DEBRE MARKOS UNIVERSITY
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By

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Description:

The ruminant and its environment; anatomy and function of ruminant GIT; microbes in the gut; voluntary feed intake, rumen fermentation of carbohydrates, nitrogen, lipids and products; kinetics of rumen function; methanogenesis, effects and control; interrelations between feeding and productivity of ruminants; Nutritional disorders in ruminants.

1. INTRODUCTION

Poor nutrition is one of the major constraints to livestock productivity in sub-Saharan Africa (SSA). This is because animals thrive predominantly on high-fibre feeds (straws, stovers and native pasture hay) which are deficient in nutrients (nitrogen, sulphur, minerals, phosphorus etc) essential for microbial fermentation. Consequently, the digestibility and intake of digestible nutrients are unavoidably low. These deficiencies can partly be mitigated by supplementing roughage diets with feeds containing the deficient nutrients. Feeding practices developed in temperate countries are often inappropriate when applied to ruminant production systems in the tropics because temperate animals are fed straw as bulk in high density diets. Roughage diets and supplements may differ vastly in quality and therefore in the quantity eaten by the animal. Previously digestibility and chemical composition were used to describe the nutritive value of fibrous feeds. This proved inadequate because these attributes give little indication of the quantity of such feed an animal will eat and the quality of nutrients derived through digestion. An understanding of the factors which affect rumen degradability of low-quality basal feeds and microbial protein production will assist scientists in designing diets that will be utilized more efficiently. In addition to determining responses (performance) from feeds, there is a need to establish causal relationships.

DEFINITIONS OF TERMINOLOGIES

Digestible energy: as the name implies, is that portion which the animal can digest. It is determined in digestion trials by subtracting fecal energy loss from gross energy intake.

Metabolizable energy: often referred to as available energy, is that left over after losses of energy in the feces, urine, and combustible gases. About 82% of digestible energy is metabolizable, that is, about 18% of digestible energy is lost in urine and gases.

Net energy: is calculated by subtracting the heat increment energy from metabolizable energy. The so-called "work of digestion" energy is converted to heat and is not available for productive purposes. However, it is utilized to keep the animal warm and thus plays a critical role in cold weather. Metabolizable energy does not show the true potential of a feedstuff for productive purposes. The determination of net energy is tedious and expensive because it involves calorimetry or comparative slaughter methods to determine heat losses or energy retention. For these reasons, the net energy content of many feedstuffs has been estimated using equations relating metabolizable energy or TDN to net energy.

Productive energy*:* is the energy remaining after maintenance requirements for energy have been met. It is utilized for productive work, tissue gain, or for production of milk, eggs, wool, and fur.

Total digestible nutrients *(TDN):* are summations of all the potential energy digested by an animal. This is determined by the following equation.

TDN = [digestible fat x 2.25) + digestible NFE + digestible fiber + digestible protein]/100

Digestible energy (DE) can be calculated from TDN by assuming that 1 kg of

TDN = 4.4 Mcal of DE. Furthermore, ME can be calculated as 1 kg of TDN = 3.62 Mcal of ME.

1. **Feeding stuffs (feedstuffs): -** This term is in general used in most books interchangeably or synonymous with feed, food or fodder, although it broader or wider meaning covering all materials included in the diet because of nutritional properties. When we say feed, we normally refer to the naturally occurring plant or animal products and the byproducts prepared from them. Feeding stuffs on the other hand embraces not only the naturally occurring plant and animal products and the by-products prepared from but chemically synthesized or otherwise manufactured pure nutrients or prepared mixtures of them (artificial or synthetic) that can be used as supplements to natural feeds. Eg. Thiamine hydrochloride, pure nutrient not a feed but feedstuff. A feeding stuff is therefore any product weather of natural origin or artificially prepared, that when properly used has a nutritional value in the diet. A certain product to be a feedstuff it has to have a nutritional value or has to play a role in nutrition. Otherwise, it is not a feedstuff.

2. **Feed or Food: -** Food is generally meant for humans while feed refers to what an animal consumes. This is any non-injurious edible material having nutritional value. This could be pasture, harvested forages, crop residues, grains or processed animal feeds. One has to know that animals have no requirement for specific feeds. Feeds are merely carriers of nutrients and the potential energy we must provide in a satisfactory diet.
3. **Ration: -**A ration is a 24-hour allowance of a feed or of the mixture of feeding stuffs (feed and water). This term is general and carries no implications that the allowance is adequate in quantity or kind (quality) to meet the nutritional need of the animal. It merely refers to the daily allocation or provision of feed. There are different terminologies in connection with ration, which include.

   3.1. Balanced ration: - This is the ration that provides the nutrients necessary for all needs in terms of quantity and quality or it is a feed mixture just sufficient for the 24-hour requirement of a specific animal (or a ration in which the proportion of carbohydrate and fat to protein agree with the standard). The balance refers to the proportions of carbohydrate; fat and protein in the ration i.e. balance in terms of primary nutrients. A ration balanced in primary nutrients may however be badly deficient for the nourishment of the animal as it lacks necessary nutrients like vitamins and minerals.

   3.2. Maintenance ration: - This is a ration that satisfies the maintenance need of an animal or a ration, which does not allow production. In other words, this is the amount of feed that is needed to support an animal when doing no work and when yielding no product. The animal body no gains or losses fats, proteins or minerals. To maintain an animal at rest without gaining or losing weight, it must get adequate supplies of the following. Heat to maintain the body temperature. For each litter of oxygen consumed, the body will lose 4.825 kcal of heat as a result of metabolism. In order to maintain an animal in energy equilibrium, the indicated amount of energy must be replaced from the food supplied to the body.

   - Energy to carry on the vital functions such as breathing
   - protein to repair the small daily waste of protein tissues
   - Mineral matter to replace the small but continuous loss of, minerals
   - Vitamins
   - water and air
   - Certain fat and fatty acids especially for young calves, lambs, pigs and chicks.

3.3. Additional ration or Production ration: -Additional ration or production ration is a portion of the total ration supplied to the animal beyond maintenance ration, which would be used for production (milk, meat, egg, wool, etc.)

3.4. Least cost ration: -This is the ration that supplies the nutrient need of an animal at least cost. Examples, ration A and ration B both gives equal levels of the different nutrients but if ration A is expensive.

4. Basal feeds or energy feeds: -This is also called low protein concentrates. As the name implies these are feeds that are high in energy. A basal feed by definition contains not more than 16% protein and 18% fiber and contain greater than 60% TDN. Nutritionally basal feeds are mainly concentrate sources of energy, being especially rich in starches and sugars. They have been described as carbonaceous concentrates. They are of low protein concentrates such as corn, barley, oats, wheat, and byproducts milled from these grains. Energy feeds are very important In animal production as more feeding problems are traceable to failure to meet energy requirements than to any other single cause.

5. Supplements: -Are feeding stuffs that will be added to the total diet of an animal to increase the nutritive value. It is a feed or feed mixture used with another to improve the nutritive balance or performance of the total diet. Supplements are intended to be:

- Feed undiluted as a supplement with another feeds
- Feed offered as free choice with other part of a ration separately available
- Feed further diluted and mixed to produce a complete feed

Feed of this kind contains large amounts of proteins, and of some mineral elements, or of some particular vitamins. A mixed protein supplement by convention is a mixture of feed that carries 30% or more protein. However, single feed that carries 20% or more protein are included in the supplement category. Example, fishmeal, meat meal, nug cake, peanut cake, liver meal, dried milk. The protein supplements could be of animal origin such as meat meal, fishmeal, or plant origin, which mainly comes from oil-bearing seeds as a by-product after the oil has been extracted from the seed. Such by-products contain more protein than even the whole seed as the carbohydrate and fat has already been removed from the original seed.

6. Diet: - This term could be used interchangeably with **ration** but the term ration is mainly used for animals whereas diet is used for humans.

7. Concentrate: - In feeding practices a feed or feed mixture which supplies primary nutrients (protein, Carbohydrate and fat) and contains less than 18% crude fiber. It is a feed material either high in protein or energy (carbohydrate and fat). It should contain greater than 60% TDN. In feed trade or officially there may be other definition in which the term is used for universally prepared supplements. In this sense it refers to a concentration of protein, of minerals or of vitamins in excess of those found in the basal feed. Such concentrates are usually mixtures.

8. Forages and roughage: - In certain books they are used interchangeably but in most books forage is used to refer fresh plant materials strictly speaking to grazing and browsing or forages are plants grown to be used specifically for feeding animals such as grasses and legumes whereas roughage is meant to imply either dry or fresh plant material used for animal feeding. By definition roughage is any material suitable for feeding of

livestock which contains more than 18% crude fiber. Therefore, roughages are bulky feeds high in fiber and low in energy. Types of roughage include pasture and other grazed forages, hay and dehydrated forages, silage and crop residues and other by-products of agriculture.

9. **Nutrient: -** This is any feed or food constituent or component or a group of feed constituents be it organic or inorganic (minerals) having the same general chemical composition that aids in the support of animals.
10. **Additives: -** These are ingredients added in small quantities to a basic feed mix for the purpose of fortifying or improving the basic mix. These are not component parts of the feed mix but are added to improve some things like the test, colour, odor; antibiotics that are not necessarily part of the basic feed but have impact in visual attraction or in combating sub clinical infection like the case of adding antibiotics thereby improving utilization of feed by the animal. Hormones can also be feed additives.
11. **Ad libitum: -** This refers to a condition where animals are given feed free of choice, or as desired by the animal. The animal will not have any type of restriction or limit; it just selects and eats as much it can. It is the opposite of restriction.
12. **Toxicity: -** The term is of Greek origin and denotes a poisonous substance. It is hard to give a precise definition for this term and is better explaining by way of illustration. Some nutrients when fed in excess of the animal requirement, it will result in sickness of the animal and in extreme cases the animal may die. Such condition is called toxicity. Toxicity mainly refers to mineral and vitamins (hypervitaminosis). There is no as such toxicity for major nutrients.
13. **Deficiency: -** Toxicity is a result of oversupply of nutrients the opposite of it is deficiency which is a result of under supply of nutrients. Deficiency is a condition of sickness resulting from under supply of nutrients. This do not only include minerals and vitamins but also possible to have a deficiency of major nutrients as opposed to toxicity.
14. **Appetite: -** Appetite is the drive to consume feed or some define as the drive to eat a specific nutrient. Some use this term as a response to meet a long-term nutrient requirement.
15. **Hunger: -** This is the urge or desire to eat in response to short-term stimuli from feeding centers in the brain.
16. **Intake: -** This is the absolute amount of dry matter consumed per unit of time. Because it has the unit of time, intake is a rate, not a pool. Traditionally intake is measured over a period of 5 to 10 days and expressed as daily amounts consumed per unit of body weight.
17. **Palatability: -** This is the characteristics of a feed indicating its acceptability usually associated with the gustatory, olfactory, or visual senses. Palatability affects the preference for a feed when several are available and the rate of eating and intake when a single feed is offered.
18. **Preference: -** Relative acceptability of a feed when given the choice among two or more feeds that are available in a cafeteria-style feeding situation. Preference is a more specific indication of palatability that affects acceptability among feeds, but does not measure intake modification when no choice among feeds is allowed (a single feed is fed).
19. **Ruminal contents: -** This is amount of wet material, DM, or volume in the rumen measured at any unspecified time when the animal is fed a diet not documented to limit intake by fill.

20. Ruminal fill: - This is volume of ruminal contents when measured immediately after the cessation of a meal when the animal is provided a diet that does not meet its energy demand and intake is limited by the bulk of the diet.

21. Selection: - Specifically defined to indicate preferential consumption among feed subcomponents, such as leaves vs stems or immature plant tops vs mature plant tops

2. DIGESTIVE ANATOMY AND DIGESTION OF FEEDS IN FARM ANIMALS

Digestion**:** - The breakdown of large molecules in to simple compounds and passes via the mucus membrane of alimentary canal to blood and lymph. Digestion may be grouped into mechanical, chemical and microbial.

2.1. Types of Digestion Processes

1. ***Mechanical digestion***-brought about by mastication and contraction of alimentary canal.
2. ***Chemical digestion***-brought about by enzymes secreted by the animal in the various digestive juices, though it is possible that plant enzymes present in unprocessed foods may in some instances play a minor role in food digestion.
3. ***Microbial digestion***-it is enzymatic digestion brought about by the enzymes secreted by bacteria, protozoa and fungi, i.e., microorganisms which are of special significance in ruminant digestion. In monogastric animals, microbial activity occurs mainly in the large intestine, although there is a low level of activity in the crop of birds and the stomach and small intestine of pigs.

Note: -both chemical and microbial digestions are enzymatic.

2.1.1. Digestion in the mouth

This is mainly mechanical, mastication helping to break up large particles of food and to mix it with saliva, which acts as a lubricant and is a medium for taste perception. The saliva is secreted into the mouth by three pairs of salivary glands: the **parotids,** which are sited in front of each ear; the **submandibular (submaxillary**) glands, which lie on each side of the lower jaw; and the **sublingual glands**, which are found underneath the tongue. Saliva is about 99% water, the remaining 1% consisting of mucin, inorganic salts and the enzymes α-amylase and the complex lysozyme.

Some animals such as the horse, cat and dog lack salivary α-amylase, whereas the saliva of other species has strong α-amylase activity. The enzyme is present in the saliva of the pig, but the activity is low. It is doubtful whether much digestion occurs in the mouth, since the food is quickly swallowed and passed along the oesophagus to the stomach, where the pH is unfavorable for α-amylase activity. It is possible; however, that some digestion of starch by the enzyme can occur in the stomach, since the food mass is not immediately mixed intimately with the gastric juice. The pH of pig's saliva is about 7.3, which is only slightly above the value regarded as optimal for α-amylase activity. This enzyme hydrolyses the α-(1-4)-glucan links in polysaccharides containing three or more α-(1-4)-linked D-glucose units.

The enzyme therefore acts on starch, glycogen and related polysaccharides and oligosaccharides. When amylose, which contains exclusively α-(1-4)-glucosidic bonds, is attacked by α-amylase, random cleavages of these bonds give rise to a mixture of glucose and maltose. Amylopectin, on the other hand, contains in addition to α-(1-4)-glucosidic bonds a number of branched α-(1-6)-glucosidic bonds, which are not attacked by α-amylase, and the

products include a mixture of branched and un-branched oligosaccharides (termed 'limit dextrins') in which α-(1-6) bonds are abundant. The enzyme lysozyme has been detected in many tissues and body fluids. It is capable of hydrolyzing the β-(1→4)-N-acetyl- lucosaminidic linkage of the repeating disaccharide unit in the polysaccharides of the cell walls of many different species of bacteria, thereby killing and dissolving them.

2.1.2. Digestion in the stomach

Viewed from the exterior, the stomach can be seen to be divided into the cardia (entrance), fundus (known as the body of the stomach and contain fundic or gastric glands) and pylorus (terminus), the cardia and pylorus being sphincters controlling the passage of food through the stomach. Here α-amylase activity may continue and there is an active microbial population, mainly lactobacilli and streptococci. The cardia area covers about one-third of the surface and secretes alkaline, enzyme-free, viscous mucus formed of a gel-forming **glycoprotein** which protects the epithelium from acid attack. The gastric gland region covers a further third of the surface and secretes glycoprotein and fucolipid mucus and contains the **oxyntic cells** which produce hydrochloric acid. In addition, this region also produces pepsinogen. The third area is **the pyloric region,** which is prior to the entry to the small intestine. This area has glands, like those in the cardia region, which secrete protective mucus. Thus, the gastric juice consists of water, pepsinogens, inorganic salts, mucus, hydrochloric acid and the intrinsic factor important for the efficient absorption of vitamin B_{12}.

Pepsinogens are the inactive forms of pepsins which hydrolyzed proteins. The acid activates the pepsinogens, converting them into pepsins by removing low molecular weight peptides from each precursor molecule. Pepsins preferentially attack those peptide bonds adjacent to aromatic amino acids, e.g. phenylalanine, tryptophan and tyrosine, but also have a significant action on linkages involving glutamic acid and cysteine. Pepsins also have a strong clotting action on milk. Rennin or chymosin, an enzyme which occurs in the gastric juice of the calf and the young piglet, resembles pepsins in its activity. The products of protein digestion in the stomach are mainly polypeptides of variable chain length and a few amino acids.

2.1.3. Digestion in the small intestine

The partially digested food leaving the stomach enters the small intestine, where it is mixed with secretions from the **duodenum**, **liver** and **pancreas**. The majority of digestion and absorption occurs in the small intestine, the duodenal area being the site for mixing digesta and secretions and the jejunal area being the site of absorption. The **duodenal (Brunner's) glands** produce an alkaline secretion, which enters the duodenum through ducts situated between the villi. This secretion acts as a lubricant and also protects the duodenal wall from the hydrochloric acid entering from the stomach. Bile is secreted by the liver and passes to the duodenum through the bile duct. It contains the sodium and potassium salts of bile acids. In all farm animals except the horse, bile is stored in the gall bladder until required. The bile salts play an important digestion by activating **pancreatic lipase** and **emulsifying fats**.

