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Ruminant Digestion

D P Visser
Department of Agriculture: Livestock Improvement Schemes

INTRODUCTION

Ruminant species occupy an important niche in modern agriculture because of their unique ability to digest certain foodstuffs, especially roughages, efficiently. In future, the direct demands for grain by human beings will make efficient utilization of roughages increasingly important. A basic understanding of ruminant digestion is essential for good management and sound nutrition of beef cattle.

THE RUMINANT DIGESTIVE SYSTEM

The fact that the ruminant can convert roughages, unsuitable for man, into useful products is normally taken for granted. What enables them to achieve this? The ruminant has three preliminary compartments in its digestive tract before the true stomach, or abomasum. These are the reticulum, rumen, and omasum. The rumen, and reticulum are not completely separated, but have different functions.

Reticulum

The reticulum is a flask-shaped compartment with a "honeycomb" appearance. It moves ingested food (ingesta) into the rumen and the omasum. The reticulum also causes the regurgitation of ingesta during rumination, and acts as a collection compartment for foreign objects.

Rumen

The rumen is a large fermentation chamber (in adult cattle its volume is about 125 litres) which has a very high population of micro-organisms, mainly bacteria, but also protozoa.

It is because the bacteria secrete the enzymes necessary for cellulose degradation that ruminants are able to utilize roughage. The rumen has a textured surface, lined with projections (up to 1 cm long), termed rumen papillae. The rumen, along with the omasum, absorb the by-products of bacterial fermentation. These by-products are volatile fatty acids (VFAs).

Omasum

The omasum, or "manyplies", contains numerous laminae (tissue leaves) that help grind ingesta. These folds assist in the removal of fluid from the ingesta on their way to the abomasum.

Abomasum

This compartment corresponds to the stomach of the non-ruminant, and is termed the true stomach. It secretes the gastric juices which aid in digestion. The pH of the abomasum is normally in the range of 2,0 to 2,5. This low pH facilitates initial protein breakdown, and kills the bacteria which have spilled over from the rumen.

Ruminants differ from monogastric animals in the following important ways:

- They have no upper canine teeth, or incisors, and have long, thick and rough tongues.
- They ruminate. Chewing the cud helps reduce feed particle size, and mixes saliva into the feed.
- The ruminant digestive system includes a fermentation chamber, called the rumen. The rumen contains micro-organisms which serve some important functions: they make it possible for ruminants to digest fibre (especially those in roughages) and they synthesize nutrients (such as B complex vitamins), and also essential amino acids which become available to the animal when the micro-organisms die, and are digested.

The ruminant represents a classic example of symbiotic association between mammal and micro-organism.

STOMACH OF THE NEWBORN CALF

Calves, at birth, are not functional ruminants. At birth, the rumen is very small, and the fourth stomach (or abomasum) is by far the largest of the compartments. Digestion in the young calf is more like that of a simple-stomached animal than that of a ruminant, and it takes approximately three to four months before the calves can be considered actual ruminants.

RUMINANT DIGESTION

Digestion of carbohydrates

Plant tissues contain about 75 per cent carbohydrates of one kind or another, and provide the primary source of energy for both the ruminal organisms, and the host animal.

In ruminants the major part of all carbohydrates, including the complex carbohydrates such as cellulose and hemi-cellulose, is digested by bacterial action in the rumen.

During microbial digestion an appreciable amount of methane gas is produced. Approximately 6 to 7% of the food energy of the ruminant is lost as methane.

The main end-products of carbohydrate digestion are volatile fatty acids. Of these, acetic acid forms the major proportion, followed in declining order by propionic, butyric, and valeric acids. The VFAs are absorbed into the bloodstream through the rumen wall, and constitute 66 to 75% of the energy derived from the feed. Carbohydrates, such as sugars and starches, that escape

ruminal digestion are digested in the abomasum, and the end-products are absorbed through the small intestine.

Digestion of protein

Dietary protein, like dietary carbohydrates, is fermented by rumen microbes. The majority of true protein, and non-protein nitrogen (NPN), entering the rumen is broken down to ammonia, which bacteria require for synthesizing their own body protein. Ammonia is most efficiently incorporated into bacterial protein when the diet is rich in soluble carbohydrates, particularly starch. Ammonia, in excess of that used by the micro-organisms, is absorbed through the rumen wall into the blood, carried to the liver, and converted to urea, the greater part is excreted in the urine. Some urea is returned to the rumen *via* the saliva, and also directly through the rumen wall.

The undegraded true protein fraction, plus the microbial protein, passes from the rumen to the abomasum, where it is digested, and absorbed into the bloodstream through the walls of the small intestine.

