesium hydroxide, magnesium oxide or sodium bicarbonate at 1 g/kg body weight initially to alkalinise the rumen, and oral electrolyte solutions, preferably those containing additional sodium bicarbonate to treat metabolic acidosis.

Severe cases should be treated by withholding concentrates, giving intravenous fluids, e.g. hypertonic saline and access to water or balanced electrolyte solutions not containing lactic acid. There can be a considerable level of dehydration of affected stock because fluid is sequestered in the rumen as a consequence of increased ruminal osmolarity.

Oral antacids, as described above, may be used, however lavaging the rumen with a wide bore stomach tube in combination with transfaunation from a healthy animal is preferable.[4]

Antibiotics including penicillins, tylosin, potentiated sulphonamides and tetracycline should be given to reduce the risk of liver abscessation.

Other supportive treatments include flunixin meglumine (1 mg/kg) for endotoxaemia, antihistamines to control the adverse effects of histamine release, and calcium/magnesium solutions either intravenously or subcutaneously to counteract secondary hypocalcaemia and hypomagnesaemia. Thiamine (10 mg/kg) intravenously every 24 to 48 hours for up to three doses may also be helpful to prevent polioencephalomalacia.

As already discussed in thie document, cattle that are successfully treated in the acute stage of acidosis often show secondary problems such as laminitis.

Rule of thumb

Heart rate in a cow with clinical ruminal acidosis can be used as a prognostic indicator. Cattle with a heart rate greater than 120 beats per minute generally have a grave prognosis, despite treatment.

Rule of thumb

Administer oral antacids to cattle at 1 g/kg body weight initially (The dilution rate for 500 g magnesium oxide and magnesium hydroxide is 5 litres water). One quarter of this initial dose can be re-administered every 6 hours.

Rule of thumb

For a 500 kg cow, 10 litres of isotonic solution (containing 125 g bicarbonate) is required intravenously for every 2% dehydration and should be administered over 10 to 20 minutes.

Treatment of sub-clinical acidosis in dairy herds

In dairy herds with signs of sub-clinical acidosis the following management methods may reduce risk.

Alter concentrate feeding

- Integrate concentrate feeding with the forage portion of the diet if possible.

- Change type of grain to one with less starch or more slowly degradable starch.

- Change processing of grain from fine to coarse.

- Reduce amount of grain fed (allow for increase intake of other feedstuffs if this option is taken).

- Add buffers, neutralising agents and rumen modifiers to the diet.

Grazing management

- Ensure the grazing rotation is optimal (three leaf in autumn/winter, two leaf in spring).

- Ensure that cows are eating pasture to a good residual height (5 cm between clumps).

- Make sure cows have access to a reasonable amount of pasture each grazing (Risk increases if cows are in the same paddock for more than one grazing with no back fence, forage (pasture) intake will vary and may increase concentrate:forage ratio on that day).

- Provide even access to pasture – hold back the herd until all are milked OR ensure that a fresh break is provided for later milked cows.

Increase effective fibre

- Provide a source of effective fibre. Straw>hay>silage. There may be a need to ensure cows eat effective fibre (ensure that fibre is palatable and of high quality) – aim for even distribution of access for cows.

- Try to feed the fibre source as close as possible to when grain is fed to stimulate more saliva flow and buffering capacity.

- Spread out feeding of 'acid dump' feeds, e.g. low pH silage, over each 24-hour period.

Conclusion

Sub-clinical acidosis is an important nutritional problem in dairy herds in Australasia, both in terms of economic impact and as a substantial health problem. Higher-quality pastures containing increasing concentrations of water soluble carbohydrates and less effective fibre, particularly in the winter/spring season, are being offered to dairy cows. This good quality pasture combined with the supplementation of increasing amounts of highly fermentable concentrates, used as a tool to boost production, predisposes the herd to acidosis. A recent Australian study found a point prevalence of approximately 10% of cows less than 100 days after calving that were acidotic. These cows had significantly lower milk fat content than other cows and herds with a high prevalence of these cows had a higher prevalence of lameness and a higher ratio of NSC to NDF.