The pancreas is a gland which lies in the duodenal loop and secret **digestive enzymes**, water and electrolytes, which together form the pancreatic juice, secreted into the duodenum through the pancreatic duct. A number of factors induce the pancreas to secrete its juice into the duodenum. When acid enters the duodenum, the hormone **secretin** is liberated from the epithelium of the small intestine into the blood. When it reaches the pancreatic circulation, it stimulates the pancreatic cells to secrete a watery fluid containing a **high concentration of bicarbonate ions**, but very little enzyme. Another hormone, **cholecystokinin** (**pancreozymin**), is also liberated from the mucosa when **polypeptides** and other digestive products reach the duodenum. This hormone stimulates the secretion into the pancreatic juice of **proenzymes** and **enzymes** such as **trypsinogen**, **chymotrypsinogen**, **procarboxypeptidases** A and B, α-**amylase**, **lipase**, **lecithinases** and **nucleases**.

The inactive zymogen trypsinogen is converted to the **active trypsin** by **enterokinase**, an enzyme liberated from the **duodenal mucosa**. This activation is also catalyzed by trypsin itself, thus constituting an autocatalytic reaction. The activation process results in the liberation of a hexapeptide from the amino terminal end of trypsinogen. Trypsin is very specific and only acts upon peptide linkages involving the carboxyl groups of lysine and arginine. Trypsin also converts chymotrypsinogen into the active enzyme chymotrypsin, which has specificity towards peptide bonds involving the carboxyl groups of tyrosine, tryptophan, phenylalanine and leucine. Procarboxypeptidases are converted by trypsin into the proteolytic enzymes carboxypeptidases, which attack the peptides from the end of the chain, splitting off the terminal amino acid, which has a free α-carboxyl group. Such an enzyme is classified as an exopeptidase, as distinct from trypsin and chymotrypsin, which attack peptide bonds in the interior of the molecule and which are known as endopeptidases.

Pancreatic α-amylase is similar in function to the salivary amylase and attacks α-(1→4)-glucan links in starch and glycogen. The breakdown of fats is achieved by pancreatic lipase. This enzyme does not completely hydrolyze triacylglycerols and the action stops at the monoacylglycerol stage. Dietary fat leaves the stomach in the form of relatively large globules which are difficult to hydrolyze rapidly. Fat hydrolysis is helped by emulsification, which is brought about by the action of bile salts. These bile salts are detergents. Lecithinase A is an enzyme which hydrolyses the bond linking the fatty acid to the β-hydroxyl group of lecithin. The product formed from this hydrolysis, lysolecithin, is further hydrolysed by lysolecithinase (Lecithinase B) to form glycerolphosphocholine and a fatty acid.

The nucleic acids DNA and RNA are hydrolysed by the polynucleotidasesdeoxyribonuclease (DNase) and ribonuclease (RNase), respectively. These enzymes catalyze the cleavage of the ester bonds between the sugar and phosphoric acid in the nucleic acids. The end-products are the component nucleotides. Nucleosidases attack the linkage between the sugar and the nitrogenous bases, liberating the free purines and pyrimides. Phosphatases complete the hydrolysis by separating the orthophosphoric acid from the ribose or deoxyribose.

The hydrolysis of oligosaccharides to monosaccharides and of small peptides to amino acids is brought about by enzymes associated with the intestinal villi. Only a small proportion of hydrolysis occurs intraluminally and arises from enzymes present in aged cells discarded from the intestinal mucosa. Most of the enzymatic hydrolysis occurs at the luminal surface of the epithelial cells, although some peptides are absorbed by the cells before being broken down by enzymes present in the cytoplasm. Enzymes produced by the villi are sucrase, which converts sucrose to glucose and fructose; maltase, which breaks down maltose to two molecules of glucose; lactase, which hydrolyses lactose to one molecule of glucose and one of galactose; and oligo-l,6-glucosidase, which attacks the α-(1-6)-links in limit dextrins. Aminopeptidases act on the peptide bond adjacent to the free amino group of simple peptides, whereas dipeptidases complete the breakdown of dipeptides to amino acids.

2.1.4. Digestion in the large intestine

The main site of absorption of digested nutrients is the small intestine; by the time the food material has reached the entrance to the colon, most of the hydrolysed nutrients have been absorbed. With normal diets there is always a certain amount of material which is resistant to the action of the enzymes secreted into the alimentary canal. The large intestine plays an important role in the retrieval of nutrients, electrolytes and water in the digesta. Extensive microbial activity occurs in the large intestine, especially the caecum. Here the slow rate of passage and abundant nutrient sources encourage the prolific growth of bacteria.

There is a complex population of aerobic and obligate anaerobic bacteria including lactobacilli, streptococci, coliforms, bacteroides, clostridia and yeasts. These metabolize a wide range of nitrogen and carbohydrate sources from both dietary and endogenous residues, resulting in the formation of a number of products including phenol, hydrogen sulphide, amines, ammonia and the volatile fatty acids, acetic, propionic and butyric. Bacterial action in the large intestine may have a beneficial effect owing to the synthesis of some of the B vitamins, which may be absorbed and utilized by the host. Synthesis of most of the vitamins in the digestive tract of the pig is, however, insufficient to meet the daily requirements and a dietary source is needed. The waste material, or faeces, voided from the large intestine via the anus consists of water, undigested food residues, digestive secretions, epithelial cells from the tract, inorganic salts, bacteria and products of microbial decomposition.

3. ANATOMY OF THE DIGESTIVE SYSTEMS OF RUMINANT ANIMALS

The major difference between starches, which can be digested by monogastric animals, and cellulose, which cannot, is the spatial configuration about the 1, 4- glucosidic bond. As shown above, glucose units are joined by β-1, 4 linkages in cellulose and α-1, 4 linkages in starch. This difference is one of the major factors that led to the evolutionary development of the ruminant animal. Ruminants and other herbivores developed symbiotic relationships with microbial populations having enzymes capable of degrading cellulose. Thus, ruminant species are of great value to man because they provide a means of capturing solar energy stored in the cellulosic bonds of plants. The fermentation of ingested feeds by the rumen microbes also has significant nutritional and metabolic implications for the host animal that will be discussed later.

3.1. Structure and development of the ruminant stomach

The ruminant stomach is divided into four compartments, namely, the reticulum, rumen, omasum, and abomasum. The reticulum and rumen are joined by a fold of tissue (reticulorumen fold) such that ingesta may flow from one compartment to another. Most microbial activity takes place in the rumen. The rumen is nonfunctional in newborn ruminants, but rumen fermentation starts within a few weeks after birth. Considerable growth of the rumen occurs during the first months of life with the main stimulus being solid food in the system. When the four compartments have attained their permanent relative sizes, the rumen constitutes approximately 80% of the total stomach volume.

Very early in the life of the ruminants, a mixed population of bacteria and protozoa becomes established in the rumen. The rumen then may be regarded as a large fermentation chamber providing a suitable environment for the continuous culture of the microbial population. The pH of the rumen ranges between 5.5 and 7.0, and the temperature stays very close to 103^{o}F which is near optimum for the many enzyme systems contained therein. The food supply to the microorganisms is provided in a more or less continuous manner. Contractions of the rumen wall help stir and mix intimately the microbes and the ingesta. The moist conditions are ideal for the reactions.

The function of the omasum is poorly understood. However, it does remove large quantities of water from the ingesta passing through this portion of the stomach; this may well be its sole function. The function of the abomasum is similar to that of the simple stomach of monogastrics. The abomasum is a glandular compartment in which hydrochloric acid and enzymes partially hydrolyze protein. Digesta is retained in the abomasum for a

3.2. Function of ruminant gastro intestinal tract

The main renewable carbohydrate resources in the world are cellulose, hemicellulose and pectin which occur in all plant cell walls in association with lignin. Lignin strengthens the plant's structure, but is often present in high concentrations and physically protects the cell-wall material from degradation by bacteria. Lignin is broken down by microbes under both aerobic and anaerobic conditions and lignin builds up to high concentrations only under certain

conditions, eg. in acid conditions where peats (a compacted deposit of partially decomposed organic debris, usually saturated with water) accumulate. Lignin is broken down steadily in most soils by microbes. Microbes in the rumen degrade lignin slowly and in general feed does not remain in the digestive tract long enough for lignin degradation to contribute nutrients to the animal.

The quantity of lignin is the major factor that limits the utilization of many plants by ruminants. In general, trees and tall-growing plants such as sugarcane and elephant grass come into this category. Ruminants can digest relatively unlignified plant cell-wall materials through microbial fermentation in the rumen, which places them in a particular niche (**Ecology**/**place in nature:** the role of an organism within its natural environment that determines its relations with other organisms and ensures its survival) in the food chain. The continued use of all ruminants for meat, milk, hide and wool or hair production is justified by:

1. Their ability to digest carbohydrate sources not digested by monogastric species
2. Their ability to use non-protein nitrogen (NPN) to supply themselves with protein through microbial growth in the rumen
3. Their efficient utilization of dietary protein, provided that it is protected from rumen fermentation
4. Their highly efficient use of dietary lipids for productive purposes.

This does not mean that ruminants should never be given feeds that can be readily digested by monogastrics, but their use of such feeds should be minimal. An example is the supplementation of ruminants on high-fibre diets with small amounts of protein meals, which correct imbalances in nutrients available for production and generally have `catalytic roles' in stimulating feed intake and often rumen function. As a corollary to this it is equally important that the breakdown of protein into non-protein nitrogen in the rumen should be minimized.

From the above introduction, it is obvious that the key to feeding ruminants is an understanding of the mechanisms involved in the fermentation of feed and the availability of end-products from that fermentation. The balance of end-products of digestion in relation to the requirements imposed by the physiological state of the animal affects the efficiency with which the end-products are utilized. Equally, an understanding of the rumen ecosystem and its inefficiencies provides the knowledge required to develop methods for manipulating the end-products of digestion to match the needs of the animal. This information can be used in a systems approach to animal production which aims to optimize the use of the available resources and to match livestock production systems to the resources available locally.

In order to discuss appropriate strategies for manipulating the rumen system and for providing dietary supplements that are largely fermented in the rumen, it is important to present an overview of rumen digestion and the associated constraints. The feed resources that are used as the basis of diets for ruminants in developing countries (particularly those in the tropics) are

1. Fibrous residues from cereal production, particularly from rice, wheat, maize and sorghum
2. Fermentable carbohydrate-rich byproducts from agro-industries, such as molasses from sugar extraction, the brans from processing of cereals and pasture.

Fiber and sugars are fermented at different rates in the rumen. However, the end-products of the fermentation are the same but vary in their proportions. Knowledge of the end-products of fermentation of different feeds is necessary in order to develop efficient supplementation of these diets.

3.3. Ruminant digestive tract

The mouth and teeth of ruminants are well adapted for the prehension and grinding of plant parts and there are well developed salivary glands in the mouth. A large volume of saliva is secreted even under lush (**growing vigorously:** producing a lot of vigorous rich young growth) pasture conditions, and this aids in mastication of the feed. The true stomach or abomasum is preceded by three divisions or diverticula which are lined with a stratified squamous epithelium. The rumen and reticulum are connected by a large orifice and the movement of digesta between these two regions is relatively unrestricted. The rumen and reticulum together with the omasum are referred to subsequently as the rumen. The reticular groove (the oesophageal groove) extends from the cardia to the omasum. It is formed by two muscular folds which can close to direct material from the oesophagus into the abomasum, bypassing the rumen. The groove is less functional in adult ruminants than in suckling animals unless the stimulus has been maintained into adult life by providing nutrients by suckling from a bottle.

The reticulo-omasal orifice is a `valve' which retains feed particles within the rumen until they are reduced to a size of 1 to 2 mm in diameter. Comminution of feed particles depends upon the extent of chewing, the rumination cycle, the rate of fermentation and physical breakdown through mixing. The particles leaving the rumen are therefore usually less than 1 mm in diameter and most of the fermentable carbohydrate has been solubilized. The omasum is spherical and covered with short blunt (**not sharp:** having a cutting edge or point that is not sharp) papillae arranged in such a way that digesta move between the laminae to the abomasum. Much of the water and electrolytes are absorbed in the omasum.

The abomasum (or true stomach), the small intestines (duodenum, jejunum and ileum) seem to have similar functions to those in monogastric animals. It is in these organs that the rumen micro-organisms and the unfermented but digestible residues of the feed are subject to enzymatic digestion and their products absorbed. The large intestine (which consists of the caecum and colon) is posterior to and joins the ileum at the ileo-caecal orifice. The caecum has one blind sac that projects caudo-laterally. The caecum and colon are areas of microbial colonization and fermentation occurs in these areas. Under most dietary conditions in ruminants fermentation in the large intestine contributes little to digestion of a feed (5-10%). Blind sacs are present in many of the fermentative organs, particularly those that are tube-like and in which little mixing occurs. The blind sac of the caecum is a reservoir of micro-organisms, which `seed' the digesta thus ensuring further fermentation in the large intestine. The persistence of fungi in the tract may depend on the presence of a blind sac with a slow rate of turnover of the contents.

3.4. The rumen and its environment

The rumen environment appears to be controlled by:

1. The type and quantity of food eaten
2. Periodic mixing through contraction of the rumen
3. Salivation and rumination
4. Diffusion or secretion into the rumen
5. Absorption of nutrients from the rumen
6. Passage of material down the digestive tract.

Only under abnormal circumstances is this environment drastically perturbed (disturbed, nervous, worried, agitated, uneasy, distressed, troubled, anxious) grain is suddenly introduced into the diet, lacticacidaemia may occur because of a drop in ruminal pH, growth of *Streptococcus bovis* and the accumulation of lactic acid. Saliva is continuously added to the rumen and maintains the contents in a fluid state, so facilitating access of micro-organisms to the plant materials. The volume of saliva secreted by ruminants is dependent on diet. The microbial community also affects salivary flow, which may be reduced by the presence of a population of protozoa. These rapidly assimilate starch and sugars and remove the need for copious (**abundant:** produced or existing in large quantities).

3.4.1. The salivation to maintain rumen pH

The saliva is a buffered bicarbonate solution of about pH 8 containing high concentrations of sodium and phosphate ions. The saliva and the movement of bicarbonate ions across the rumen epithelium maintain the pH within narrow limits. The buffered rumen liquor is a favorable medium for growth of anaerobic bacteria, fungi and protozoa and it allows VFAs to accumulate in the fluid (up to 0.2 molar).

Neutral conditions in the rumen are maintained by continual adjustment of the pH of the ruminal fluid by the above processes and by absorption of VFAs, thus ensuring continuous fermentation. The biomass of microbes in the rumen is maintained at a constant level by the passage of microbes down the digestive tract, and also by death and lysis of the micro-organisms within the rumen. Methane and carbon dioxide are produced as end products of fermentation. At low rumen pH, carbon dioxide comes out of solution and accumulates in a pocket in the dorsal sac. Methane and carbon dioxide are largely eliminated by eructation. At high rumen pH most of the carbon dioxide produced by fermentation or entering in saliva is absorbed and excreted via the lungs.

3.4.2. Rumination

Regurgitation of a `bolus of rumen digesta is a reflex mechanism that is superimposed on the cyclic contractions of the rumen. Generally, feeds are eaten with only a small amount of mastication and are regurgitated and masticated later. Recent work has demonstrated that on grass-based diets about twice as much dry matter passes through a

rumination cycle as is consumed. On ground diets or ground and pelleted diets rumination is absent or much reduced.

3.5. Microbes in the gut

The microbial ecosystem in the rumen is complex and highly dependent on diet. The vast majority of ruminants consume a mixture of carbohydrates, of which cellulose and hemicellulose are the largest components. However, at times the diet can contain large amounts of soluble carbohydrates or starch (eg. molasses or grain). Plants have developed molecular structures in their cell walls specifically to deter invasion by micro-organisms. In the rumen the main agents that break down carbohydrates are anaerobic bacteria, protozoa and fungi. The anaerobic bacteria are the principal agents for fermenting plant cell-wall carbohydrates but the anaerobic phycomycetous fungi may, at times, be extremely important.

There appears to be a close relationship between fungi and the other microbes in the rumen since the fungi appear to be the first organisms to invade plant cell wall, which allows bacterial fermentation to start and to continue. Some rumen microbes synthesize enzymes that degrade the most complex plant structures, whilst others use only simple compounds such as cellobiose or glucose. Some bacteria in the rumen assume a syntropic association, where one organism uses the products of fermentation of another and the removal of the end-product allows further fermentation of the primary feed resource by the first organism.