DIGESTIVE DISORDERS

Since different substances are digested by different classes of bacteria, provision must be made to allow a new population of bacteria to establish itself when changes are made in the ration. Changing the composition of a ration, therefore, should be made gradually and it may take up to six weeks for the ruminal organisms to adapt to a change in diet.

A large number of digestive disorders can occur in ruminants. Only three of the more important disorders will be discussed:

BLOAT

Bloat, to which all ruminants are subject, can be of the frothy or the free gas type, and can be either acute or chronic. The term bloat is generally used to describe any condition caused by an excessive accumulation of gas in the rumen.

Frothy bloat (Pasture bloat)

Plant-induced bloat is usually of a frothy kind, the gas in the rumen being trapped in a stable foam which cannot be eructated by belching.

The daily production of gas, principally carbon dioxide, and methane, is about 800 litres in adult cattle. Frothy bloat occurs when the rate of foam formation is greater than the rate of foam breakdown, and this process is maintained for some time. The foam persists, and accumulates, causing an increase in the volume of ruminal digesta. This makes it difficult for the animal to clear digesta from the entrance of the oesophagus (the cardia). Clearance of the cardia is necessary for eructation (belching), but eructation is not stimulated, because the foam also induces a swallowing reflex. The characteristic abnormal distention of the rumen, a marked bulging of the left flank, results from the trapping of the gas in the foam.

Frothy bloat does not result from a simple mechanism, but the main factors to consider are the plant and the animal factors.

Plant factors

Some legumes (clover and lucerne, especially) have high bloat potentials, whereas other legumes very seldom cause bloat. The dangerous phase of forages for causing bloat is considered to be the young, fast-growing, leafy phase.

Animal factors

The susceptibility to bloat is believed to be inherited, and greedy feeders are more prone to bloat. The effects of saliva on bloat cannot be overlooked. The type of feed affects saliva flow. Roughage increases it and grain decreases it. Roughage also stimulates rumen motility, and belching efficiency.

Production losses due to bloat include:

- Losses due to death, and therefore a financial loss.
- Cost of protein supplements if legumes are not utilized.
- Expense of veterinary treatment, and labour.

Practical considerations to prevent bloat:

- Much can be done to prevent bloat, *e.g.* roughage, especially dry hay, should be fed before, or during, the grazing of lush, immature legume pasture. This will reduce the risk of bloat occurring. The dry roughage stimulates salivation, and sufficient saliva will help prevent bloat.
- Veterinary recommendations to inhibit foam formation, or gas formation, should be followed.
- Animals suffering consistently from bloat should be tested for TB, because TB can be the fundamental cause of recurrent bloat.

Treatment of bloat

An anti-foaming agent has to be introduced into the rumen to break the froth. Any suitable vegetable oil can be used for this purpose in an emergency, if the commercial pharmaceutical preparations containing recognized anti-foaming agents are not available.

Genuine turpentine, at a rate of 1 ml/kg body mass, administered by mouth, is a well-known treatment, and probably causes the froth to break.

Remember that bloat is due not so much to excessive gas production but rather to the failure to eructate.

Free gas bloat

Acute bloat, caused by free gas, results primarily when the normal gases of rumen fermentation are trapped due to oesophageal obstruction. The only way to relieve this condition is to remove the obstruction, or in the case of emergencies, to relieve the pressure by use of a trocar.

Metabolic disorders

Metabolic disorders such as urea poisoning, and acidosis, often cause bloat in cattle.

Urea Poisoning

Urea, and other NPN compounds, are very useful compounds, especially in winter licks. However, when improperly fed and formulated, urea can be deadly poisonous.

The toxicity of urea, and other NPN compounds, is caused by excessive levels of ammonia in the blood. Urea reaching the rumen is rapidly converted to ammonia. When large amounts of urea are consumed over short periods, the microbes cannot utilize the ammonia for synthesis of microbial protein as rapidly as it is produced. The quantity of ammonia (NH_3) absorbed by the ruminant is greatly influenced by both the concentration of ammonia in the rumen fluid, and the pH of the rumen fluid.

Absorption is more rapid when the rumen fluid is alkaline (pH>7). Rumen fluid is not as well buffered against an increase in pH as against a decrease. Since the chemical structure of ammonia is alkaline, increasing concentrations of ammonia elevates the pH, and increases its absorption across the rumen wall. Absorbed ammonia is converted to urea in the liver.

Symptoms of urea poisoning are:

- Animal becomes uneasy, and nervous.
- Excessive amounts of saliva are secreted.
- Defaecation, and urination, are frequent.
- Animal struggles violently, and bellows.
- Bloat often occurs.
- Tetany (painful muscular cramps caused by intense and repeated nervous stimuli), and death occur.

