

**ACID-BASE BALANCE
IN THE RUMINANT
Economic Importance to the Dairy Producer**

By

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Ruminants are naturally grass consumers. Ruminant animals and rumen microorganisms have a symbiotic relationship that facilitates fiber digestion, but domestic ruminants in developed and developing countries are often fed an abundance of grain and little fiber. This is the result of breeding genetic potential for extremely high and efficient ruminant products (meat, milk, wool) and animal nutritionists and veterinarians trying to have this potential fulfilled with diets that are energy and protein rich. In many instances these diets become marginally deficient in the fibrous feeds needed to maintain the rumen ecosystem in optimal performance. When ruminants are fed fiber-deficient rations, physiological mechanisms of homeostasis are disrupted, ruminal pH declines, microbial ecology is altered, and the animal becomes more susceptible to metabolic disorders and, in some cases, infectious disease. Some disorders can be counteracted by feed additives (for example, antibiotics and buffers). These additives can alter the composition of the ruminal ecosystem in an attempt to restore rumen function.

The energy provided by forage crops is insufficient to challenge dairy animals to reach their genetic potential for milk production. Reaching that potential requires the addition of feedstuffs high in energy; normally the substitution of cereal grains for part of the forage.

Cattle challenged to produce to their potential through "hotter" rations, however, walk a tightrope between maximum production and digestive upset. Even with proper cattle management, high concentrate-low roughage diets may result in metabolic changes in the cow that impair productivity and efficiency, bringing an economic loss to the producer.

Many digestive disturbances have been tied to the delicate balance between acids and bases and their complex function in the rumen. The objectives of this review are as follows:

1. Outline the vital workings of the healthy ruminant.
2. Describe the digestive problems due to the greater emphasis on high milk production.
3. Discuss the signs of the acid-base imbalance.
4. Discuss the impact of acid-base imbalances on nutrient availability to the cow.
5. Examine the role of buffers in dairy cattle nutrition.

I. WORKINGS OF THE NORMAL RUMINANT

A. Anatomy and physiology. The ruminant stomach is divided into four compartments: the rumen, reticulum, omasum and abomasum. The rumen and the reticulum are joined by a fold of tissue (the reticulo-rumen fold) that allows feed to flow freely from one to the other. Most microbial activity during digestion takes place in the reticulo-rumen area.

Hastily eaten food is first swallowed into the reticulo-rumen where it is drenched with rumen fluids. It is then regurgitated back into the mouth as a bolus and thoroughly chewed and saturated with saliva. The diet is fermented in the reticulo-rumen and then the liquid phase is passed to the abomasum. (The role of the omasum in the digestive process has not been clearly defined.) When large feed particles are ruminated, surface area and fermentation rate are both increased. Rumination also triggers saliva flow, which maintains a favorable pH for the microbes and the animal. Muscular contractions mix fresh feed with microorganisms and wash the epithelium with fermentation fluids so the microbial short-chain organic acids can be absorbed. Specialized contractions hold feed materials away from the esophagus so that fermentation gases can be expelled by a process known as eructation. Rumen physiology is largely dictated by the presence of fibrous materials in the rumen and the pharynx. If ruminants are fed fiber-deficient diets, then mixing motions, eructation, rumination, and saliva flow decrease; fermentation acids accumulate; and ruminal pH declines (*Allen, 1997; Hungate, 1966*).

Chemical digestion of dietary components and rumen microflora occurs in the abomasum, the only compartment which has glands in its walls for secretion of gastric juices. The abomasum often is referred to as the true stomach.

Development of the stomach after birth relates to the size and/or age of the animal and the diet. A liquid diet, for example, delays development of tissue thickness, weight and papillary surface of the reticulo-rumen.

On the other hand, rapid growth of the reticulo-rumen results once the young animal starts to eat solid feed. Roughage stimulates growth as indicated by an increase in tissue thickness. But the presence of fermentable materials which yield the volatile fatty acids (VFA) appear to be necessary for the growth of papillae, finger-like projections in the rumen that aid in nutrient absorption. Stomach maturity is reached at approximately five to six months of age in cattle.

Ruminants, unlike simple-stomach animals, have the ability to digest large quantities of roughage. Feedstuffs consumed by ruminants are altered by chemical and microbiological action in the reticulo-rumen before final chemical digestion takes place in the fourth stomach compartment, the abomasum. Carbohydrates, for example, are fermented or broken down into volatile fatty acids, the most important being acetic, propionic, and butyric.