3.5.1. The Phycomycetous fungi

The anaerobic fungi of the rumen have only recently been isolated and cultured from the rumen. Anaerobic fungi have been shown to be present in the rumen of a number of animal species including sheep, goats, cattle and members of the deer family. They have also been found in the caecum of horses and elephants and in the `pseudo rumen' of kangaroos. Thus, they are probably present in all herbivorous animals and may exist in anaerobic environments those that occur in deep-sea sludges and in slurry in methane digestors.

For many years the flagellate organisms observed in the rumen, particularly in defaunated sheep (ie. no protozoa in the rumen), were described as protozoa. These were probably the motile infective stage of fungi (zoospores). Although flagellate protozoa occur in the rumen, the zoospores can be identified by the more rapid movement of their flagellae. The anaerobic roll tube is now used to culture the zoospores, which gives an estimate of their numbers and is more reliable than counting live zoospores.

The vegetative state of the fungi consists of a sporangium arising from rhizoids (similar to hyphae) which grow through the plant tissues. These are able to reach newly ingested fibre and invade the tissue, usually via damaged parts of the plant or through the stomata of leaves. They then encyst, germinate and grow through the plant particles.

Refractory materials, such as the leaves of wheat straw, when suspended in nylon bags in the rumen of sheep or cattle are heavily colonized by anaerobic fungi, with the areas around the leaf ribs the most densely populated.

The fungi appear to be the first organisms to invade and commence digesting the structural plant components, beginning from the inside. They reduce the tensile strength of these particles and thus increase particle breakdown in rumination. The damage to digesta particles by fungi allows bacteria to colonise the cell materials. They are thus extremely important initiators of fermentative breakdown of insoluble plant cell wall materials and their presence must reduce any lag-phase of fibre digestion. The species of fungi isolated from the sheep's rumen include Neocallimastix frontalis, Piramonas communis and Sphaeromonas communis. But more are being discovered. These fungi digest some of the plant structural components. It appears to be a reasonable assumption that fungi break hemicellulose-lignin complexes and solubilise lignin but that they do not actually degrade the lignin. This may allow fibre that is physically protected by lignin to be fermented by rumen bacteria.

3.5.2. Protozoa

Protozoa occur in the rumen of sheep and cattle on fibrous diets (which are low in soluble sugars) but their population densities are low (less than 100,000/ml) whereas on diets high in starch or sugars they can reach densities of 4,000,000/ml of rumen fluid. The diet also determines the species of protozoa in the rumen but little is known about the factors that determine the balance of protozoal species or their biomass. For the purpose of this presentation protozoa are divided mainly into the small entodineomorphs (largely *Entodinia* spp.) and the large holotrich protozoa (mainly *Isotricha* or *Dasytricha* spp.). The former occurs in animals fed starch-and/or fiber based diets, whereas the latter have been mainly reported to occur in animals fed sugar/fibre diets (sugarcane) and on fresh grass pastures, which are usually a combination of soluble and insoluble carbohydrates.

Some protozoa are cellulolytic but the major substrates appear to be sugars and starches, which are rapidly assimilated and stored as poly-dextran; this is mobilized as required to provide energy for the growth and maintenance of the protozoa. In this way they often `buffer' the pH of the rumen. Volatile fatty acids are also made available over a more prolonged period. When the population of protozoa is high, they may constitute up to 70% of the biomass of the organisms in the liquor with bacteria comprising only 30%.

There are a number of possible ways in which protozoa are preferentially retained in the rumen. These include:

1. Sequestration to large particles. This seems likely since protozoa are attracted to the highest concentration of soluble carbohydrates which, some time after feeding, is likely to be close to large feed particles. Also electron micrographs prove conclusively that protozoa adhere to feed particles.
2. Sequestration onto the rumen wall: some scientists found clusters of holotrichs on the reticulum wall of cattle that had been starved for one day. This is a most interesting finding as it suggests that there is communication between protozoa---how else could they come together in such highly concentrated

groupings? Perhaps chemical secretions by protozoa result in groupings of protozoa, as occurs in free-living paramecium.

3. Increased density of protozoa: Protozoa that have stored starch or sugars become dense and settle in the rumen. Experiments showed that samples taken through the rumen cannula had lower concentrations of protozoa than those taken from mixed rumen contents after slaughter.
4. Retention of protozoa in the bolus: In rumination, a bolus containing bacteria, protozoa and food particles is regurgitated into the oesophagus and reflexly squeezed as it moves up to the mouth. The liquid and small particles that are separated in this way are swallowed and immediately enter the reticulum and quickly move to the omasum. The protozoa, because of their size, cilia and attachment to large particles, are likely to be retained in the bolus, which when re-swallowed pitches onto the rumen pillar to re-enter a cycle of digesta movement through the rumen.

Although the number of protozoa in omasal fluid is less than that in the rumen, this may not be indicative of the number of protozoa flowing down the tract. If protozoa are retained in the rumen, it is likely that they will be also retained in the omasum with its more complex shape. Therefore multiplication of liquid flow rate from the rumen by protozoal concentration in omasal fluid will not give values for protozoa outflow rate.

3.5.3. Bacteria

- Bacteria are normally the largest microbial biomass in the rumen. There are a number of distinct groupings of bacteria including:
- Bacteria free in the liquid medium (usually 30% of the total)
- Bacteria attached to feed particles (about 70% of the total)
- Bacteria adhering to the epithelial lining of the rumen
- Bacteria attached to protozoa (mainly methanogens).

The continuous flow of particles out of the rumen necessitates that a proportion of the bacteria detach from particles that have been already largely digested, in order to colonize new material entering the rumen. The number of bacteria in the liquid phase is therefore important in determining the rate of colonization and therefore the rate of fermentation of feed particles. The bacteria floating free in the rumen are therefore the ones that depend on soluble nutrients but there are also those that are in `transit' between plant particles.

The most important bacteria for fibre digestion are *Ruminococcus flavefaciens, Ruminococcus albus, Bacteriodes succinogenes and Butyrivibrio fibrisolvens*. In some situations, *Cillobacterium cellulosolvens* and various *Clostridium* spp. become revalent. Hungate (1966) isolated *Clostridium lochhaedii* from the rumen of cattle fed salt treated forages. In addition to being a spore former (in contrast to normal rumen bacterial species), this organism has cellulolytic activity many-fold greater than that of the average rumen organism.

3.5.4. Major species of rumen bacteria degrading cell wall polysaccharides

Cell wall polysaccharide Species
Cellulose *Bacteroides succinogenes*
Ruminococcus flavefaciens
Ruminococcus albus
Butyrivibrio fibrisolvens
Cillobacterium cellulosolvens
Clostridium lochheadii
Cellulomonas fimi
Eubacterium spp.
Hemicellulose *Butyrivibrio fibrisolvens*
Ruminococcus albus
Ruminococcus flavefaciens
Bacteroides ruminacola

Peptic substances

Hemicellulolytic species plus:
Lachnospira multiparus
Streptococcus bovis
Succinovibrio dextrinosolvens

Information now suggests that cellulolytic organisms produce a raft of enzymes which is often associated with a capsule. These enzymes attack the complex carbohydrate degrading it to cellobiose, glucose or VFAs. On sugar-based diets, the population of viable bacteria floating free in solution seems to be small. It is possible that the large populations of protozoa in the rumen of animals fed these diets have displaced the bacterial biomass. However, it seems reasonable to suggest that many of the bacteria that use sugar are attached to the fibrous part of the digesta. The rumen bacteria present in animals on molasses diets were *Bifidobacterium bifidus*, *Catenabacterium catenaforme* and other species, as well as *Butyrivibrio* spp. and *Peptostreptococcus provistii*. On diets based on a large proportion of grain, Streptococcus bovis predominates, particularly at low pH.

3.6. Microbial Interactions in the Rumen

Rumen microbial populations vary within an animal, with time after feeding, between days in the same animal and, apparently, in animals in different countries on similar feeds. The end-products of fermentation, however, are

virtually the same. For this reason, only the interactions between the major groups of organisms and their involvements in rumen fermentation are discussed.

3.6.1. Bacteria-bacteria interactions

Both on particulate digesta and on rumen epithelial tissue, bacteria associate with related organisms and function as a consortium, one organism growing on the end products of metabolism of another.

The sequential fermentation process involving different species of organisms converting cellulose to VFAs is well recognised, as are the interrelationships between hydrogen-producing and hydrogen-utilising organisms. Within the rumen there are often very close associations of bacterial species, dependent on simple materials liberated by each to the mutual benefit of both (syntropic associations). These interactions of rumen bacteria appear to be highly beneficial and there appears to be little that can be done to manipulate these associations, other than inhibition of methanogenesis.

3.6.2. Protozoa-bacteria interactions

There is conclusive evidence that there are marked interactions between protozoa and bacteria. Protozoa ingest and digest bacteria and reduce the bacterial biomass floating free in solution in the rumen and thus they may reduce the rate at which bacteria colonise ingested food particles. With readily digestible feeds this may not be significant but with refractory feeds, predation may increase the lag phase of degradation of particles. Protozoa effectively compete with bacteria for the soluble sugars and starches, storing these carbohydrates within their cells. In this way the protozoa reduce the severity of acidosis on some diets. On sugar-based diets (eg. sugarcane) the protozoal biomass is probably larger than the bacterial biomass.

3.6.3. Interactions of bacteria, fungi and protozoa

Some scientists found that the number of flagellate protozoa (motile zoospores) increased following Defaunation of the rumen. If these flagellates were zoospores, then it suggests that protozoa either `compete' for nutrients with fungi or reduce fungal growth in other ways. To investigate these interactions, studies have been made on the effects of defaunation on fungal growth and digestibility of feed in the rumen. The digestibility of fibrous feeds in nylon bags in the rumen of sheep that were faunated, then defaunated (and remained unfaunated) and then refaunated showed that the unfaunated state of the rumen resulted in an increased rate of disappearance of straw dry matter (by 6-10 units/24 h). This was associated with more zoospores in rumen fluid and a greater growth of fungi as indicated by the numbers of sporangia on fibre that had been incubated in the rumen in nylon bags for 6-12 hours.

Elimination of protozoa in the rumen leads to an increase in the number of bacteria in the liquid pool. In studies with sheep using total faecal collection procedures, the apparent digestibility of dry matter was increased by 18% when

protozoa were not present. A scientist called Veira in 1986 has recently reviewed the literature on the effects of the faunated state on apparent digestibility of dry matter in ruminants over a wide range of diets. He found that digestibility is generally higher in the animals that are faunated as compared to their pen fed counterparts.

Any manipulation of a diet must be viewed in the light of the interactions among protozoa, bacteria and fungi. For instance, feeding concentrate supplements to ruminants on roughage-based diets often decreases the intake of roughage. The net effect of adding concentrates (and also molasses blocks) to a roughage diet may be to increase protozoal numbers. The interactions are obviously complex and the results of any research into manipulation of the rumen that does not measure responses in the biomasses of protozoa, bacteria and fungi will be difficult to explain. There is an urgent need to develop simple methods for estimating the biomass of the bacteria (in fluid and on particles), protozoa and fungi in order to explore these interactions more fully.

3.6.4. Energy Transactions in the Rumen

3.6.4.1. Fermentation of carbohydrate

The universal end-products of fermentation of all diets in the rumen are the VFAs (acetate, propionate, and butyrate), carbon dioxide and methane. Energy is lost as both heat and methane. The ATP produced by conversion of feed to VFAs and intermediary compounds used in cell growth is the main source of energy for the growth of microorganisms. Some of the potentially fermentable feed will inevitably escape fermentation and will be digested in the intestines. Recent research has demonstrated that a proportion of some feeds invariably escape intact to the lower tract (eg. maize grain). Others can be manipulated relatively easily (eg. protein meal) to avoid the fermentative processes in the rumen. Feeds that escape fermentation are described as bypass nutrients. The terms that are used in other countries to describe bypass protein (ie. rumen undegraded proteins) are not descriptive of all forms of bypass protein. Suckled milk for instance, is a form of both bypass protein and bypass carbohydrate (lactose).

Both these components of milk are readily fermented in the rumen when they enter, but because of the reflex closure of the oesophageal groove while suckling this bypass the rumen and become available for digestion in the intestines. l Bypass protein is defined as any portion of a protein meal that escapes the rumen intact and is available for digestion in the intestines l Bypass energy (mainly starch) is that part of the feed that escapes fermentation and is digested and absorbed from the small intestine.

One of the costs of the ruminant mode of digestion is that fermentation of readily digestible feeds results in up to 20% of the metabolizable energy intake being lost as heat and methane. A second major disadvantage is that proteins that are fermented in the rumen are lost as sources of essential amino acids. In subsequent chapters it is argued that in the developing countries protein is too valuable to be fermented since protein fermentation is

inefficient as a source of ATP for microbial growth (about half that of an equivalent weight of carbohydrates). Also, the N for microbial growth can be supplied in more elemental form (ie. as non-protein nitrogen-urea).

3.6.4.2. Fermentation of fat in the rumen

Fat in the diet of ruminants varies from negligible amounts to levels in excess of 10% of the dry matter in leafy forages or where animals are able to select leaf-tip materials. Most of the lipids in pasture plant materials are phospholipids and glycolipids. The major long-chain fatty acid components of these are linolenic (50%), linoleic (10%) and palmitic (15%).

The complex lipids of plants are rapidly hydrolyzed in the rumen by bacterial lipases to fatty acids, galactose and glycerol; the last two are fermented to volatile fatty acids. The long-chain fatty acids are largely unsaturated and as soon as they are released, they are adsorbed onto particles in the rumen where they are hydrogenated by microbes. These long-chain fatty acids (now largely stearic, palmitic and oleic acids) are absorbed only from the intestines. Rumen bacteria incorporate some of the long-chain fatty acids into their cellular components.

3.6.4.3. Microbial growth and fermentation

Anaerobic conditions of the rumen limit the availability of ATP for microbial growth. In aerobic microbial systems the carbohydrate is converted to carbon dioxide and water with a yield of 36 moles ATP/mole of glucose oxidized. By contrast, anaerobic fermentation yields only about 4 moles ATP/mole of glucose converted to VFAs. Rumen micro-organisms use ATP for essentially two purposes:

- For the energy to synthesise their own cells
- To provide the energy for maintenance

The ATP available for microbial growth depends on that required to maintain the organisms. The efficiency of ATP generation and cell growth also depends on the substrates that provide the `building blocks of the micro-organisms. The composition of bacterial cells is fairly uniform and the cost (in ATP terms) of synthesis of the individual components of cells can therefore be calculated.

The point being stressed in this here is that if the microbial cell components are synthesized from the glucose (eg. from cellulose), then growth is highly efficient. If, however, the end-products of fermentation (ie. VFAs) are used; the synthesis of microbial cells is much lower per unit of organic matter fermented. The estimates of microbial cell yield in terms of carbohydrates fermented indicate that it is the intermediates in the breakdown of glucose in the rumen that are used to synthesise microbial cells. Microbial growth efficiency is expressed in terms of YATP: YATP is defined as the weight (g) of dry cells that is produced per mole of ATP available. The ATP available is usually calculated from knowledge of the reactions in the fermentative pathways.

3.6.4.4. Factors affecting the quantities of rumen microbes available for digestion in the small intestines

- The major factors that affect microbial cell synthesis in the rumen are:
- The availability and/or concentration in rumen fluid of precursors (eg. glucose, nucleic acids, amino acids, peptides, ammonia and minerals (including S, K and P))
- The maintenance requirements of the microbes
- The turnover of microbial cells
- The destruction of bacteria by predatory protozoa

A continuous supply of fermentable carbohydrates to maintain both fermentation and the supply of precursors for cell growth is paramount to efficient use of ATP. The rate of fermentation must be synchronised to the rate of uptake and therefore availability of ammonia, sulphur and/or peptides, amino acids, and other microbial nutrients.

3.6.4.5. Role of ammonia in rumen fermentation:

Forty to 60% of the DM of the microbial cells is protein and therefore, the synthesis of amino acids and proteins are the main reactions that require ATP. The pathways of synthesis of amino acids in rumen microbes are not clearly defined. It is however; abundantly clear that ammonia is highly important for the efficient synthesis of amino acids and therefore microbial protein. At low ammonia levels in rumen fluid, reactions that fix ammonia into amino acids require ATP whereas, when ammonia levels are above a certain optimum, the ammonia is incorporated into amino acids without using ATP.

It has been suggested that maximum microbial synthesis rate occurs at ammonia concentrations between 5 and 8 mg N/100 ml. Different optima have been found by other researchers, suggesting that diet influences the optimum ammonia level. Recent studies suggest the value may be as high as 15-20 mg N/100 ml depending on diet. The high ammonia concentration needed for maximum cell growth suggests that the rumen micro-organisms probably have similar mechanisms for incorporation of ammonia to those in soil microbes, which assimilate ammonia via glutamate dehydrogenase. However, bacteria grown under low ammonia concentrations fix ammonia in a two-step process involving glutamine synthetase and glutamate synthase. These reactions involve conversion of glutamate to glutamine and then a reductive transfer of the amide-N of glutamine to 2-oxoglutarate and this step requires ATP.