While various diagnostic tools have been employed to detect acidosis, particularly the prevalence of cows with ruminocentesis pH <5.8, better measures for diagnosis need to be developed and extended. A presumptive diagnosis of acidosis may be made based on a high prevalence of scouring, lameness, low or falling milk fat content, and rations high in NSC and low in effective fibre. Veterinarians need to be thoroughly aware of this condition in Australasian dairy herds. Methods of prevention and treatment are outlined in this document for dairy and beef cattle and sheep.

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Appendix

It is important to consider the regulatory status of veterinary medicines when selecting a rumen modifier.

To find out the regulatory current status of a veterinary medicine or to view product labels, veterinarians and other animal health advisors should consult the approved product database at www.apvma.gov.au.

Any use of a veterinary medicine that is not consistent with labelled directions is considered off-label use. An appropriate approach to off-label use that has been developed by the AVA is summarised in the following table:

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extract from:

Australian Veterinary Association Guidelines for prescribing, authorising and dispensing veterinary medicines

Version 1 February 2005

'Off-label' use

'Off-label' prescribing is writing a prescription or authorisation to a client to allow them to use a registered drug or veterinary chemical in a manner outside the range of uses permitted by the approved label directions - including species of animal, dosage, treatment interval etc. (but not contrary to a specific label restraint - see section 22.6).

Veterinarians are permitted to exercise professional judgement in the 'off-label' use or supply of most drugs or other veterinary medicines. This gives veterinarians access to beneficial drugs which may be registered for human use or which have limited registration for veterinary use. However, veterinarians must be aware that access to such drugs is the subject of concern in the community, and that misuse of such drugs may lead to withdrawal of this authority.

The veterinarian assumes full responsibility for the use of any drug contrary to the drug's registered use pattern as reflected on the manufacturer's label. If using drugs in any manner outside the range of uses permitted by the manufacturer's label or product insert, it is essential to inform the client of this, the reasons for the choice of drug, any other options available to the client and to document the informed consent of the client in the clinical records.

Use of unregistered chemicals or human medicines in food-producing animals should be limited to those cases where appropriate veterinary drugs do not exist or where they are known, or can reasonably be anticipated to be, ineffective. Legislation in most jurisdictions restricts such treatment or supply to single animals only – Consult the relevant legislation for the local definition of 'single animal'. It is unacceptable to use a human medicine for common disease conditions in food-producing animals where approved veterinary drugs e.g., antibacterials, anti-inflammatory agents etc, are available. A veterinarian assumes full responsibility when an unregistered chemical or human medicine is used rather than a registered veterinary medicine.

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'Off-label' use in food-producing animals should only be considered when:

- a careful diagnosis and evaluation of the condition for which the drug is to be used has been made;

- the veterinarian is operating within the bounds of a valid veterinarian-client relationship;

- a deliberate determination is made that there is no other appropriate veterinary drug available, ie. there is no marketed veterinary drug specifically labelled for the disease condition to be treated or the veterinary drug has been found clinically (or in laboratory tests) to be ineffective by the veterinarian in the animals to be treated;

- in the case of food-producing animals, adequate steps to prevent the occurrence of illegal residues in edible animal products have been taken. This should include a review of the best available toxicological and tissue distribution and tissue residue depletion data and establishment of an appropriately long withholding period, to ensure that no detectable residues will occur. The animal owner or manager should be given explicit written withholding period instructions, and the veterinarian should be very confident that these instructions will be faithfully followed. Where a long withholding period is provided, to ensure no residues will remain, this period should also be satisfactory as an export slaughter interval.

- the drug has been approved for use in at least one major food-producing species (for other than single animals).

Important! Some form of regulatory action may still be considered by state/territory authorities when an illegal residue occurs even if a veterinarian has followed these precautions. As indicated above, when unregistered (veterinary) chemicals are supplied, or registered chemicals are used 'off-label', the veterinarian is legally responsible if the withholding period specified on the label supplied by the veterinarian proves to be inadequate.