The volatile fatty acids are produced in addition to other acids and/or their salts, which naturally occur in the rumen: carbonic, lactic, sulfuric, and phosphoric. The potential for the production of excess acid in the rumen naturally exists. Thus, the importance of maintaining a proper alkaline reserve in the rumen.

Ruminants are fed a variety of additives to alter fermentation, and these supplements are widely used (*Weimer, 1998*). Heat-treated proteins decrease ruminal deamination and provide an additional source of amino acids; rapidly fermented grain supplements increase energy availability and allow for more efficient use of ruminally available nitrogen by the microbes; buffers counteract grain-dependent declines in ruminal

pH; ionophores (e.g., monensin) inhibit Gram-positive bacteria that produce hydrogen, ammonia, or lactic acid; and other antibiotics inhibit pathogenic bacteria in the gut and animal tissues.

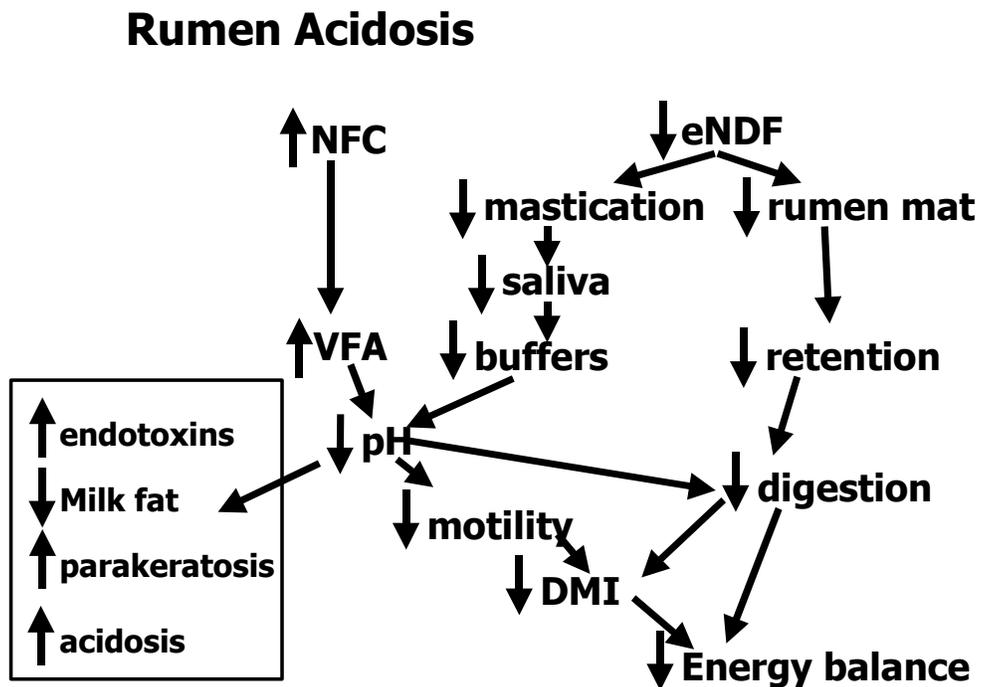
Dairy cattle capable of high milk production are often supplemented with 50 to 60% grain. When cattle are switched abruptly from forage to grain, the rumen can become severely acidic (ruminal pH, 5.5). This can be typical in managerial situations where high forage based diets are fed prepartum and rapidly

switched to a high grain production diet at, or soon after, calving. This acute acidosis is caused by the overgrowth of starch-fermenting, lactate-producing bacteria (*S. bovis* and *Lactobacillus* spp.) (Owens *et.al.*, 1998). If the dietary shift is gradual, *M. elsdenii* and *Sel. ruminantium* can convert lactic acid to acetate and propionate, the ruminal pH is not as severely affected (Owens *et.al.*, 1998), and the ruminal ecology is not so drastically altered (Tajima, 2000), however, it is, none the less, altered. Even though the rumen microbial population adapts more favorably when diets are gradually changed from high forage-based diets to higher grain contents, high concentrations of volatile fatty acids can cause subacute ruminal acidosis (Owens *et.al.*, 1998), and pH-sensitive ruminal bacteria (e.g., cellulolytics) are inhibited if the ruminal pH is 6.0 (Russell and Wilson, 1996).