3.6.4.6. Role of other nutrients in microbial cell synthesis:

As reported by one scientist the synthesis of microbial protein in the rumen of sheep on semi-purified diets was more efficient when casein was the nitrogen source than when urea was the N source. The efficiency of microbial protein synthesis in washed micro-organisms is increased by amino acids and peptides. This has led to the

suggestion that one of the roles of protein supplements is to increase the efficiency of microbial protein synthesis in the rumen through the slow release of amino acids, in addition to the beneficial role of the portion that bypasses the rumen. However, the relative supply of amino acids or ammonia-N has little effect on microbial yields since, at high ammonia concentration, synthesis of amino acids from the corresponding keto acid and ammonia does not require ATP.

The moderately high milk yields of cows fed semi-synthetic diets containing starch, sugar, cellulose and urea indicates a high efficiency of microbial protein production in the rumen with urea as the sole source of nitrogen. The high apparent efficiency may be due to: (i) using carbohydrate sources with differing rates of fermentation thus ensuring a relatively constant supply of precursors for microbial synthesis; and (ii) the absence of protozoa.

The role of other nutrients required to maximize the efficiency of microbial growth include amino acids and peptides, most of the macro and trace minerals (with emphasis on sulphur and cobalt) and other, as yet, unidentified compounds that increase microbial growth efficiency. An example of the presence of possible unidentified growth factors is seen in the stimulation of production that occurs when chicken litter is used to supplement urea as a source of nitrogen in molasses-based diets suggesting that there are compounds in the litter that stimulate certain groups of micro-organisms.

3.6.4.7. Microbial cell turnover in the rumen:

The rate of growth of microbes in the rumen is always greater than the rate at which microbes flow from the rumen. This is because:
Protozoa are retained and only small proportions move down the tract. Those retained apparently lyse in the rumen and are fermented.

- Protozoa engulf and digest quite a proportion of the bacterial pool (Coleman 1975)
- Bacteria and protozoa spontaneously lyse either due to the action of infective agents, lack of substrate or a change in the environment, such as a reduction in the pH of the rumen fluid.

The net effect of these interactions is considerable recycling of nitrogen within the rumen. Nolan and Stachiw found that up to 50% of the microbes that were produced were lysed in the rumen of sheep on a straw-based diet. Periodic fasting of animals may also result in lysis of a large proportion of the microbial pool in the rumen. Hespell (1979) showed that 60% of rumen bacteria died and 30% lysed when they were without substrate for 2 hours.

3.6.4.8. Influence of rumen protozoa on bacterial cells available for digestion:
At high population densities of protozoa in the rumen, a considerable proportion of the bacterial pool is engulfed and digested by the protozoa. At high population densities of *Entodinia* species of protozoa (ie. 2,000,000/ml) Coleman (1975) calculated that all the free-floating bacteria in the rumen may be engulfed, removing some 30% of the total

biomass. Recent studies have indicated that 16-30% more protein enters the duodenum of sheep when they are unfaunated than when they contain high population densities of protozoa.

The net effect of a large population of protozoa in the rumen is to decrease the protein-to-energy ratio in the end products of fermentative digestion that are available for absorption. For example, if the YATP of the rumen fermentation was 14, the ratio of microbial protein synthesized to VFAs produced would be 25g protein/MJ VFA energy, but predation by protozoa reduces the actual availability of protein to between 12 and 14 g protein/MJ VFA energy. The VFA energy available would be increased by some 25-30% and the protein available for digestion decreased by about the same amount.

3.6.4.9. Energy losses in fermentation

Energy is lost as heat in the rumen when carbohydrate is fermented to VFAs and microbial cells. The energy losses as heat are always small but influenced by YATP. The productions of VFAs, methane and cells were then calculated from the stoichiometry given earlier. The energy balance in the rumen was then calculated. These calculations show that as the efficiency of cell synthesis increases methane production and heat of fermentation decrease. Thus any factors that increase microbial cell yield may increase the availability of metabolisable energy. However, microbes are between 75 and 95% digestible. If the digestibility of microbes is considered to be 75%, the extra losses of energy as microbial residues in the faeces would remove any energetic advantage. However, proteinto-energy ratios (P/E) in the nutrients available for digestion would be increased markedly.

If, however, the rumen microbes were 100% digestible, as suggested by recent isotope studies, then an increase in YATP results in a significant increase in metabolisable energy of a diet. If different physiological states (eg. pregnancy and lactation) result in increased digestion of microbes or increased absorption of amino acids (because of hypertrophy of the intestines and/or increased enzyme secretion) an increase in microbial cell yield would be highly beneficial, increasing the efficiency of feed utilisation considerably.

3.6.4.10. The balance of microbial protein to VFA energy (P/E ratio)

To facilitate translation of YATP values into P/E ratios, the latter were calculated and the relationships are shown for two distinct fermentation patterns in the Rumen. The effect of different efficiencies of microbial growth on the ratio of protein to VFA energy (P/E ratio) available from the rumen of a steer consuming 4kg of organic matter which is totally fermentable. If YATP can vary from 8 to 25, then, in animals dependent on rumen fermentation, the protein available for digestion in the intestines relative to the energy absorbed as VFAs (the P/E ratio) can be as low as 9g protein/MJ of VFA energy or as high as 34g protein/MJ of VFA energy. This highlights the problems of a

system for calculating recommendations for N requirements when the efficiency of microbial growth is assumed to be constant (ie. 30 g N/kg of organic matter apparently digested in the rumen).

In all situations, if a nutrient needed for microbial growth is limiting, the P/E ratio will decrease irrespective of which nutrient is limiting (eg. ammonia, sulphur etc). The P/E ratio in the absorbed products is of applied significance as will be demonstrated later. It can be markedly altered by the incorporation in a diet of protein which bypasses the rumen fermentation and provides amino acids for absorption. When the objective of a feeding strategy is the production of milk, meat, hair or wool then microbial protein output from the rumen should be at a maximum relative to the energy in VFAs. The more microbial protein that is produced from a low-cost carbohydrate source, the less will be the requirements for supplementary bypass protein (which is usually the most expensive portion of a diet).

3.6.5. Protein fermentation and protein energy ratio (P/E ratio)

Protein that is fermented in the rumen is largely wasted because:l Dietary protein is fermented and essential amino acids are deaminated l Fermentation of 1g of protein generates only half the ATP that would be produced from 1g of carbohydrate. This means that only 30 to 60 g of microbial protein become available to the animal for digestion for **every kilogram** of dietary protein that is fermented in the rumen.

3.6.6. Significance of P/E ratio

Is the protein to energy ratio important in terms of applied animal production? With the exception of the working animal almost always it will be biologically beneficial to maximize the amount of amino acids absorbed relative to energy. The amino acid supply affects a large number of biological functions within the animal. Obviously where products containing protein are the objective of the feeding system, the amount of protein absorbed relative to energy will be highly related to the level of production achieved. For example, wool growth, milk production and growth in young animals will respond to increases in P/E ratio.

Considerable research has demonstrated that in animals fed low nitrogen diets, supplementation with fermentable nitrogen stimulates rumen function which stimulates feed intake. On many diets low in protein, supplementation with bypass protein stimulates feed intake. This appears to hold for a wide variety of diets from fibrous cereal residues through to diets based on starches (barley) and sugars (molasses). Amino acids absorbed from the digestive tract provide essential amino acids for synthesis of tissues, and in addition provide precursors for other compounds required in tissue growth. They also provide a proportion of the glucogenic precursors to the animal and in this way have an effect on hormonal secretions, which influences the animal's reproductive capacity.

3.6.7. N-Transactions in the Rumen

3.6.7.1. Dietary nitrogen

In the industrialized countries, only relatively small amounts of urea are fed to ruminants and protein provides most of the dietary nitrogen. The non-protein-nitrogen fractions of such feeds include amides, amines, amino acids and nitrate; the last mentioned may be present in significant quantities in immature pasture. In the developing countries, where crop residues are fed to ruminants, urea or ammonium salts and materials such as chicken manure are the most appropriate sources of fermentable N. The major non-protein-nitrogen component in chicken manure is uric acid.

3.6.7.2. Degradation of dietary protein in the rumen

Between 20 and 100% of the protein in many diets based on high-protein forages, protein meals and grains may be soluble. It is assumed, for practical purposes, that the solubility of protein-N in buffer solution indicates the degradability of the protein of a meal in the rumen. However, soluble proteins such as serum albumin, ovalbumin, and chloroplast protein extract and soluble proteins from soya bean meal and rapeseed meal have variable resistance to degradation in the rumen.

The chemical form of protein and the presence of disulphide bonds and perhaps phenolics (tannins) have major influences on degradability of proteins in the rumen. Degradation of protein to peptides and amino acids is by bacterial proteases and peptidases. Some scientists showed that lysis of leaf protein in the rumen was high but affected by diet and that soluble protein was adsorbed rapidly onto the bacterial cell prior to lysis. Small particles such as chloroplasts were engulfed directly by protozoa and then degraded only slowly. Factors affecting the extent of ruminal degradation of the less-fermentable proteins in food particles have received little study. Fermentation of particulate proteins depends on the length of time that they are in the rumen, and factors such as their rates of solubilisation and enzymatic degradation.

In addition to chemical factors affecting rates of degradation of soluble proteins (ie. cross-linking and number of accessible hydrolysable sites in the protein molecules, enzyme concentration, and pH), physical characteristics of particles also affect accessibility of proteins to enzymatic action. The surface area of the protein that is accessible to microbial proteases may be reduced by formation of fibrous proteins by treatment with formaldehyde and by lipids or other water-insoluble substances used to encapsulate the protein. Rates of disruption of these substances are major factors that affect the rate of breakdown of such protected proteins.

3.6.7.3. Outflow of dietary and endogenous nitrogenous materials from the rumen

The amount of dietary N leaving the rumen is determined principally by the total N in the diet, the rate of its fermentation and its residence time in the rumen. Protein of dietary origin in abomasal digesta is often estimated by an indirect method (ie. from the total flow of N in digesta after the microbial fraction has been identified by microbial markers and an allowance has been made for endogenous N). The endogenous component, however, should not be disregarded. The method of estimating undegraded protein leaving the rumen contains all the inaccuracies of estimating or measuring the other fractions

3.6.7.4. Peptides and amino acids in rumen fluid

In animals fed high-protein diets, a high proportion of the N in the rumen may be derived from peptides and amino acids in the feed protein. Peptides or amino acids are degraded rapidly by bacterial peptidases and deaminases, and peptides are present in the rumen in significant quantities only when the protein is fermented at high rates. Amino acids are absorbed from the rumen but probably only in small amounts as the majority of free amino acids are probably deaminated to give rise to branched-chain volatile fatty acids (VFAs), carbon dioxide and methane. The level of branched-chain VFAs in the rumen fluid is an index of amino-acid degradation in the rumen, as these generally arise from the fermentation of valine (isobutyrate), leucine (isovalerate), isoleucine (2-methyl butyrate) and proline (valerate).

3.6.7.5. Rumen ammonia pool

The sources of ammonia in the rumen include proteins, peptides and amino acids and other soluble-N materials. Urea, uric acid and nitrate are rapidly converted to ammonia in the rumen. Nucleic acids in rumen fluid are probably also degraded extensively to ammonia. The ammonia pool is a focus for studies of metabolism of nitrogen in the rumen, and much knowledge has been gained from measuring fluxes of N through this pool. Ammonia N is lost from rumen fluid by:

- Incorporation into microbial cells that pass out of the rumen
- Absorption through the rumen wall
- Passing out of the rumen in fluid.

The ammonia pool in the rumen is relatively small and turns over rapidly. The amount of ammonia entering the pool varies over a wide range according to quantity and degradability of protein in the diet and with the extent and method of supplementation of urea. Concentrations of ammonia N in the pool can be expected to change rapidly even when animals have continuous access to food. The amount of ammonia that flows out of the rumen in fluid is relatively small, and it follows that ammonia produced in the rumen that is not incorporated into microorganisms is

absorbed mainly through the wall of the reticulo-rumen. To maintain a high level of ammonia in rumen fluid over 24 hours on low-protein diets requires urea to be taken in continuously. This can be ensured by spraying urea on the basal feed or by providing a urea block or liquid mixture which is licked at regular intervals. Urea given in a single meal is unlikely to maintain rumen ammonia levels above the minimum required for efficient fermentation for more than a few hours per day.

3.6.7.6. Recycling of N to the rumen from plasma urea

Movement of urea from blood into the rumen and conservation of urea in the body by reduced excretion of urea in urine are mechanisms for supporting rumen ammonia levels above a minimum needed by the microbes. Ruminants have evolved these mechanisms to ensure efficient microbial synthesis in the rumen at moderate N intakes. However, studies with cattle and sheep on low-N pastures and straw-based diets have shown that continuous supplementation with urea increased: (i) feed intake; (ii) digestibility of the feed; (iii) N balance; (iv) protein availability from the rumen; and (v) productivity. This demonstrates that N recycled through plasma urea at times cannot meet the N requirements of the rumen organisms for maintenance or production on such diets.

In studies with sheep on straw-based diets, the amounts of urea from blood plasma entering the rumen were relatively small (0.5 to 2.3g N/d). Although urea can enter the rumen through the epithelium, it seems probable that for sheep consuming these low-quality diets, most of the urea enters the rumen via saliva. In contrast, Kennedy and Milligan (1978) found that considerable amounts of urea (6 to 10 g urea N/d) were recycled in sheep given brome grass (Bromus *inermis*), and Potthast et al. (1977) calculated that 9.5 g urea N/d entered the rumen of sheep given an N-free basal diet plus 300 g sucrose/d. Under these conditions considerable quantities of urea must have entered the rumen by transfer through the rumen wall and the suggestion is that there is a mechanism for this that is ``switched on'' when sugar enters the rumen.

This has never been proven. It is possible that a high osmolarity in the rumen through the addition of sugar causes water to flow from the blood into the rumen and this carries with it urea.
It should be noted that these studies generally involve isotope dilution techniques which may not give unequivocal results where the pools of nutrients being investigated are very small (ie. rumen ammonia) and where the samples can be contaminated with extraneous N. Any N contamination would increase the apparent recycling rate. The applied studies demonstrate responses to urea supplementation of animals fed low-nitrogen diets. This suggests that under long-term nitrogen deprivation, urea recycled to the rumen is relatively unimportant when compared with the effects of urea supplementation.

3.6.7.7. Urea (ammonia) toxicity

So called urea toxicity is characterized by neurological symptoms and possible derangement of brain metabolism. In practice, it occurs following rapid intake of urea that could be due to:

- Insufficient mixing of urea in compounded diets
- Sifting of urea to the bottom of a loose feed mixture
- Leaching of urea in troughs, permitting animals to drink solutions containing high concentrations of urea
- Excessive consumption of urea from blocks that have been softened by rain or rain water collecting in holes licked in the blocks or by over-consumption of liquid mixtures.

The likelihood of toxicity is greater in animals that have not been adapted to urea supplements. Animals that have been fasted for a day or more, and in those with liver dysfunction (eg. fluke infestation or damage from toxic plants) that prevent them from converting ammonia to urea, are also at risk.

Urea itself is not toxic; it is the ammonia produced from urea in the rumen that is toxic. When large amounts of urea are consumed, the pH and the concentration of ammonia in the rumen increase, and more ammonia is absorbed which is normally converted to urea in the liver and excreted in the urine? The usual explanation of urea toxicity is that the liver cannot cope with the increased absorption of ammonia; that the level of ammonia in peripheral blood rises; ammonia is carried to the brain in the blood and brings on the clinical symptoms. Any malfunction or inefficiency in the liver obviously increases the likelihood of ammonia toxicity. An alternative explanation that has recently been put forward is that when ammonia is in high concentrations in the rumen, some diffuses into the peritoneal cavity and from there goes by lymph drainage to the jugular vein, thus bypassing the liver. If this is the case, the efficiency with which the liver removes ammonia is less important than previously supposed.

The clinical neurological symptoms may not be the only manifestation of urea toxicity. Milder forms of toxicity may decrease growth rate without producing any obvious clinical symptoms. If this is so, it is a further reason for not supplying urea in excess of requirements. The implication is that intake of urea should be limited to the minimum needed to make good use of the available feed.