Important practical considerations in the use of NPN:

- Animals unadapted to urea are most susceptible to urea poisoning
- The amount of urea fed should be increased slowly over a period of at least three weeks.
- Animals showing a definite demand for salt should be allowed only limited access to a lick containing urea.
- Diets composed primarily of poor-quality roughage often do not provide enough readily-fermentable carbohydrates for efficient urea utilization.
- Fasting, or a relatively empty rumen at the time urea is fed, increases the probability of toxicity, because the microbes are not able to utilize the rapidly-formed ammonia.
- Restricted water intake reduces rumen fluid volume, and increases ammonia absorption by the animal.
- Urea toxicity is often associated with improper feed-mixing, and/or the composition of a ration or lick containing urea.
- Rations, and licks containing urea, should be fed under cover (protected from the rain).

Therefore:

- Including urea in a ration or lick for cattle should be done with the utmost care, and consideration.
- Generally, supplemental urea should not exceed more than 1% of the total ration.
- Daily intake of 0,2 g urea/kg body mass of the animal should not be exceeded.
- Special attention must be devoted to supplying readily-fermentable carbohydrates.
- Urea poisoning can be successfully treated with vinegar. Depending on the size of the animal, five litres of diluted vinegar (*i.e.* 1 part vinegar to 4 parts water) is normally dosed to the animal *via* a stomach tube. The treatment can be repeated six to 12 hours later if necessary.

Acidosis

The production of short-chain fatty acids in the rumen is directly related to the various types, and number, of bacteria and protozoa that make up the rumen microbial population. The organisms, in turn, are primarily determined by the types of feedstuffs fed to the ruminant. Some types of bacteria, and protozoa, can digest cellulose and hemi-cellulose, but not starch. Others can digest starch, but not cellulose and hemi-cellulose, and some can utilize both, with a preference for one over the other.

When the ration is changed from predominantly a roughage to a grain ration, the population shifts towards more starch-digesters. Starch-digesting bacteria produce more propionic acid relative to acetic acid, than do cellulose-digesters. Grain consists mostly of starch, which is rapidly digested. Therefore a change to a high grain-content ration will result in a rapid increase in rumen acidity, due to the rapid production of short-chain fatty acids. During this sudden change of the ration to large amounts of grain, the starch-digesting bacteria, *Streptococcus ovis,* increase rapidly, and 80 to 85% of the acid produced by this species is lactic acid. At the same time, the rumen pH falls considerably, to approximately 5, and lower, and there is a tremendous increase in histamine that could lead to laminitis.

Small quantities of lactic acid are normally efficiently converted to propionic acid, but when excessive quantities of lactic acid are produced they accumulate in the rumen, and eventually are absorbed *via* the rumen wall into the bloodstream.

The outcome of such a high lactic acid level in the bloodstream normally results in one or more of the following conditions:

- Rumen stasis (the digestive function of the rumen comes to a standstill)
- A major shift in the population of rumen microbes.
- Dehydration.
- Dehydration.
- Liver abscesses.
- Bloat.
- Rumenitis, and damage to the rumen villae.
- Damage to the mucosal tissue of the rumen.
- Cattle go off their feed, become weak, and appear depressed.
- Increased heart and respiratory rate.
- Laminitis.

When cattle in feedlots become adapted to high grain-content rations, a new microbial balance develops in which the proportion of *Streptococcus ovis* is not high, and in which other species predominate. Cellulose, and hemi-cellulose are digested more slowly than are starch, and soluble carbohydrates, resulting in a lower total concentration of acids at any one time.

THUS: One can switch rather rapidly from a grain to a roughage ration, but NOT from a roughage to a grain ration.

Important practical considerations

- Inefficient management skills on the farm or feedlot are the main cause of acidosis (for example, when the door of the feed shed is left open, or unattended.)
- Strict control over rations should be exercised, especially when switching from roughage, veld or pastures, to grain or complete rations. Changes to a grain ration should be made gradually, over a period of at least three weeks.
- Some breeds, notably the *Bos indicus* breeds, seem to be more susceptible to acidosis than others.
- Acidosis seems to appear more often during the summer months.
- If a severe case of acidosis is experienced, a veterinarian should be called immediately.
- Mild cases of acidosis can be treated by dosing with:
  - 300 g of magnesium oxide powder 800 g of activated charcoal dissolved in 5 litres of water
- If no improvement is seen within 12 hours, a veterinarian must be consulted.

CONCLUSION

The ruminant is a unique animal in many ways. A working knowledge of the ruminant's digestive tract is essential for making intelligent feeding decisions. **The slow and gradual introduction of the ruminant to a specific ration is an absolute pre-requisite for proper and efficient rumen functions.**