The ruminal epithelium is not protected by mucous, thus even brief periods of subacute acidosis can cause inflammation, ulceration, and scarring (Owens *et.al.*, 1998). Lactate accumulation promotes the growth of *Fusobacterium necrophorum*, a lactate utilizing bacterium that infects ruminal ulcers (Nagaraja and Chengappa, 1998). If *Fus. necrophorum* passes from the rumen and colonizes the liver, abscesses develop. *Fus. necrophorum* also produces leukotoxin and endotoxic lipopolysaccharide (Nagaraja and Chengappa, 1998). If pH is chronically acidic, the epithelium releases metalloproteinases that cause tissue degradation (Rowe, 1999). If these metalloproteinases enter the bloodstream, the laminae above the hoof become inflamed, the animal becomes lame, and, in extreme cases, the hoof can fall off.

Figure 1 shows the results of feeding diets with high non-forage carbohydrates (NFC) and/or low contents of effective neutral detergent fiber (eNDF). Through slightly different pathways, both situations lead to reduced dry matter intake (DMI) and energy balance that can severely hamper productivity and compromise animal health.

FIGURE 1. The development of rumen acidosis via high non-fiber carbohydrate (NFC) or low effective neutral detergent fiber (eNDF) diets.



B. Natural buffering in the rumen. Cattle and sheep secrete large volumes of saliva in the normal course of eating and ruminating. From information on hand, it has been estimated a 650 kg cow, consuming hay and grain, will produce 280 liters of saliva in a 24-hour period. Components of this saliva include 2982 gm of NaHCO_3 (sodium bicarbonate), 1028 gm of NaHPO_4 (sodium biphosphate), as well as salt, urea and other nitrogen compounds.

Ruminant saliva has the following functions:

1. Maintains buffering capacity in the rumen.
 - ✓ A buffer is the salt of a weak acid and, when in equilibrium with the weak acid, it will resist changes in the pH. The term pH is a relative measure of acidity or alkalinity of a solution measured on a scale from 0 (acid) to 14 (alkaline). A pH of 7 indicates neutrality. The lower the pH value, the more acid the environment.
2. Aids mastication and swallowing.
3. Promotes activity of enzymes.
 - ✓ Enzymes are proteins that cause chemical breakdown of other substances.
4. Furnishes nutrients for rumen microorganisms.
5. Contains anti-foaming properties.

The large quantity of bicarbonates secreted in saliva helps buffer the rumen against pH changes caused by the acids produced during fermentation.

Mucin, urea, phosphorus, magnesium, and chloride are also present in relatively high concentrations in saliva. They supply readily available nutrients to rumen microorganisms. Anti-frothing properties of saliva are considered to be important in combating bloat.

Researchers have noted regular variation in rumen acidity depending on the type of diet and the amount of time that elapses between eating and measurement of the rumen pH. Changes in rumen pH reflect changes in the quantities of organic acids that accumulate and the amount of saliva that is produced. Rumen acidity will generally reach a high from two to six hours after feeding, depending on the nature of the diet and how rapidly a meal is eaten. A recent theory that has been proposed is that this cycling of rumen pH between higher and lower values is required for optimal microbial protein synthesis. However, if the nadir of pH is too low, subacute rumen acidosis develops. (*Krajcarski-Hunt et al., 2002; Dewhurst et al., 2001*)

The buffering capacity of rumen contents depends on relative concentrations of bicarbonate, phosphate and volatile fatty acids. The large amounts of organic acids produced in the rumen by microorganisms breaking down feedstuffs have been calculated to yield pH values of 2.78 to 3.03. This suggests that the rumen has considerable natural buffering capacity within the normal pH range of the rumen (5.0 to 7.0), bicarbonate and phosphate being important buffering components.

Sodium bicarbonate is the primary buffering system in cattle. Under natural grazing conditions, cattle secrete over 2 kg of bicarbonate daily in their saliva. This aids in the maintenance of the acid-base balance in the rumen. Buffering capacity of the rumen depends on the amount of saliva produced and the amount of buffers in the saliva. Saliva production increases when an animal chews its cud, or ruminates. Total saliva

production decreases as the animal reduces the time spent chewing or ruminating, which is related to the effective NDF content of the diet.

C. Microbiology of the rumen. The reticulo-rumen hosts a large and varied population of microorganisms, mainly bacteria and protozoa. These organisms, along with saliva and the feedstuffs eaten, are the most important factors affecting the environment of the rumen. Absorption and passage rate of ingesta out of a reticulo-rumen to the true stomach also are important factors.

Optimal conditions for microbial growth in the healthy rumen are:

- (a) Rumen pH, under natural, pasture conditions, normally ranges from 6.0 to 6.9.
- (b) Temperature of 38 to 41 degrees C is near optimum for the action of most digestive enzymes.
- (c) Food for the microflora is provided more or less continuously.
- (d) Stomach contractions help to bring the microorganisms into contact with freshly eaten or ruminated feed.
- (e) Moist environment.
- (f) End products of fermentation are removed by absorption into the bloodstream with the remainder passing into the stomach.