3.6.7.8. Other sources of non-protein nitrogen (NPN)

Urea is not the only source of NPN. Ammonium salts and biuret are other possible sources. The ammonium salts, like urea, provide ammonia almost instantly for microbial protein synthesis in the rumen. As with urea, large intakes over a short time can lead to toxicity and death. Ammonium salts do not seem to have any other advantages that would warrant their use in place of urea, and in some cases they appear to be more dangerous. Ammoniation of moist feeds high in sugar by injection of anhydrous ammonia creates conditions (high temperature) which favour the formation of toxic imidazole (**chemical that inhibits histamine:** an organic white crystalline base that inhibits the action of histamine (Formula: $C_3H_4N_2$) and it is a compound which cause bovine hysteria (frenzy, madness, emotion, excitement, mania). Biuret as an N supplement should have advantages over urea in that it is degraded to

ammonia slowly, and thus provides the micro-organisms with a continuous supply of ammonia. It is relatively non-toxic even when consumed in large amounts and has been used as a substitute for urea. Biuret is made by heating urea to high temperatures, causing two urea molecules to condense to biuret and ammonia.

2urea biuret + ammonia, $CO(NH_2)_2$ NH_2-CO-NH-CO-NH_2 + NH_3

Biuret is broken down to ammonia in the rumen by the enzyme biuretase. The ruminal micro-organisms will only produce this enzyme when they are presented with biuret regularly over a period. Depending on the protein content of the diet, it may take up to 6 weeks before sufficient enzyme is produced. Moreover, if the supply of biuret is withdrawn from the adapted animal for 2 or 3 days, a readaptation period is needed to regain the same capacity to degrade biuret. During the adaptation period, much of the biuret is absorbed. It is not metabolised by body tissue, but is excreted in the urine. Even when the rumen micro-organisms are fully adapted to biuret it seems likely that a large proportion of the biuret escapes degradation in the rumen and is either absorbed or passes down the digestive tract.

There is thus a considerable degree of inefficiency of utilisation inherent in the use of biuret as an NPN supplement. Another disadvantage is the high cost---about three times that of urea. Moreover, on sugar-based diets, the ammonia from biuret does not become available fast enough to match the rate of fermentation of the carbohydrate. These disadvantages offset the lower toxicity and the theoretical advantage that the slow breakdown should provide.

3.6.8. Sulphur Nutrition of Ruminants

Animals cannot synthesise the sulphur amino acids cysteine and methionine from sulphate. The ruminant relies on micro-organisms in the rumen to convert sulphate to hydrogen sulphide which is used to synthesise methionine and cysteine for microbial growth. In the industrialized world, sulphur is never likely to be deficient because of the incidence of acid rain which contains appreciable amounts of sulphur dioxide, forming sulphuric acid in the soil. However, in the less industrialized countries sulphur deficiency can be extremely important. Even in countries where the soils are derived from volcanic rock, sulphur may be deficient in the soil. This is mainly because the soils are highly leached and the sulphur salts are highly soluble and are labile in the soil. Sulphur can be the first limiting nutrient for efficient rumen fermentation, the primary effect being decreased availability of microbial protein and, as a result, loss of appetite.

3.6.8.1. Sulphur utilization in the rumen

On protein rich diets, fermentation of sulphur amino acids in the rumen leads to production of hydrogen sulphide, which is absorbed across the rumen wall, converted to sulphate and excreted in the urine. In forage plants the nitrogen-to-sulphur ratio may vary from 4:1 to 55:1, with an average value of about 15:1. The ratio of N:S for efficient microbial growth in the rumen appears to lie between 10 and 14:l for sheep and between 14 and 15:1 for cattle. If the ratio is greater than these, sulphur supplementation is needed.

Sulphur supplements that are commonly used include elemental sulphur, various sulphate salts and, to a lesser extent, the sulphur amino acids of feed proteins. Molasses contains appreciable amounts of sulphur (about 0.3%), because sulphur dioxide is used in the preparation of sugar from the sugarcane juice. The anaerobic fungi, which appear to be so important in the initial digestion of fibrous feeds, are highly dependent on a source of sulphur for their growth.

3.6.8.2. Sulphide production in fermentation:

In general, Desulfovibrio species are responsible for converting sulphate to hydrogen sulphide in the rumen. *Desulfovibrio ruminus* reduces sulphate at a rate about 20 times that previously recorded for *Desulfovibrio desulfuricans*. Analysis of microbial protein from the rumen has suggested that there is little variation in the amino acid composition of mixed bacteria and protozoa. However, the amounts of methionine and cysteine in the protein of Ruminal bacteria show considerable variation between strains.

The uptake of sulphide by micro-organisms for amino acid synthesis is determined by the size of the sulphide pool. This in turn is determined by the balance between sulphide generation, absorption and incorporation into microbial organic matter. Absorption of hydrogen sulphide is extremely rapid and concentration dependent. Sulphur thus appears to behave similarly to ammonia. The sulphate is very rapidly converted to hydrogen sulphide which is absorbed, in the same way as urea is rapidly converted to ammonia and absorbed. Sulphate recycling via the saliva is an important means of maintaining rumen sulphide levels. If the sulphide level in the rumen falls below l mg/liter of liquor, microbial growth and dry-matter digestibility of the feed are reduced.

3.6.8.3. Utilization of cysteine and methionine in the rumen:

Fermentation of soluble proteins containing cysteine and methionine is extremely wasteful. Considerable amounts of S-amino acids are degraded to VFAs, ammonia and hydrogen sulphide when high-protein diets are fed to ruminants. Feeding high levels of cysteine shows that the organisms that use cysteine can be saturated and large amounts of cysteine may flow to the lower digestive tract. For instance, when sheep were fed 5 g of cysteine in 800 g of feed, 200-400 mg of free cysteine flowed out of the rumen/day. Such large amounts of free cysteine obviously do not occur in the rumen and normally no S-amino acids per se flow from the rumen to the omasum.

3.6.8.4. Recycling of sulphur

Absorbed sulphide is oxidised to sulphate in the liver, enters the blood and is distributed through the extra cellular fluid. Sulphate is recycled to the large intestine via secretions and to the rumen via saliva. Some is lost via the urine. The amount of sulphur returned to the rumen in saliva is related to plasma sulphur levels and in cattle there is a strong positive correlation between salivary and blood sulphate. The direct flow of sulphate across the rumen wall is of minor importance and the majority of sulphur that recycles to the rumen is through saliva. On entering the rumen,

sulphate is reduced and made available for amino acid synthesis in much the same way as ammonia released from urea is available for microbial growth.

3.6.8.5. Sulphur availability in rumen fermentation:

The continuous availability of sulphur for fermentation in the rumen is important. As with urea supplementation, providing a sulphur supplement as a single meal may produce a peak concentration of sulphide in the rumen which is out of phase with the need for sulphur for microbial growth. Multinutrient blocks based upon molasses and urea may be beneficial to sulphur metabolism by favouring slow and continuous intake of sulphur. Where proteins are being fed to ruminants to provide fermentable nitrogen these also probably provide fermentable sulphur. Therefore, sulphur is unlikely to be deficient where large quantities of high-protein meals and concentrates are being fed. Where protein is a small proportion of the diet this source of S is insignificant and the animal must depend on elemental S to support efficient microbial growth in the rumen. Thus S supplementation is likely to be required in feeding systems based on crop residues and byproducts.

3.6.8.6. Toxicity of sulphur

Some individuals found that sulphur toxicity occurred where pastures were heavily contaminated with industrial effluent. In the non-industrialised countries sulphur toxicity is rare except where high levels of sulphur are given as feed supplements or are used in agro-industrial processes. The sulphur content of molasses sometimes exceeds the apparently safe levels for ruminants. The effects of this are unknown. The safe level of sulphur in a diet is between 0.1 and 0.2% and appears to vary between diets. The primary effect of a slight excess of sulphur in the diet is to reduce feed intake. High levels of dietary sulphur lead to the generation of large quantities of hydrogen sulphide gas which when eructated enters the lungs and causes severe nervous and respiratory stress. Rumen stasis (stability, stillness, immobility) is also observed in cases of severe sulphur poisoning in cattle. Concentrations exceeding 0.3 - 0.4% may cause toxic effects leading to death. A large amount of sulphur in the diet is harmful to cellulolytic organisms in the rumen and reduces fibre digestion. The breakdown of starch by rumen micro-organisms was unaffected by high sulphur levels and the effects on sugar-fermenting bacteria are not known.

3.7. Fermentation in the lower gut

Residues of feed, bacterial cells and endogenous secretions passing into the intestines are fermented in the caecum and large intestine. The stoichiometric relationship between VFA production and microbial cell synthesis is likely to be similar to that in the rumen. The VFAs produced are absorbed and little VFA appears in faeces. **Acetic acid** is the main VFA produced in the caecum and large intestine. Fluid from the caecum and large intestine has a high proteolytic activity. It is therefore, probable that the microbes present are degraded by the action of phages or other infective or predatory organisms followed by fermentation of the lysed cells. This suggestion is consistent with the high level of branched-chain VFAs present in caecal fluid relative to that in rumen fluid.

The main nitrogen source for caecal organisms is almost certainly ammonia, largely from urea entering from blood. However, other endogenous-N materials from gut epithelium cells, enzymes and rumen bacteria may all be degraded to give ammonia. Amino acids from the bacterial cells degraded in the large intestine may be taken up as such, but the apparent production of large amounts of branched-chain and higher VFAs suggests that the degraded bacterial cells are absorbed largely as VFAs and ammonia. Infusing carbohydrates into the caecum of sheep increased excretion of protein in the faeces.

The increased N excretion appeared to be bacterial cells which were presumably produced from the infused substrate. Caecal digestion of fibrous feeds may increase where nutritional (eg. Sulphur deficiency) or other factors result in considerable amounts of unfermented feed (that requires fermentation for its digestion) escaping the rumen. The nature of the fermentation in the caecum is likely to influence productivity of the animal because an increased partitioning of fermentative digestion to the caecum will decrease the proportion of essential amino acids relative to energy available for metabolism. The net result of this will be a decrease in feed intake because the Protein/Energy ratio will be lowered, particularly where ruminants are fed low-protein/ high-carbohydrate feeds.

3.8. Absorption of VFAs

The significance of the VFAs as major substrates for ruminants has been stressed earlier. It appears that about 60% of the digestible energy of a feed comes from VFAs and approximately 30% from bacterial cell constituents. If it is assumed that the organisms are digested to the extent of 80% and they are approximately 60% protein, 20% nucleic acids, 10% polysaccharides and 10% lipid, then the absorbed VFAs and products from bacteria may contribute to available substrate in roughly the following proportions: VFAs 60-70%, amino acids 20%, carbohydrate 4%, lipid 8%. To this could be added small quantities of dietary long-chain fatty acids and a variable quantity of dietary protein or starch, depending on the diet.

VFAs are absorbed from the rumen, apparently by diffusion across the rumen wall. About 25% of the VFAs are absorbed from the post-ruminal tract since they leave the rumen in the digesta. A large proportion of the fluid from the reticulum passes unchanged to the abomasum by way of the omasal sulcus (a shallow groove or depression).

But most of the water and electrolytes (particularly bicarbonate ions) are absorbed from the omasum. The VFAs are metabolized in the epithelial wall of the rumen and omasum. There are few studies which quantitatively account for the metabolism of VFAs by these organs. Since the absorption of VFAs from the rumen appears to be by simple diffusion, the requirements for substrate of the rumen wall are mainly to meet (i) the energy requirements for active transport of electrolytes and (ii) the maintenance energy requirements for the turnover of the tissue and the replacement of worn-off rumen epithelium.

The individual VFAs are metabolized by the rumen epithelium. A proportion of the acetate is oxidized to CO_2; propionate is oxidized to CO_2 but, contrary to previous suggestions, little or no conversion to lactate occurs.

Butyrate is oxidized to CO_2 and converted mainly to ketone bodies. The extent to which the VFAs are modified in passing through the rumen wall is not known. It appears that butyrate is converted quantitatively to ketone bodies which account for most of the ketone bodies in circulation in feeding animals. Acetate and propionate appear to be absorbed as such. Acetate enters the blood of the sheep at up to twice the rate of its production in the rumen. This indicates that quite large amounts of acetate are produced from absorbed **long-chain fatty acids** or that fat depots are continually turning over giving rise to acetate. Propionate is removed almost totally from blood by metabolism in the liver. Propionate contributes extensively to glucose synthesis and possibly produces 80-90% of the glucose synthesized.

The formation of B-hydroxybutyrate together with acetate, which passes unchanged through the liver, conserves substrate for oxidation in extrahepatic tissue. The liver probably obtains its substrates from propionate and butyrate and it may also remove some of the long-chain fatty acids that are absorbed from the digestive tract.

4. VOLUNTARY FEED INTAKE AND ITS REGULATION

4.1. Voluntary intake

Voluntary intake is determined by offering animals a known quantity of feed and determining the amount remaining at the end of the feeding period. Digestion and retention coefficients are determined by collecting all the excreta (mainly urine and faeces) and analyzing feed and excreta samples. The amounts of some of the nutrients absorbed and retained in the body or stored can also be determined by analyzing urine and products such as milk.

Preparing for an experiment:

- Choose an appropriate experimental design.
- Set aside enough of the experimental feed for at least 42 days' feeding particularly for voluntary intake estimations.
- One week before the experiment, confine the animals in a barn, preferably in pens with a slatted floor. Deworm the animals and start feeding them the experimental diet. Ensure that water and mineral blocks are available *ad libitum*.
- Fit the animals with faecal collection bags, if they are not kept in stalls that permit faecal collection manual collection can also be done.

4.1.1. Direct method

The experiment takes 23 days if feed is in short supply, otherwise 28 days are preferable. Offer the animals with 50 g DM/kg LW (dry matter/kg live weight) of feed daily (for roughage, e.g. stovers) or a minimum of 20–25% uneaten feed. Uneaten feed should not be refed. Follow this procedure throughout the experiment. The first 14 (or 21) days of the experiment are a preliminary or adaptation period. Days 15 through 21, or 21 through 28 (7 days), form the intake measurement period.

4.1.2. Feed-intake measurement and collection of faeces and urine

(a) Weigh the animals on the first day of the experiment and place them in individual metabolism cages. Attach the faeces bags and provide feeds according to the experiment design. The type of metabolism cage will determine whether bags are needed or not. If faeces bags are available, however, they should be preferred because their use will reduce the chances of contamination of urine by faeces.

(b) On each of the intake measurement period days collect a sample (5 – 10%) of the feed offered and save it in a large bin with an airtight lid, in a plastic bucket, or in strong plastic bag. Freeze samples of fresh feed (e.g. silage and green forage).

(c) Clean the feeders thoroughly before feeding on day 15. Each day, collect all or a fixed proportion of the feed refused by each animal and save it in a paper sack. The uneaten feed collected on days 16 through 22 corresponds to the feed offered on days 15 through 21 (7 days). Use one sack for each animal for the 7 days. Freeze fresh material samples.

(d) Empty faeces bags daily throughout the experiment. This should be done immediately after removing and weighing uneaten feed and before feeding. On day 16, the faeces bags are emptied completely. Note: Harnessing bags from day 1 is to give the animals sufficient time to adapt to the idea of carrying faecal bags. Collection starts from day 17 so bags should be emptied completely on day 16.

(e) On days 17 through 23 (7 days) collect all the faeces voided during the previous 24 hours in a bucket. Weigh, mix and take a sample for dry matter (for each day). Place a sample 5 –10% aliquot (**dividing into something exactly**: describes a number or quantity that will divide another number or quantity without leaving a remainder) in a plastic bag and save it frozen or dried pending chemical analysis. Collect and sample urine in the same way. Add acid (e.g. 0.2N HCl, 0.1N H_2SO_4) to ensure that pH is less than 3 to avoid loss of nitrogen (N).

(g) Weigh the animal after faecal collection and before feeding on day 23.

Note: When voluntary intake is being estimated, the measurement time is better extended to 28 days.

4.2. Rumen fermentation of carbohydrates

Quantitatively, carbohydrates are very important to the ruminant animal. Plant tissues contain about 75% carbohydrates. Cellulose is the most abundant organic compound in the word and composes from 20 to 50% of the dry matter of most plants. Consequently, fibrous carbohydrates, such as cellulose, are the primary source of energy for ruminants fed plant-based diets. The carbohydrates found in plant tissues are primarily polysaccharide, including hemicellulose, cellulose, pectins, fructans, and starches. Hemicellulose, cellulose and pectins are considered fibrous carbohydrates (FC), whereas fructans and starch are nonfibrous carbohydrates (NFC). The nutritive value of FC is variable and can be affected by the inherent properties of a plant material (e.g., lignification), by processing (grinding and pelleting), and by conditions occurring in the rumen (e.g., pH, particle passage rate). The nutritive value of NFC is primarily affected by the type of grain and the method of processing.

The main end-products of microbial carbohydrate metabolism in the rumen are short-chain organic acids, referred to as volatile fatty acids (VFA). The VFA provide 50 to 80% of the total metabolizable energy supply to the host. For grazing ruminants and those maintained on high-forge diets, little NFC passes from the rumen to be absorbed as glucose in the small intestine. Consequently, glucose is derived primarily from the gluconeogenic activity of the liver.