It has been suggested that the microbial mass, which primarily consists of bacteria and protozoa, may comprise 10 percent of the rumen fluid. Protozoal species found in the rumen of domestic ruminants are known to vary according to diet, season of the year and by geographical location.

In contrast to the situation with ruminant bacteria, establishment of protozoa populations in the young ruminant requires contact with older animals with a protozoal population. The protozoa are primarily responsible for the digestion of more fibrous feedstuffs.

Bacterial organisms that have been identified in the rumen include the following:

- (a) Carbohydrate-digesting bacteria.
- (b) Sugar-utilizing bacteria.
- (c) Acid-utilizing bacteria (lactic acid, succinic acid, formic acid, acetic acid, oxalic acid, etc.).
- (d) Protein-utilizing bacteria.
- (e) Ammonia-utilizing bacteria.
- (f) Methane-producing bacteria.
- (g) Fat-digesting bacteria.
- (h) Vitamin-synthesizing bacteria.

Many factors affect the rumen microbial population, both in quantity and quality. Microbial growth rates at a particular time will depend on the adaptation of an organism to its environment and to the nature and concentration of available nutrients. The nutrients required by some organisms may not be present in the diet but are end products of fermentation by other organisms - examples being water-soluble vitamins, and volatile fatty acids (VFA). Other variables include such things as the rapidity of eating, selectivity when grazing, soil fertility, geographical location and climate.

Rumen pH is a critical factor and one which closely reflects the diet. For example, protozoal population in adult sheep was greatly depressed by a pH of 5.4 in the rumen. A depressed pH, due to feeding grain, resulted in lowering protozoal population. The effect of lower pH in adult animals can be rather drastic, resulting in a rapid growth of streptococcus and lactobacillus, both lactic acid-producing organisms.

Overproduction of unfavorable organisms causes an imbalanced rumen population, resulting in an accumulation of end products different from those produced by the normal population.

II

HOW ACID-BASE IMBALANCE OCCURS

A. The chain of events. In the effort to increase milk production, the dairy industry has sought to maximize energy intake by feeding rations rich in highly fermentable carbohydrates. Feeding these rations, which often contain high levels of cereal grains or high-moisture silage, to the reduction or exclusion of roughages, may cause the following chain of events to occur:

- ✓ The ruminant eats large amounts of readily fermentable, grain-type feed.
- ✓ Production of volatile fatty acids in the rumen is more rapid than normal.
- ✓ Rapid fermentation of the feed enables the animal to spend less time ruminating.
- ✓ With decreased rumination, the salivary glands receive less stimulation and less saliva is produced.
- ✓ The lower production of saliva reduces the ruminant's natural buffers, mainly bicarbonates and phosphates.
- ✓ Acid-base imbalance occurs in the rumen (low pH) and digestive upset results.
- ✓ Changes in the normal microbial population occur, cellulose-digesting bacteria are greatly reduced, protozoa disappear, and streptococcus and lactobacillus increase.

B. Recognizing acid-base imbalance. Outward signs of acid-base imbalance differ depending on the magnitude of ruminal acidity (pH level), the resulting local and absorptive changes in the digestive system, and the stage of the problem. Mild imbalance shows a temporary reduction in feed intake or loss of appetite, suppressed rumen contractions, with a pasty to soupy consistency of the feces with a gray-green color, later followed by dry, firm, gray-green feces. Both types of fecal material may be apparent at the same time in a pen and this is indicative of continuing digestive upset. In dairy cows, milk production drops and fat content may fall.

In situations where outward signs are not noticeable, the severity depends on the ability of the rumen microorganisms to rapidly adapt to concentrated feed, or the time lag in correcting the composition of the ration. High heat conditions or exercise can cause a normally functioning ration to cause problems due to the increased demands made on the animal's bicarbonate reserves. But if the ration is corrected in time, appetite usually reaches normal within a few days. Milk production and fat content will not, however, reach their original level for two to three weeks.

Severely affected animals show obvious indigestion with marked intoxication within 8 to 24 hours after overfeeding. The animal may show signs of appetite loss, indigestion and irritability progressing to dullness. The animal will generally stand with its head down and slobber, obviously in discomfort, the rumen may bloat, the back may be arched to relieve the discomfort.

Progressive signs include:

- ✓ Cessation of rumen contractions.
- ✓ Increased fluid contents in digestive tract.
- ✓ Increased pulse and respiration rates.
- ✓ Variable rectal temperature.