4.2.1. Rumen Physiology and Energy Requirements

Propionate and other substrates are used to synthesize glucose. Significant amounts of non-fiber carbohydrates (NFC), primarily starch, enter the small intestine in finishing cattle fed high grain diets and dairy cattle consuming large amounts of feed. However, net absorption of glucose from the gut appears to be low. Consequently, gluconeogenesis still supplies the majority of glucose needed by the animal.

4.2.2. Structural carbohydrates

The greatest activity of the rumen, and probably the least understood, is the reduction of cellulose to its constituent units. Some authors describe this as a "**three-stage" process**: a). cellulose is broken down into smaller polysaccharides which are insoluble, b). the second stage similar to hydrolysis of other polysaccharides to glucose and cellulose, and c). the hydrolysis of cellobiose to glucose. It is the initial stage that is not very well understood. A number of enzymes are involved in the hydrolysis of cellulose. The sources of such enzymes are bacterial, but some authors have suggested that protozoa and fungi may contribute to the pool of cellulases, which are capable of breaking down cellulose. Xylans and pentosans, which are found in hemicellulose polysaccharide, constitute a variable proportion of fibrous carbohydrates (FC) of grasses and legumes. Hemicellulose is degraded to varying extents in the rumen and the association of hemicellulose sugars with lignin can be a major factor affecting breakdown.

Other polysaccharides, such as galacturonic acid, galactans, and arabans, are found in pectin or are associated with pectin. Pectin concentration can be especially high in temperate legumes and certain by product feeds, such as soybean hulls. Pectins are generally rapidly degraded to VFA in the rumen. Technically, pectins are considered FC, but because they are rapidly and extensively degraded in the rumen, they have properties more similar to NFC.

4.2.3. Non fibrous carbohydrates

Ruminal fermentation of starch is an inefficient process compared to intestinal breakdown. Fermentation of starch is only about 70% as energetically efficient as hydrolysis and absorption of glucose from starch in the small intestine. Methane that arises from ruminal starch fermentation represents an energy loss as does the heat of fermentation which occurs in the rumen. However, starch fermentation supplies energy to the ruminal microbes and these results in the synthesis of microbial protein.

Consequently, the extent of ruminal starch degradability has important implications in protein nutrition of the ruminant. The origin of starch affects its utilization by the rumen microorganisms. For example, corn starch is much more readily degraded than potato starch. In Europe, where potato starch is utilized as a ruminant feed, it is readily obvious that some method of steaming or cooking potatoes be provide to make such feed more digestible. In general, about 80% of total tract starch digestion occurs in the rumen. A notable exception is barley starch, for which ruminal digestion accounts for 90% or more of total tract digestibility.

When diets rich in starch are fed to ruminants that are not accustomed to such diets, a radical change occurs in the acids present. Lactic acid content rises rapidly as does the proportion of propionic acid. Under these conditions, pH declines (acidity rises) and marked changes in the microflora occur. The above conditions exist to a lesser extent when the introduction of higher levels of starch is made more gradually; thus the practice of "bringing cattle up to a

full feed gradually" can be explained. Higher starch diets consistently result in the above conditions, but cattle fed such diets gradually tend to adapt quite well.

The concentration of sugars in most diets fed to mature ruminants is low. The fermentation of glucose, fructose, and sucrose results in the production of lactic, acetic, propionic and butyric acids. Maltose, lactose, and galactose are fermented more slowly. Rate of fermentation of glucose, for example, is related to the diet. It is more slowly fermented when low-quality rather than high-quality hay is fed.

4.2.4. Volatile Fatty Acids

The total concentration of VFA in the rumen and the proportions thereof are dependent on diet. Acetic acid tends to predominate under most conditions, with propionic acid and butyric acid following, respectively. Diets high in starch favor propionic acid production. In general, feeds which are fermented rather rapidly, as is starch, give rise to less acetic acid. Glucose is a key intermediate in the fermentation of cell wall carbohydrates and starches to VFA. One molecule of glucose is converted by rumen microorganisms to two molecules of 3-carbon pyruvic acid. Pyruvic acid is a second key intermediate in ruminal carbohydrate metabolism in that ultimately it can be converted to any of the VFA.

Acetic acid is produced from pyruvic acid following the loss of one carbon as CO_2.

$$1 \text{ pyruvic acid} + H_20 \longrightarrow 1 \text{ acetic acid} + CO_2 + H_2$$

Propionic acid resulted from addition of hydrogen to pyruvic acid.

$$1 \text{ pyruvic acid} + H_2 \longrightarrow 1 \text{ propionic acid} + H_2O$$

Butyric acid is formed by the condensation of two molecules of acetic acid.

$$2 \text{ acetic acid} + 2\ H_2 \longrightarrow 1 \text{ butyric acid} + H_2O$$

Methane is derived from the reduction of carbon dioxide by hydrogen

$$1 \text{ carbon dioxide} + 2\ H_2 \longrightarrow 1 \text{ methane} + 2\ H_2O$$

Carbon dioxide and **hydrogen** are produced as a result of acetic acid formation. The production of both carbon dioxide and methane resulted in energy lost to the host ruminant because neither is a form of energy that can be utilized. The production of propionic acid in the rumen does not result in energy losses from gas production. Thus, propionic acid production results in more efficient energy production in rumen fermentation than is true for either acetic acid or butyric acid production. It is not possible to generalize as to the relative proportions of the VFA in the rumen because diet type has a profound effect. However, **cattle fed high roughage diets** will have a ratio of 70% acetic, 20% propionic, and 10% butyric acid; those fed high concentrate diets tend to have a ratio of 50% acetic, 40% propionic, and 10% butyric acid.

The VFA are absorbed into the portal blood largely through the rumen wall (about 76%); some are absorbed from the omasum and abomasum (19%) and a small amount is passed on to the intestine (5%). Acetic acid is the major end-product of the fermentation of cell wall carbohydrates by rumen microorganisms; also, the degradation of protein resulted primarily in acetic acid formation. The importance of acetic acid in ruminant nutrition cannot be

overemphasized because it is a major energy source. In the lactating animal, acetic acid is used for milk fat production whereas in the finishing animal acetic acid is a precursor for fat synthesis. Decreasing acetic acid production can result in lower butterfat production. As an example, feeding higher levels of concentrate or finely ground forage in diets fed to dairy cows can reduce acetic acid production and result in milk fat depression. Most of the propionic acid that is absorbed from the gut is converted to glucose by the liver. A small amount may be metabolized to lactic acid by ruminal epithelium. Propionic acid is a precursor for about 80% of the glucose synthesized by the liver with amino acids and lactate being minor substrates for glucose synthesis.

Butyric acid is largely metabolized by the ruminal epithelia as an energy substrate. The end-products of metabolism are the ketones β-hydroxybutyrate, acetoacetate, and acetone. The ketones are further oxidized by cardiac or skeletal muscle or used for fatty acid synthesis by adipose or mammary tissues. Forage diets will have a ratio of 70% acetic, 20% propionic, and 10% butyric acid those fed high concentrate diets tend to have a ratio of 50% acetic, 40% propionic and 10% butyric acid. The VFA are absorbed into the portal blood largely through the rumen wall (about 76%); some are absorbed from the omasum and abomasum (19%) and a small amount is passed on to the intestine (5%).

Acetic acid is the major end-product of the fermentation of cell wall carbohydrates by rumen microorganisms; also, the degradation of protein results primarily in acetic acid formation. The importance of acetic acid in ruminant nutrition cannot be overemphasized because it is a major energy source. In the lactating animal, acetic acid is used for milk fat production whereas in the finishing animal acetic acid is a precursor for fat synthesis. Decreasing acetic acid production can result in lower butterfat production. As an example, feeding higher levels of concentrate or finely grinding forage in diets of dairy cows reduce acetic acid production and result in milk fat **depression**.

Most of the propionic acid that is absorbed from the gut is converted to glucose by the liver. A small amount may be metabolized to lactic acid by ruminal epithelium. Propionic acid is a precursor for about 80% of the glucose synthesized by the liver with amino acids and lactate being minor substrates for glucose synthesis. Butyric acid is largely metabolized by the ruminal epithelia as an energy substrate. The end-products of metabolism are the ketones β-hydroxybutyrate, acetoacetate, and acetone. The ketones are further oxidized by cardiac or skeletal muscle or used for fatty acid synthesis by adipose or mammary tissues.

4.3. Rumen fermentation of protein

4.3.1. Nitrogen Metabolism

Nitrogen metabolism in the rumen is a striking example of the influence of rumen microorganisms on the nutrition of the host animal. It has been recognized for more than a century that non protein nitrogen could be used by ruminant animals. Nitrogen metabolism in the ruminant is extremely complex. The majority of protein entering the

rumen is degraded to ammonia, with protein solubility having a major effect on the extent of degradation. Protein that is relatively insoluble is degraded less, whereas more soluble protein is degraded almost totally. Casein, a highly soluble protein, is almost totally degraded.

Fishmeal protein and blood albumin are much more resistant, and a large portion of these proteins escapes ruminal proteolysis. Forage and grain proteins are intermediate in their resistance to microbial breakdown. The solubility of feed proteins may be altered to the extent that they are less soluble in the rumen and thus bypass degradation. Such proteins reach the small intestine and are available for absorption. Balancing diets for optimum concentrations of ruminally degradable (RDP) and ruminally undegradable (RUP) protein is important for maximizing protein utilization. Heating of soybean protein to approximately 300°F appears to make it less subject to rumen degradation. Complexing of soybean protein with heavy metal salts, such as zinc, is another technique which apparently decreases ruminal solubility.

In addition to consuming preformed protein, ruminants obtain some dietary non protein nitrogen (NPN). For example, corn protein is 4% NPN, alfalfa protein is 10 to 20% NPN, and corn silage protein is 50% NPN. A portion of NPN may be indigestible, but the majority of it is converted to ammonia in the rumen. It is apparent that ammonia is a common and important intermediate in both protein and non protein nitrogen digestion in the rumen. A goal of ruminant protein feeding systems is to maximize the conversion of ammonia to microbial proteins and to minimize ammonia loss from the rumen by absorption. One might expect urea to be more useful in diets containing high total digestible nutrients (TDN); this is not the synthesis declines at higher concentrate levels. Consequently, less urea nitrogen is needed by the rumen microbes. These values should be used with caution because they are based upon equations and have not been verified in extensive feeding studies. However, the following generalizations concerning urea feeding are apparent:

1. The efficiency with which urea is utilized is not constant but varies depending upon the composition of the diet prior to supplementation
2. Urea becomes more useful when feed intake is high rather than low
3. Increasing the amount of bypass protein in the diet increases the need for a ruminally available nitrogen source, such as urea. Some ammonia is inevitably absorbed from the rumen and carried by the blood to the liver where it is converted to urea. Urea formed by the liver may take one of two possible routes:

1. It may be excreted from the body by dissolving in the urine or
2. It may be recycled into the rumen via saliva or directly through the rumen wall. The quantity of recycled urea is variable depending on the level of dietary nitrogen; 10 to 70% of dietary nitrogen may be recycled in this manner, perhaps eventually becoming synthesized microbial protein. For most feeding conditions in which diets contain adequate levels of protein, about 15% of dietary nitrogen is recycled to the rumen. In addition to ammonia absorbed from the rumen, ammonia resulting from normal protein metabolism in the body also is detoxified by the liver. Ammonia in more than token quantities is toxic to the animal. It must, therefore, be converted to urea to render it harmless. "Urea toxicity" is a misnomer because it is actually ammonia that causes toxicity. Such a condition occurs

when the concentration of ammonia in the rumen is so great that its rate of absorption into the bloodstream overwhelms the ability of the liver to convert it to urea.

Although some rumen bacteria need preformed amino acids as their nitrogen source, by far the majority of the rumen bacteria grow abundantly with ammonia as their sole nitrogen source. Ammonia is not only formed from degradation of true protein, but also from breakdown of NPN in the feed. In addition, saliva contains urea which is formed in the liver. Maintenance of ruminal ammonia concentrations in excess of the bacterial capability for utilization results in nitrogen waste.

Microbial protein is passed to the lower gastrointestinal tract where it is digested and utilized by the host animal much the same as ingested intact protein. Microbial protein composes 50% or more of the total protein entering the small intestine. The quality of protein or balance of essential amino acids of microbial protein is quite good compared with that of plant and animal proteins commonly fed to ruminants. Because it is a high-quality source of protein for the animal, careful consideration should be given to balancing diets to supply adequate energy, protein, and other nutrients necessary for maximal synthesis of protein by the rumen microbes.

4.3.2. Rumen fermentation of lipids

Fats or lipids are a group of naturally occurring substances characterized by their insolubility in water and their solubility in solvents such as ether, chloroform, boiling alcohol, and benzene. The lipid group includes not only the true fats but also materials which are related chemically (lecithin) and materials which have comparable solubility properties (cholesterol, waxes). The true fats are of interest not only because they are a concentrated source of energy (2.25 times more energy than carbohydrates) but also because a number of vitamins are associated with fat (fat-soluble vitamins, A, D, E, and K). In addition, even though fat is not considered as an indispensable nutrient per se, nutritionists recognize that certain fatty acids are essential (linoleic, linolenic, and arachidonic acids).

Of the nutrients in beef cattle nutrition, fat is generally found in small quantities, except when fat is added to the diet. Even when fat is added to the diet, it usually represents no more than from 3 to 5% of the total DM. Common feedstuffs contain fairly low levels of fat, ranging from practically none up to 2% in hays, 4 to 5% in grains, 7 to 10% in distillery by-products, 13% in rice bran, and up to 98% in oils and tallows. Thus a diet based on hay, corn, and oil meal contains less than 4% fat. However, 4% of digestible fat will contain the energy equivalent of 9% of digestible carbohydrate or protein due to its concentrated energy.

In the rumen, fat is hydrolyzed to glycerol and constituent fatty acids. Unsaturated fatty acids are hydrogenated by the ruminal microorganisms to form saturated fatty acids. About three-fourths of the lipids arriving at the abomasum are of dietary origin, while the remaining lipid is derived from phospholipids of microbial origin. In the small intestine, bile plays a role in forming fat micelles which contact microvilli of the intestinal mucosa.

Free fatty acids are absorbed in the upper small intestine and travel via lymphatic circulation in the form of chylomicrons, which consist predominantly of triglycerides and smaller amounts of phospholipids, free fatty acids, cholesterol, and cholesterol esters. The fatty acids found in chylomicrons are used as energy substrates by body tissues or as substrates in the synthesis of adipose or milkfat. Although fat may be deposited in various portions of the animal body, it is stored primarily (1) in intramuscular connective tissue, (2) in the abdominal cavity, and (3) in subcutaneous connective tissue.

When fat is to be used as a source of energy, the first reaction is hydrolysis to glycerol and the three constituent fatty acids. Glycerol enters the tricarboxylic acid cycle; the fatty acids are oxidized to CO_2 and water via β-oxidation in which oxygen acts upon the β carbon of the fatty acid moiety, eventually resulting in the release of a 2-carbon fragment.

5. KINETICS OF RUMEN FUNCTION

5.1. Flow Rates

The extent of digestion of a feed depends on its rate of digestion and on the time the feed spends in the digestion pool. Animal's requirements are met from the digested component of intake. This section describes methods for calculating flow rate constants. Flow rate is the rate (mass/time) at which digesta leaves a compartment. Fractional outflow rate is the proportion of a component of feed or of a marker which leaves the compartment per unit time. Flow rate or fractional outflow rate are estimated to determine the mean duration that feed remains in the gastro-intestinal (GI) tract, usually called the mean retention time (MRT). The time available for digestion in each pool ($t1/_2$) is also estimated since it is reported to have a strong positive correlation with organic matter (OM) digestibility. Markers or rumen evacuation can be used to estimate both rumen volume and passage rates.

5.2. Rumen-evacuation technique and its Implementation

The total weights of rumen contents can be estimated by manually emptying the rumen of each animal at different times. There should be a minimum of 24 hours between consecutive emptying.

Procedure for estimating rumen volume by evacuation:

- Remove the cover of the rumen cannula and empty all rumen contents by hand into a barrel (size depending on total weights of rumen contents). If possible, keep the barrel in a container with warm water.
- Weigh all the material, mix thoroughly and take a sample (2.0 - 2.5 kg).
- Return the remaining material to the rumen as soon as possible.

5.3. Methanogenesis its effects and control (Assignment**)**

6. FUNCTION OF FEEDS AND FEEDING STANDARDS

6.1. Principles of nutrient requirements of farm animals

The quantifications of amount of nutrients in food required for specified animal functions has been a problem occupying the attention of nutritionists since the time of Thaer and the concept has persisted of absolute requirements that refined feeding studies might be determined. As a matter of practical reality, it is obvious that the only unit of energy that might satisfy any concept of universal constancy will be Net energy (NE). Any quantification of requirements in terms of DM, TDN or even ME, will require some definition of the ration type consistent with the tabulated figures. Generally, energy requirements inherit the entire problems attendant with NE evaluation of feeds; regardless of in what form of unit they are expressed.