- ✓ Sunken eyes.
- ✓ Loss of skin elasticity.
- ✓ Incoordination.
- ✓ Collapse and coma.

Early in the syndrome, the animals develop a profuse diarrhea. After the diarrhea passes, the droppings may have a grayish color, show slimy streaks through it and be quite dry. In warm dry weather, undisturbed dry fecal material may show dried white salts on its surface. Death may ensue in one to several days. In severely acute hyperacidity, animals may "go down" and become comatose within 12 to 24 hours. Circulation is severely affected (pulse rate 100 to 140 per minute). The body temperature, initially normal or slightly raised, falls to subnormal. The animals may die within a short time. This is more common in beef feedlot situations than in dairy herds.

Animals that survive the acute phase but develop severe inflammation of the rumen wall, remain "off feed," become weak and emaciated and may die within two weeks. Other signs include apathy, muscular tremors, groaning and grinding of the teeth, and a reduction in urinary volume. Surviving animals may later show lameness that is attributable to laminitis. Examination reveals an increased heart rate (93-120 per minute) and usually an increased respiratory rate, lack of rumen movement, and an elevated rectal temperature (up to 41-43°C). Generally, when a stomach tube is passed into the rumen, a large volume of watery fluid will pass out. The fluid is quite acid with a characteristic smell. The animals show an unsteady, staggering gait.

Not all cattle in a pen may show all the signs at the same time. Some cattle may be panting, some with diarrhea, and some with dry fecal material with epithelial lining present.

C. Chemical and physical changes in the rumen. Several chemical and physical changes take place in a rumen that is subjected to an acid-base imbalance. In addition to the changes in microbial population, a rapid fall in the pH of the ruminal content occurs. The lowering of the pH is initially due to an increased production of lactic acid and the volatile fatty acids. A fall in pH from 7 to 5 is accompanied by a fall in the molar percentage of acetic acid from about 70 to 30 and a rise in the molar percentage of butyric acid from about 10 to 45. The molar ratio of propionic acid remains rather constant to pH 5.5, below which it rapidly rises due to conversion of lactic acid into propionic acid and acetic acid.

Below pH 5, the activity of the rumen changes to predominantly lactic acid formation, and the concentration of lactic acid rises. Within 24 hours after overeating, VFA's disappear from the rumen and are replaced by lactic acid. High lactic acid concentration of the rumen contents cause a rise in the level of lactic acid in the blood, resulting in an accelerated drop in the ruminal and intestinal pH. Due to the attendant high osmotic pressure, the rumen contents draw water from the body, upsetting fluid balance.

In dairy herds afflicted with subacute rumen acidosis, rumen pH seldom drops below 5.5 and the primary contributor to the reduced pH is the volatile fatty acids. Lactic acidosis is seldom a problem in dairy herds. Lactate does not usually accumulate in the rumen until pH drops below 5.5.

The first physical effect of acid-base imbalance is damage to the absorptive surface of the rumen (epithelium) and other parts of the gastro-intestinal tract. Epithelium damage is characterized by the formation of small fluid-filled sacs, loss of sulfur-rich protein, invasion by white blood corpuscles, loss of tissue surface layers, death of epithelial cells, and small hemorrhages. In later stages, damage and even ulceration may occur in the rumen, omasum, abomasum and small intestine.

Toxic substances (histamines) have been isolated in the rumen contents of sheep and cows suffering from hyperacidity (high acid concentration). Histamines block the reabsorption of water from ingesta in the lower gastro-intestinal tract, causing the diarrhea often associated with acid-base imbalance. These substances also depress blood pressure and rumen contractions. The high acid concentration (low pH) causes a reduction in rumen contractions, which reduces the movement of materials out of the rumen; however, the large movement of water into the digestive track will cause the observed diarrhea. While movement in the rumen is becoming impaired, the absorption of lactic acid proceeds. Large increases in lactate concentration of the blood are observed and the bicarbonate level and pH of the blood falls.

Lactic acid is highly corrosive to the epithelium. Papillae, finger-like projections that form the absorptive surface of the rumen, may clump together or fall off. This may occur in both mild and severe cases of acid-base imbalance, leaving scar tissue and permanently impairing nutrient absorptive capacity. The maximum effect is reached between 24 and 36 hours after initiation and is followed by a return toward normal values.