There is another fundamental problem related to the apparent efficiency above maintenance. An underestimation of the maintenance requirement will cause the apparent decrease in efficiency of a feed to be low, which is the same effect of the NE value of the feed being overestimated. Unfortunately, in most causes of this sort, definitive information is lacking, as to which is the error. Practically, this means that one adjusts feeding standards to account for production, or alternatively one adjusts feed values to accord with animal responses relative to the fixed standards. Respiration experiments could settle the issue; however, with estimated NE values and varying conditions of feeding, the urge to adjust feed values or standards to fit experience is not easily resisted. The results in either case are a set of relative requirements and feed values that may not represent the real quantities of energy involved.

American systems do not distinguish between growth and fattening. Despite much work on gain in respect to tissue composition, standards and feed values have not thus far benefited. The understanding of growing animals, lead to compensatory growth at the expense of the animals' own energy storage. These lean animal growths with a phenomenal efficiency upon full feeding.

The starch equivalent and the Californian system may underestimate the value of forage in predicting gain. The error could be in the tabular values in the digestibility and/or its conversion to NE or in the requirements quoted for maintenance and gain. The NRC tables may contain high values for forages that fit the supplementation of feedlot diets but over-value for forages when they are the main dietary ingredients. The problem here may also have to do with differing body compositions at low and high rate of gain, i.e. the failure of NE gain to distinguish between growth and fattening that might have different efficiencies.

6.2. Uses of energy in the body of animals

Energy in animal feeds is stored in organic compounds, broadly known as carbohydrates, proteins and lipids, having their heats of combustion, **17.4, 24.0 and 39.8 MJ kg^{-1} DM**, respectively. Differences in heat of combustion among

nutrients are caused mainly by the ratio in which hydrogen and oxygen (H: O ratio) are present. On a molar basis, H: O ratios approximate to 2.0, 4.4 and 11.6 in carbohydrates, proteins and lipids, respectively. In order to provide 'fuel', needed for metabolic processes, the energy has to be present in the form of ATP. The energy input required per mole of ATP also differs between the different nutrients, particularly when one takes the original ingested or digested feed as the energy source. In the case of the volatile fatty acids (VFAs), acetic acid (AA), propionic acid (PA) and butyric acid (BA), losses occur in the rumen due to energy losses in **CH_4**, and **fermentation heat.** With a good quality dairy diet, AA, PA and BA formed in a molar ratio of **60:25:15,** respectively**.** Some **12%** of digestible energy (DE) is lost in the form of **CH_4**, the other 6% in the urine and an additional 7% in fermentation heat.

Nutrients absorbed and available in the rumen for ruminant animals are the VFAs (acetic acid, propionic acid and butyric acid). The rest of the nutrients such as glucose from starch digestion, long chain fatty acids from lipids digestion either of feed or of microbial origin, and amino acids from feed proteins escaping rumen degradation or from microbial proteins are available and absorbed for the ruminants in the small intestine.

From the absorbed nutrients, **propionic acid and glucose** are ***glucogenic***; **acetic acid, butyric and long chain fatty acids** are ***lipogenic***, and **amino acids are potentially *aminogenic*** but can also be **either *glucogenic*** or ***lipogenic***. Varying the ration composition and feeding strategy may alter the ratio in which lipogenic, glucogenic and aminogenic nutrients become available. Shifting the site of digestion of protein and starch from the rumen to the intestine results in more protein and starch reaching the small intestine and increases the proportion of aminogenic and glucogenic energy, respectively. The fermentation pattern in the rumen can also be altered. **Rumen degradable starch** usually results in a shift towards the production of more **propionic acid** whereas more **cell walls** result in the production of more **acetic acid;** on the other hand, the inclusion of **soluble sugars** often resulted in an increase of **butyric acid** production.

6.3. Energy requirement for maintenance

6.3.1. Fasting metabolism (basal metabolism**)**

Energy is required for ***involuntary*** functions in the body. By keeping the animal with no movement and in a fasting condition, we can determine the energy required for maintenance. The **0.3 MJ/ kg BW $^{0.75}$** is the heat production when the animal is in a fasting condition and with no movement. Heat loss is not the same for all types of animals but it is similar. Surface area is highly related to body weight measurement in kilograms. For example, a body weight of **80 kg animal** has a surface area of **$80^{0.75}$** and this is called the **metabolic body weight**. ***0.3 MJ/kg BW $^{0.75}$ /day is a basal metabolism per day***. Basal metabolism is **net energy required for maintenance**. Example, a 300 kg body weight cow has a maintenance requirement of about **20.63 MJ/day** and **calculated as 0.3 X 300 $^{0.75}$**

6.3.2. Balance trail

Other methods used to calculate energy requirement is by the method of feeding animals and subtracting the loss through faces and urine e.t.c. **Energy intake** – **losses of energy** = **zero,** is the energy required for maintenance. This requires an instrument known as **animal calorimeter** to measure the losses of heat from the body of animals.

6.3.3. Feeding trial method

The third method is practical in our country but it still requires more money, labor, and time. The method is employed by feeding the animal bellow and above maintenance and the place where intake **minus losses = zero** is the maintenance requirement for energy.

6.3.4. Energy requirements for production

The simplest way to determine the energy required for milk production can be by determining the amount of energy contained in the milk. By doing so it is possible to know the energy required for the production of milk and other products. The energy from the milk is determined by **bomb calorimeter**. For milk production, ME for lactation (L) = 5.3 MJ/kg fat corrected milk (FCM). For example, from 1kg corn, we can get 14 MJ energy, so to get 5.3 MJ of energy to produce a kilo of milk we must use **0.378 kg of corn**. Therefore, it is possible to produce about 2.65 kg of milk from a kilo of corn. The energy in milk is from proteins, fats, and CHO (lactose) and 4% of the milk is fat.

6.3.5. Energy requirements for body weight changes (growth, fattening and conception)

- ✓ Growth MEg (protein and fat) = 34 MJ/kg change in body weight. Net energy requirement is 18/0.53 = 33.96 MJ ~ 34 MJ.
- ✓ On the other hand ME for fattening = 60MJ/kg body weight change and net energy requirement is 36/0.6 = 60MJ
- ✓ Conception (MEc) = 25MJ/kg body weight change = 5/0.2 = 25. The mother gives priority to supply their fetus and 20% of the ME utilized is used for their fetus.
- ➢ Maintenance requirement (MEm) $= \frac{0.3 \text{ MJ X Kg } W^{0.75.}}{0.7}$

The efficiency of conversion of ME to NEm is 70%. e.g. for a 300 kg body weight cow we can calculate the energy requirement for maintenance as:

- ➢ $\frac{0.3 \text{ MJ X } 300 \text{ kg } W^{0.75.}}{0.7} = \frac{21.63}{0.7} = 30.88 \text{ MJ}$

6.3.6. Lactose syntheses in milk

The syntheses of lactose from glucose can be attained with high degree of efficiency but in ruminants carbohydrates are fermented to volatile fatty acids and lactose is synthesized from propionic acid. In the process of gluconeogenesis to synthesize glucose from propionic acid some energy is wasted as a result the efficiency of utilization of ME for lactose synthesis in ruminants is lower.

6.3.7. Efficiency of utilization for fattening: -

In fattening non-ruminants, fat ME is more efficiently utilized than the ME of carbohydrates and proteins. Energy is not required in Acetyl COA making in fats but for protein and carbohydrates, some energy is used. For ruminants, the case is similar but the efficiency of utilization is lower in ruminants. Considering the volatile fatty acids, acetic acid is less efficiently utilized than fatty acids of higher molecular weight. In reference to the purposes, efficiency of utilization of ME for fattening is less than the efficiency of utilization for maintenance. The reason for this is that some energy is lost during anaerobic processes. The efficiency of utilization of ME for growth is higher than the efficiency of utilization of ME for fattening and the efficiency of utilization of ME for growth in young growing animals is higher than that of older animals. The reasons is that in younger growing animals energy is stored in the form of fat and proteins and protein synthesis is believed to be more efficient than fat synthesis and efficiency of ME utilization for lactation is higher than the efficiency of utilization of fattening because: -

1. In milk about half of the energy is contained in protein and carbohydrates.
2. Fatty acids of milk have lower molecular weight than that of body fat.

CONCLUSION: - In terms of Efficiency of utilization of ME for any purpose:

Efficiency of utilization for maintenance > E.U for lactation > E.U for growth > E.U. for fattening. The same pattern/principle follows in the cases of NE (net energy) as that of metabolizable energy i.e. NEm > NEl > NEg > NEf. NE for any purpose is equivalent to ME/k, where k is the efficiency of utilization. NE = ME/k

Example. A certain food has metabolizable energy of 14 and k = 0.7, then the efficiency of utilization is calculated as 14/0.7 X100 = 20 %.

One should know that net energy value is not constant because it could vary according to the use for maintenance, lactation, growth or fattening. Other factors that can affect the utilization of ME include:

- ✓ Associative effects of feed stuffs.
- ✓ Level of feeding (related to rate of passage)

6.3.8. Measurement of energy value and energy requirement

The evaluation of food energy

This includes the different systems

1. ME system
2. NE system
3. DE/TDN system

6.3.8.1. Digestible energy or metabolizable energy system

Though **NE** is the most accurate system, people use **DE** or **ME** systems. DE = GE – FE. DE indicates the energy that is apparently absorbed. It is not very precise because it does not include losses in urine and as a form of gas but still it is an important indicator of the energy value of food. The losses that occurred do not affect the energy value of foodstuffs.

6.3.8.2. Total Digestible Nutrient (TDN) system

TDN refers to the chemical composition analysis and digestible energy trials combined.

TDN = $\frac{\text{DCF + DCP + DNFE + (DEE X 2.25)}}{\text{Kg DM}}$ = X, and can be expressed as X g/Kg DM

- ✓ **Total DM = 44.12% = 441.2 g DM/ kg feed**
- ✓ **DCF = 30% = .30 x 44.12% = 13.236% =132.36 g/kg feed**
- ✓ **DCP = 1.315% = 0.01315 X 44.12% = 0.58 % = 5.8 g/kg feed**
- ✓ **DEE = 3.52% = (0.0352 X 44.12%) X 2.25 = 3.34% = 34.94 g/kg feed**
- ✓ **ASH = 11.745% = 0.11745 X 44.12% = 5.182% = 51.82 g/kg feed**

DNFE = Total DM% - DCF%+ DCP% + DNFE% + (DEE% X 2.25)

DNFE = 44.12 – (13.236% + 0.58% + 3.34% + 5.182%) = 22.338% = **44.12% – 22.488% = 21.632 % = 216.32 g/kg feed**

TDN = $\frac{\text{132.36 + 5.8 + 34.9 + 216.32}}{\text{Kg feed}}$ = $\frac{\text{389.38}}{\text{1kg feed}}$ = 389.38 g/Kg feed

The advantage of TDN

1. Its value can be readily determined by direct measurement
2. The TDN of any feed is more constant than its **net energy value**. The efficiency of utilization of ME greatly affects the value of NE. The disadvantage of TDN is that it is less accurate than any particular methods used to predict the performance of productive animal. TDN does not consider energy lost through urine and heat increment.

6.3.8.3. The Metabolizable Energy System (ME System)

It is another method of telling the energy value of feedstuffs. **ME** can be determined by subtracting energy losses in the faeces, urine, and in the form of gas from the gross energy.

ME = GE – (FE + UE + GE)

One complication in the ME system is that a feeding level could depress digestible energy and this results on the under estimation of metabolizable energy (ME). When feeding level increases the rate of passage through the

alimentary canal increases as a result digestibility could be depressed. Generally, when fecal energy ↑ ME↓. Processed roughages pass through the digestive system at a higher rate than roughages such as long grasses. These factors lead to the depression of ME.

ME = GE – FE – UE - GE

In practical conditions consideration of metabolizability = ME⁄GE. Metabolizability is important to insure normal intake. In addition, it is found that feeds that have ≥ 0.62 have ME values that do not change much due to the levels of feeding. Only poor-quality diets have low metabolizability. In practical situations low quality diets do have poor palatability and they are also bulky and as a result their consumption (intake is low). All feedstuffs that have the metabolizable value of < 0.62 are not supplied to animals for production purposes E.g. for milk production. With all these conditions ME, system is modified by many workers. E.g. Blaxter's ME system (an English man). In this system, the energy value of food is expressed in ME. This new system considers the major factors known to affect the utilization of food energy including metabolizability or concentration of ME in dry matter. It considers efficiency of utilization for maintenance, lactation, growth, and the level of feeding. Besides the level of feeding, he also suggested that the efficiency of utilization of energy for maintenance and production is different. Therefore, this is to be considered in determining the energy values of feeds.

6.3.8.4. Efficiency of utilization

- ✓ **Efficiency of utilization** = $\frac{\text{Change in ME retention}}{\text{Change in ME intake}}$ **X 100**

In most cases, this is expressed in percent and that is why it is multiplied by 100. The value of efficiency of utilization can vary due to certain factors such as the **nature or the chemical compounds in which the ME is contained**. Carbohydrates and fats provide ME that can be utilized more efficiently than the ME in protein.

- ✓ This is because about 20% of the ME in protein is expended to synthesized urea.
- ✓ The other important point that affects the efficiency of utilization is the purpose to which the nutrients are used by the animal. In protein synthesis say for example energy is utilized in the linking of amino acids.

If the process were straight forward, the efficiency of utilization for protein syntheses would have been relatively high but the case is different because when some proteins are synthesized others undergo degradation. As the result, the efficiency of utilization of ME for protein syntheses would be reduced relatively.

6.3.8.5. Lactose syntheses in milk

The syntheses of lactose from glucose can be attained with high degree of efficiency but in ruminants, carbohydrates are fermented to volatile fatty acids and lactose is synthesized from propionic acid. In the process of gluconeogenesis to synthesize glucose from propionic acid some energy is wasted as a result, the efficiency of utilization of ME for lactose synthesis in ruminants is lower.

6.3.8.6. Efficiency of utilization for fattening

In fattening non-ruminants, they utilize fat ME more efficiently than the ME of carbohydrates and proteins. Energy is not required in Acetyl COA making in fats but for protein and carbohydrates, some energy is used.

For ruminants, the case is similar but the efficiency of utilization is lower in ruminants. Considering the volatile fatty acids, **acetic acid is less efficiently utilized than fatty acids of higher molecular weight**. In reference to the purposes, efficiency of utilization of ME for fattening is less than the efficiency of utilization of ME for maintenance. The reason for this is that some energy is lost during anaerobic processes. The efficiency of utilization of ME for growth is higher than the efficiency of utilization of ME for fattening and the efficiency of utilization of ME for growth in young growing animals **is higher than that of older animals**. The reasons is that, in younger growing animals energy is stored in the form of fat and proteins and protein synthesis is believed to be more efficient than fat synthesis. Efficiency of ME utilization for lactation is higher than that of the efficiency of utilization of fattening because: -

- ✓ First, in milk, about half of the energy is contained in protein and carbohydrates
- ✓ Secondly, fatty acids of milk have lower molecular weight than that of body fat

CONCLUSION: - In terms of Efficiency of utilization of ME for any diet

Efficiency of utilization for maintenance > E.U for lactation > E.U for growth > E.U. for fattening. The same pattern/principle follows in the cases of NE (net energy) as that of metabolizable energy i.e. NEm>NEl>NEg>NEf. NE for any purpose is equivalent to ME/k, where k is the efficiency of utilization. NE = ME/k

E.g. A certain food has metabolizable energy of 14 and k = 0.7, then the efficiency of utilization is calculated as 14/0.7 X100 = 20%. One should know that net energy value is not constant because it could vary according to the use for maintenance, lactation, growth or fattening. Other factors that can affect the utilization of ME include - ► Associative effects of feedstuffs

► Level of feeding (related to rate of passage)

7. THE ROLE OF VITAMINS AND THEIR REQUIREMENTS BY ANIMALS

7.1. What are vitamins?

Vitamins are usually defined as organic compounds required in minute or small amounts (in ml) for normal growth and maintenance of life of animals compared to other nutrients. They are now known to have important role in cellular metabolism. Vitamins are discovered in different times starting in the beginning of the 20th century. Experimental animals mostly rats were fed with purified diet containing adequate fats, proteins, carbohydrates, and inorganic salts showed abnormal growth and poor health condition. This prevents that some essential factors were lacking. These factors were eventually found to be vitamins. The name vitamin is derived from Vital Amins because; during the earlier times scientists thought that the essential factors contained nitrogen compounds such as amides. This was however not absolutely true, because many of the vitamins do not contain nitrogen.