When acid production in the rumen resulting from fermentation of organic matter exceeds the capacity for the animal to absorb these acids or be neutralized, ruminal pH falls. This acidosis, even at the subclinical level, will cause a reduction in cellulolytic bacteria in the rumen and contribute to an overall reduction in feed digestibility. Furthermore, the acidosis reduces rumen motility and efficacy of mixing rumen contents, which reduces the amounts of VFA near the rumen wall. Consequently, VFA absorption is reduced (*Allen, 1997*). As rumen motility and mixing decline so does rumination, which reduces the amount of saliva flowing into the rumen. The secretion of buffers in the saliva is equivalent to more than 3 kg of sodium bicarbonate and 1 kg of disodium phosphate (*Erdman, 1988*).

When NFC present in the rumen increases abruptly and rapidly, the production of VFA increases proportionately (*Owens et al., 1998*). If these VFA are not absorbed rapidly they will accumulate in the rumen (*Schultz et al., 1993*). Different problems are caused by rumen acidosis depending on the quantity of acids that accumulate in the rumen. When there is a mild accumulation, DMI and production can decline in spite of the cow appearing to be in good health (*Owens et al., 1998*). Also, the milk produced could have reduced fat. Certain species of bacteria and protozoa in the rumen are extremely sensitive to acidosis and release endotoxins and histamine. These substances can contribute to laminitis (*Nocek, 1997*). As the acid accumulation continues more severe problems can occur with DMI and production and also with overall health (*Schultz et al., 1993*).

It appears that this subclinical rumen acidosis is insidious and omnipresent in transition dairy cows as well as cows in early lactation. It is beyond the scope of this presentation to adequately describe this subject area, however, there are numerous publications dealing with its description and detection (*Yokoyama and Johnson, 1988; Nordlund, 1994; Allen and Beede, 1996; Hall, 1999*).

III

MANAGEMENT OF ACID-BASE IMBALANCE

Management of acid-base imbalance in the rumen can be divided into two separate and distinct segments:

1. Cultural prevention of the imbalance through proper nutrition and feeding management, and
2. Veterinary treatment of the acid-base imbalance when the imbalance results in danger to the well-being of the animal.

- A. Nutritional (cultural) management.** Nutritionally-induced acid-base imbalance may be prevented, or minimized, by gradual adaptation of the ruminant to ration changes. In mild cases of acid-base imbalance, an increase in the percentage of crude fiber in the ration (for instance, by addition of eNDF) may suffice to reestablish normal rumen digestion.
- B. Veterinary Treatment.** In severe cases of acid-base imbalance, a veterinarian should be consulted

In the case of dairy cows, rations should be changed slowly, making sure adequate fiber levels. Concentrate levels should be changed slowly with special attention given to cows entering the milking string.

Buffers boost milk yields

A summary of 24 research studies (2,087 cows) from 1975 to 1985 found an increase of 1.1 kg of 3.5% fat corrected milk in buffer treated cows. There was an economic response of \$2.30 for each dollar (U.S.) invested. Milk yield responses summarized from controlled research studies vary (see Table 1), depending on the type of buffer, level of forage and forage type.

A New York DHI survey of 5,700 producers found 67.6% were feeding a buffer; of that number 76.9% were continuous users. A Mid-States DHI field survey of 2,684 dairy farmers found 54.5% used buffers, producing 570 Kg more milk per cow annually, than herds not using buffers.

TABLE 1. Sodium Bicarbonate Studies - 1975-1985

Parameter	No.	Control	Bicarb	Difference	% Increase
3.5% FMC, Kg	33	28.3	29.3	+1.0	+3.5
Milk, Kg.	33	28.7	29.2*	+0.5	
Milk Fat, %	33	3.43	3.51*	+0.08	+2.3
DM Intake, Kg	25	18.2	18.6*	+0.4	+2.0
FE (FCM/DMI)**	25	1.62	1.65*	+0.03	+2.0

*All values differed significantly from control (p<.01).

**Feed efficiency equals fat corrected milk divided by dry matter intake.

Product components

Buffers are a combination of a weak acid and its salt, which resist change in hydrogen ion concentration (pH). Alkalinizing agents increase the rumen pH. Buffers increase rumen pH and osmolarity, maintain a favorable rumen fermentation and volatile fatty acid pattern and increase rumen fluid outflow.

Sodium bicarbonate is the primary buffer product and has a broad research base. (Table 1). Sodium sesquicarbonate acts similarly to sodium bicarbonate in the rumen although there is less research data available on this product (Table 2). Sodium carbonate (found in sodium sesquicarbonate as an integral part of the molecule) is a strong alkalinizing agent and does not buffer but raises pH. Sodium carbonate is very difficult and a bit dangerous to use. Its very high pH can be destructive to mucous membranes and the dusty

nature of the product makes it rather unsuitable for the animal, the person mixing the feed and the equipment due to its corrosive nature.

Research results comparing sodium sesquicarbonate to sodium bicarbonate (positive control) generally conclude both products are equally effective compared to negative control diets (no buffer). Sodium bicarbonate and sodium sesquicarbonate can be fed at comparable amounts (1.5 to 2.0% of the grain mixture or .75 to 1.0% of the total ration dry matter).

Magnesium oxide is an alkalinizing agent and a source of magnesium (54%). It can influence rumen fermentation and increase milk fat precursors in the mammary gland. Magnesium oxide particle size affects reactivity and effectiveness.

Magnesium oxide may be fed with sodium bicarbonate or sodium sesquicarbonate in a ratio of 3 parts sodium bicarbonate or sesquicarbonate to 1 part of magnesium oxide.

Other materials such as dolomitic limestone or bentonite are not effective buffers in the rumen.

Table 2. NATURAL SODIUM SESQUICARBONATE (NSC)

RESEARCH

Location: Oklahoma State University and University of Illinois

Design: Full Lactation Trials

NSC fed at 1% of total ration dry matter for 170-180 days and 0.5% thereafter

Performance Data (170 days)

	Univ. of Ill.		Okla. State Univ.	
	Control	NSC	Control	NSC
No. of Cows	24	24	25	25
Intake (Kg/day)	22.4	21.9	21.1	22.6
Milk (kg/day)	38.2	38.1	37.8	38.8
Fat (%)	3.41	3.57	3.27	3.42
Protein (%)	2.97	2.94	3.01	3.08
4% FCM (Kg/day)	34.9	35.5	33.7	35.4

Combined

Control	NSC	Diff. (Kg.)	NSC
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No. of Cows	49	49	0	0
Intake (Kg)	21.8	22.3	+0.5	+2.3
Milk (kg)	38.0	38.5	+0.5	+1.2
Fat (%)	3.34	3.50	+0.16	+4.8
Protein (%)	2.99	3.01	+0.02	+0.7
4% FCM (Kg)	34.3	35.5	+1.2	+3.5

Table 3. RESPONSE TO SODIUM BICARBONATE AND MAGNESIUM OXIDE IN EARLY LACTATION

Parameter	TREATMENTS			
	Control	1.5% NaHCO₃	.8% MgO	1.5% NaH₂PO₄ +.8% Mg
Milk, Kg/day	34.4	36.0	34.8	38.2
Fat, %	3.80	3.96	3.62	4.05
4% FCM, Kg/day	32.4	35.0	32.2	38.0
Feed Intake, Kg/day	18.5	20.6	19.1	20.5

From Erdman et al., 1980.

The role of buffers

Veterinarians traditionally recommend buffers to prevent, treat or as insurance against rumen imbalances.

□ When using the preventative approach, the level of sodium bicarbonate or its equivalent is 180-250 g per cow per day (1.5 to 2.0% of the grain mixture or .75 to 1.0% of the total ration dry matter), depending on ration components, feed processing and the feeding system (Table 4).

□ The treatment approach consists of administering 400-600 g of buffer to a cow that has gone off feed, is scouring or sick. An example would be treatment for the accidental grain over-load of a cow that has access to free choice grain. Drenching would be required to administer this high level treatment.

□ In an insurance role, buffers are used to minimize the potential for low rumen pH, due to daily cow variation, weather, changes in eating patterns or other management factors that could drop the rumen pH

below 6 (Figure 2). If the rumen pH drops below 6 (one definition of subacute rumen acidosis) for 1-6 hours, dry matter intake will decrease - affecting production. The insurance level is 1.5 to 2.0% of the grain mixture or .75 to 1.0% of the total ration dry matter.

Research on free choice feeding has had varied results. Minnesota researchers found cows consumed less than 45 g of buffer per day (in a 19-month study) when offered in a cafeteria-style program. Some cows would not eat the buffer if used as a top dressing.

Some high producing, early lactation cows ate significant amounts (135-225 g) when the buffer was offered free choice. Consumption increased and decreased depending on the ration and weather changes. If the buffer is being fed free choice, the practitioner should monitor intake and avoid buffer packs high in palatable ingredients: molasses, distillers grain or alfalfa meal - to assess adequate and excess intake.

The best practice is to provide the proper level of buffer in the grain mix or total mixed ration (TMR) and then provide buffer free choice as an indicator of ration adequacy.

Table 4 provides guidelines developed by Dr. Hutjens as to conditions that require buffer feeding. Buffer feeding should be routine part of good dairy management. All cows need supplemental buffers in early lactation, and depending on those factors identified by Dr. Hutjens, most cows may need buffers for the entire lactation.

Dry cows are a special case and buffers should not be included in dry cow rations.

Table 4. Feeding factors that would warrant buffer supplementation in a preventive role (*Hutjens, 1988*)

Factor	Measurement
High corn silage diets	Over 50 percent of the forage dry matter
Wet rations with fermented feeds	Over 50 percent moisture in the total ration
Low fiber rations	Under 19 percent acid detergent fiber (ADF)
Limited hay	Less than 2-5 Kg of long hay
Short haylage	Theoretical chop less than 1.5 cm or particles less 4 cm inch long (over 75 percent)
High concentrate rations	Over 55 percent concentration in the total ration dry matter
Slug feeding of concentrate	Over 3 Kg of concentrate dry matter per meal
Finely ground concentrate	Pelleted or powdered (flour-like) concentrate
Wet concentrate	High moisture grain over 30 percent
Highly fermentable concentrate	Crude fiber below 5 percent
Heat stress	Over 27C (wet bulb)

Depressed feed intake	Decline of dry matter intake (over 5 percent of normal)
Low fat test	Cows one full point below herd average (3.8 vs 2.8) or milk fat: milk protein inversions (milk protein tests higher than fat tests)

Other Nutritional considerations during subacute acidosis.

Diet formulation for dairy cattle relies heavily upon the assumption that the rumen is functioning properly. Energy, protein, bypass nutrients and vitamins that we assume that the cow is receiving all depends upon these fermentative processes in the rumen. It is obvious from the discussion above that when a cow has subacute rumen acidosis it is unlikely that any formulated diet will achieve the health and productive objectives originally desired and expected from a particular diet. Even the best nutritionist cannot formulate a diet to account for the problems of acidosis. Any attempt to do so (increasing bypass nutrients and or grains, reducing forage, etc.) will further aggravate the problem and cause acute acidosis.

The only manner to formulate for production and health is to avoid subacute rumen acidosis by formulating diets to assure a healthy rumen. Be certain that minimum fiber levels are respected; monitor forage particle size; avoid drastic dietary changes and forage source changes; use a buffer routinely to assure against dietary or environmental changes that can cause the onset of subacute rumen acidosis.

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- Allen, M. S. *J. Dairy Sci.* **80**, 1447 (1997).
- Allen, M.S. and D.K. Beede. in *Tri-State Dairy Nutrition Conference*. M.L. Eastridge, ed. (1996)
- Dewhurst, R. J. et al., *J. Dairy Sci.* **84**:2721 (2001).
- Erdman, R. J. *J. Dairy Sci.*, 71:3246 (1988)
- Hall, M.B. in *Proc. 10th Annual Florida Ruminant Nutrition Conference*. University of Florida, Gainesville, FL. (1999).
- Hungate, R. E. *The Rumen and Its Microbes* (Academic Press, New York, ed. 3, 1966).
- Hutjens, Michael F. *Proc. Pacific Northwest Nutrition Conference*, pp. 189-195 (1990).
- Krajcarski-Hunt, et al., *J. Dairy Sci.* **85**, 570 (2002).
- Nagaraja, T. G., M. M. Chengappa, *J. Anim. Sci.* **76**, 287 (1998).
- Nocek, J. E. *J. Dairy Sci.* 80:1005–1028 (1997)
- Nocek, J., J. B. Russell, *J. Dairy Sci.* **71**, 2070 (1988).
- Nordlund, K. in *4-State Applied Nutrition and Management Conference*, LaCrosse, WI (1994).
- Owens F.N., D.S. Secrist, W.J. Hille D.R. Gill. *J. Anim. Sci.* 76 : 275-286 (1998).
- Rowe, J. B. *Recent Advances in Animal Nutrition in Australia*, J. L. Corbett, Ed. (Univ. of New England Press, Armindale, Australia, 1999), pp. 81–89.
- Russell, J. B., D. B. Wilson, *J. Dairy Sci.* **79**, 1503 (1996)
- Schultz, L.H., L. Allenstein et G. Oetzel. *Fresh cow problems : how to control them*. W.D. Hoard & Sons Company. Fort Atkinson, WI (1993).
- Tajima, K. *et al.*, *Anaerobe* **6**, 273 (2000).
- Weimer, P. J. *J. Anim. Sci.* **76**, 3114 (1998).
- Yokoyama, M.T., et K.A. Johnson.. in *The ruminant animal: Digestive physiology and nutrition*, page 125. D.C. Church ed. Prentice-Hall, Englewood Cliffs, NJ. (1988)