The vitamins were discovered by doing experiments with purified diets. The associations of certain deficiency diseases with some unknown essential factors were recognized earlier. E.g. British naval physician called by the name James Lind has written that scurvy could be cured by eating salads and summer fruits. Earlier from this time cod- liver oil was preventing in some places in the middle and Far East like India known to be cured by eating brown rice rather than polished rice.

There are at least 14 vitamins which have been accepted as essential food factors. It is convenient to group vitamins in to two. They are: -

1. **Fat soluble**
2. **Water soluble.**

Table …. Fat- and water-soluble vitamins

S.No	**Fat soluble vitamins**	**Water soluble vitamins**
1	A (retinol)	B- Complex vitamins
2	D2 (ergocalciferol) D3 (Chole calciferol) D-vitamins	B1- (Thiamin) B2 (Riboflavin)
3	E (Tocopherol)	B6 (Pyridoxines).
4	K (Phylloquinone)	B12-cynocobalamine C- Ascorbic acid Unnumbered B vitamins: - Nicotinamide (Niacin), Pantothenic acid, biotin. Folic acid (Folacin), Choline

7.1.1. Vitamin A (retinol)

Vitamin A is a chemical substance which is solid in normal temperature and soluble in fat solvents. It is normally characterized by a pale-yellow color. Vitamin A is destroyed upon exposure to air and light due to the presence of the double bond of the side chain. Its chemical structure is

The function of vitamin A is classified into three:

1. Vitamin A has the role in the stimulation of light passing in to the brain and this can be shown chemically as follows: - There are reactions that close and open the transition of message to the brain.
2. Vitamin A is involved in the synthesis of mucopolysaccharides. Mucopolysaccharides are compounds of proteins containing carbohydrates that are important in the development of bone, mucus membrane and epithelial cells. In addition to this mucopolysaccharides are required in the formation of antibodies and also the secretion of mucus.
3. Vitamin A is required in the syntheses of steroid hormones. If vitamin A is deficient vision is hindered /affected/. Development of bones, mucus membrane, and epithelial cells and reproduction is affected.

7.1.2. Deficiency symptoms (problems) of Vitamin A

Vitamin A is sometimes known as epithelial tissue protective vitamin because in cases of deficiency even in mild conditions the skin becomes dry and scaly. The epithelial cells of the digestive and the respiratory tract are likewise affected. This is particularly true in babies and young animals than the adults. If the deficiency is more or sever, the glands of the eye reduced their secretion and this leads to night blindness. If there exists lack of retinol light stimulation will be low, as a result closure and opening may be difficult and slow movement of iris causing blurred vision.

In the deficiency conditions of vitamin, A animal would not be able to see in dim light and this problem is commonly known as night blindness. On the other hand, there will be excessive lacrimation of them causing softening and cloudiness of cornea. This leads into the drying of conjunctiva causing the disease known as **Xerophthalmia**. When there is excessive deposition of minerals the development of the skull is affected causing the optic nerve to be constricted leading to permanent blindness. In mild vitamin A deficiency conditions, the hair becomes rough, the skin dries and becomes scaly and the young animals are susceptible to infectious diseases. Scouring and pneumonia are common in this group of animals. In breeding animals infertility and abortion resulted from vitamin A deficiency. Considering farm animals, the condition of vitamin A is more prevalent in young animals which lack colostrum and in adult animals which are fed in doors on high cereal ration and excessively irradiated hay (forages), and foodstuffs which are stored for a long time.

7.1.3. Sources of vitamin A

Vitamin A is found in liver, egg yolk, and milk fat. The liver is the storage organs of vitamin A. and the amount found in it varies according to the type of the diets. Vitamin A doesn't exist in plants. Instead plans contain provitamins or precursor of vitamins in the forms of carotenoides. Of all vitamins, β- carotene is the most active and most widely distributed.

Green feeds are excess sources of β - carotene but this potency can decline rapidly when the food is exposed to air and sun light. Yellow maize is also good sources of β- carotene. Apart from this cereals are devoid of β-carotene.

7.1.4. Vitamin D

Vitamin D represents steroid molecules which are important in the absorption of Ca and P. The two molecules known as cholecalciferol and ergocalciferol are classed as Vitamin D. These two are synthesized from two precursors. Ergocalciferol is synthesized from 7- dehydrocholesterol.

Ergo sterol	U.V. Light ⟹	Ergocalciferol
7- Dehydrocholesterol	U.V. light ⟹	Cholecalciferol (is available under the skin of animals

7.1.5. Functions of vitamin D

Vitamin D is now known to induce the synthesis of certain specific protein, which is involved in the absorption of Ca, a process takes place in the microvilli of the intestine. The function of vitamin D in stimulating the synthesis of Ca carrier proteins is regulated by parathyroid hormone. Activated parathyroid hormone is in turn regulated by the Ca level in the blood. When the Ca level in the blood is low the parathyroid glands are stimulated to release parathyroid hormone. The parathyroid activates the production of more active vitamin D. The active form of vitamin D, besides the involvement in the absorption of Ca in the small intestine, they also increase the degree of absorption of P from the small intestine and enhance the reabsorpition or resorpition of Ca and P from the kidney.

7.1.6. Vitamin D deficiency

When vitamin D is deficient, major types of problems is rickets in young animals and osteomalacia in adult animals.

- Vitamin D deficiency can occur in young housed animals which are fed with feed stuff that are not irradiated adequately.
- Another cause that of recently known factor is rachitogenic factors. Rachitogenic factors are substances that have the potential of causing rickets. Fresh grain cereals and yeast are now known to have rachitogenic properties against mammals. Raw liver and soybean protein are reported to have similar effect on poultry. But heat can destroy the rachitogenic properties.

Vitamin E

Vitamin E represents 8 closely related active compounds which are naturally occurring. Among the compounds which are the most active vitamin E is α- Tocopherol.

Functions of vitamin E

Vitamin E has two very closely related functions.

1. It prevents peroxide formation
2. It destroys any peroxide which is formed in biological material before causing damage to any cell.

The first function however is nonspecific in that it prevents all cells from oxidative damages because of this effect vitamin E is known as nonspecific biological antioxidant.

The second role of vitamin E is in destroying peroxides. But the same function is also known to be performed by selenium (Se) containing enzymes known as Glutathine peroxidase and it is this function that relates vitamin E and selenium. A more recent development indicator is that vitamin E is involved in the development and function of **immune system**, but its role in this regard is not yet very much clear. The effect of vitamin E deficiency in smoothen skeletal muscles is due to their oxidation of the phospholipids in the membranes of cells. Most of the symptoms are related to this effect.

7.1.7. Deficiency symptoms

The deficiency problem that affects all animals is known as **muscular dystrophy**. In such conditions muscles are affected or oxidized, in such a way that they wouldn't perform their normal functions when skeletal muscles are affected. Animals loose strength and it becomes difficult for it to support itself, and when the deficiency symptom is extended it becomes **lame.** On the other hand, when the heart muscle is affected death may occur suddenly. This actually occurs in later stages of deficiency. In different types of animals different types of symptoms are observed. This may include **sterility** as observed in rats, **encephalomalacia** in chicks. Encephalomalacia is a kind of madness in chicks that occurred when brain is affected by peroxides. Other problems, such as liver necrosis and exudative diathesis, are observed in rates, chickens and other animals. Diathesis is abnormal permeability of blood vessels. **H_2O_2** are highly active oxidant that can alter the property of organs.

H_2O_2	Tocopherol or glutathione peroxide ⟹	$2H_2O_2$

1. If foodstuff or grains are excessively irradiated vitamin E will be destroyed that resulted in vitamin E deficiency.
2. Long time storage of especially cereal grains is also the causes for the deficiency of vitamin E. cereal grains are naturally good sources of vit. E. but their vitamin E potency will deteriorate with time of storage.

7.1.8. Vitamin K

A number of this type of vitamins are known vitamin K_1 known as phylloquinone is found in plants and it is the most important naturally occurring active compounds. Vitamin K_2 Menaquinone is produced by bacteria and is also considered as vitamin K activative. Chemically both vitamin k1 (phylloquinone) and Vitamin K_2 (Menaquinone) are derived from K_3, which is known as Menadoine. Another substance as a precursor of this is Naphthoquinone. In the liver al the vitamin K's including (Phy, Mena, and Menadoine) are all converted to Menaquinone. As a result, menaquinone is converted to be a metabolically active form of vitamin K.

7.1.9. Uses of vitamin K

It is necessary for the formation of Prothrombin which is important for the **clotting of blood**. Some conditions are known to cause deficiency of vitamin K. There are some foodstuffs such as spoiled clover which contains a chemical **dicumarol.** This chemical is reported to have antagonistic effect on vitamin K, otherwise, the problem is not common. Ruminants may not require vitamin K supplement from food origin because the microorganism in the reticulo-rumen can synthesize it. The major deficiency symptom associated with vitamin K is delayed blood clotting. All green feed are good sources of vitamin K.

7.1.10. Water soluble vitamins (Vitamin B_1 (thiamine)

These water-soluble vitamins include all the vitamin B's and C. Thiamine consists of a molecule of pyrimidine joined to a thiazole ring. It is highly soluble in water and has a meaty flavor. It is destroyed by heat especially in the presence of alkali. This vitamin is a component of Co-enzymes and is involved in the oxidative decarboxilation of α-keto acids such as pyruvic to Acetyl CoA and α- ketogluterate to succinyl CoA. The passage from pyruvate to the TCA is blocked due to thiamin deficiency. This deficiency problem of thiamine is associated with the accumulation of pyruvic acid and its reduction product, lactic acid, in the tissue. It is because of this that there is more effect on the nervous tissues of the animal. In the case of pigs there is a loss of appetite, reduction in the growth rate, diarrhea and vomiting. Birds develop polyneuritis on thiamine deficiency diets. This condition is characterized by nerve degeneration and paralysis.

7.1.11. Deficiency causes of thiamine

A number of conditions can cause thiamin deficiency. One condition could be related to washing or polishing of cereal grains. Raw fish can cause thiamine deficiency because of the presence of an enzyme known as thiaminase, which can breakdown thiamine. Recently researches indicated that some bacteria can produce these enzymes. In general thiamin is widely distributed in cereal grains if not polished and all green feeds and milk. Beriberi, a nervous system disease, is caused by a lack of thiamin in the diet.

7.1.12. Riboflavin (vitamin B2)

It is an important component of flavo-proteins, such as **flavin adenine dinucleotide** (**FAD**) and flavo-protein in here are concerned with a biochemical reaction involving H transport. Important conditions that could cause riboflavin deficiency are that all cereals are poor sources of riboflavin. Otherwise green forages in cases of animals and green crops in cases of human beings are good sources of riboflavin and milk is also a good source of it.

7.1.13. Deficiency symptoms

In cases of chicks **curled toe paralysis** occurs which is the intermediate effect of acids in the metabolic process.

Nicotine amide (NAD, NADP)

Nicotine amide consists of two Co-enzymes which are involved in the transfer of hydrogen in the living cells. These Co-enzymes are

1. **Nicotine amide adenine dinucleotide (NAD)**
2. **Nicotine amide adenine dinucleotide phosphate (NADP).** They are involved in the metabolism of energy.

The feedback mechanism will be blocked and the food appetite is closed due to the blockage of the energy metabolism. So vitamin B tablets are used or given for this treatment.

Important conditions that causes nicotine amide deficiency is when the predominant component for the diet is maize. Maize is poor in nicotine amide and also in the amino acid tryptophan, which is a precursor of nicotine amide. All protein rich diets and cereals except maize provide adequate amount of nicotine amide.

7.1.14. Vitamin B_6 (pyridoxine)

Pyridoxine in the form of pyrdoxal phosphate plays a role as a Co-enzyme in the reactions concerned with the transformation of amino acids. Transaminase is responsible in activating the reaction. And with this the pyridoxal phosphate dependant enzymes are identified. The vitamin can also enhance amino acid absorption from the intestine. It is widely distributed in a number of types of food and food contents. It is not a type of vitamin that causes practical problem.

7.1.15. Folic acid (Folacin)

It is involved in the transfer of one carbon group such as methyl and formyl which are important for the synthesis of choline methylin and also the synthesis of thymine, and purines. It is also essential for the synthesis of adrenalin hormones. It is involved in the metabolism of proteins. Besides this it is involved in the metabolism of serine and glycine.

7.1.16. Choline

The classification of choline as a vitamin is questionable since it can be replaced in the diet by other compounds. It can be used for the synthesis of choline components of lipids such as lecithin and sphingomyelin. It is not known to involve as a Co-enzyme that is why it is not classified as a vitamin. In mammals it can be synthesized from met lysine, serine, folacine, and vitamin B12. Cyano cobalamine. In birds the metabolic methylation is difficult; as a result, cholline is a dietary requirement so that it has to be supplied with the feed. Common feeds are fair to good sources of choline. Since choline is the component of lecithin and sphingo myelin, fats are good sources of cholline.

7.1.17. Panthotenic acid

It is widely distributed in nature so its deficiency symptoms are not to occur. It is the component of co-enzyme A. CoA is important in activating substances and pantothenic acid is used for this process.

7.1.18. Biotins

Biotin is a prosthetic group of several enzymes involved in the transfer of CO_2 from one substance to another. Propionyl CoA is converted to methyl malonyl CoA. That means biotin is important in the gluconeogenesis path way. A number of carboxilase require biotin. The important condition to mention here about biotin is that row egg which contains **avidin** which is inactivator of biotin, a protein that can react with biotin casing the deficiency problem. Otherwise biotin is widely distributed in common foods.

7.1.19. Cyanocobalamin B_{12}

B_{12} is involved in the methylnation of body molecules. It is required in the methylnation of cystine to methyline. It is required in the synthesis of cholline and in the synthesis of nucleic acid. It is involved in protein metabolism. Conditions that causes B_{12} deficiency is deficiency of cobalt. Cobalt is important in the synthesis of B_{12}. Feed or foods of plant origin are poor in B_{12}. Protein supplementation of animal origin and fermentation products are good sources of B_{12}. In general, all vitamin B and vitamin K are synthesized by microorganisms. As a result ruminants are not deficient in these vitamins. Its deficiency is important only for simple stomached animals.

8. ROLE OF MINERAL AND REQUIREMENTS

8.1. Essentiality of Minerals

In 1981, 22 mineral elements were believed to be 'essential' for the higher forms of animal life. These comprised 7 major or macronutrient minerals; calcium, phosphorus, potassium, sodium, chlorine, magnesium and sulphur and 15 trace or micronutrient mineral elements such as; iron, iodine, zinc, copper, manganese, cobalt, molybdenum, selenium, chromium, tin, vanadium, fluorine, silicon, nickel and arsenic. Since then, aluminum, lead and rubidium have been shown to be beneficial in some circumstances.

8.2. Mineral Nutrition of Livestock

Mineral elements exist in the cells and tissues of the animal body in a variety of functional, chemical combinations and in characteristic concentrations, which vary with the element and the tissue. The concentrations of essential elements must usually be maintained within quite narrow limits if the functional and structural integrity of the tissues is to be safeguarded and the growth, health and productivity of the animal are to remain unimpaired. Continued ingestion of diets that are deficient, imbalanced or excessively high in a mineral induces changes in the form or concentration of that mineral in the body tissues and fluids, so that it falls below or rises above the tolerable limits.

In such circumstances, biochemical lesions developed, physiological functions are affected adversely and structural disorders may arise, in ways which vary with the element, the degree and duration of the dietary deficiency or toxicity and the age, sex and species of animal involved. Homeostatic mechanisms in the body can be brought into play which delay or minimize the onset of such diet-induced changes. Ultimate prevention of the changes requires that the animal be supplied with a diet that is palatable and non-toxic and which contains the required minerals, as well as other nutrients, in adequate amounts, proper proportions and available forms. Large numbers of livestock in many parts of the world consume diets that do not meet these exacting requirements.

In consequence, nutritional disorders arise, which range from acute or severe mineral deficiency or toxicity diseases, characterized by well-marked clinical signs, pathological changes and high mortality, to mild and transient conditions, difficult to diagnose with certainty and expressed merely as unthriftiness or unsatisfactory growth, production and fertility. Mild deficiencies or toxicities assume great importance in the nutrition of livestock because of their extent and the ease with which they can be confused with the effects of semi-starvation due to underfeeding, protein deficiency and various types of parasitic infestation.

Inadequate or excessive intakes of a single mineral element are uncommon in most natural environments. They are often exacerbated or ameliorated, i.e. 'conditioned', by the extent to which other dietary components interact metabolically with the mineral. With copper, the question of mineral balance or dietary ratios is of crucial importance because of the potent influence of molybdenum and sulphur on copper retention, but it should be

recognized that metabolic interactions which significantly affect minimum requirements and maximum tolerances are widespread among the mineral elements. The incidence and severity of mineral malnutrition in livestock can be further influenced, both directly and indirectly, by climatic factors, such as sunlight and rainfall.

Sunlight promotes vitamin D formation in the animal, which in turn facilitates calcium and phosphorus absorption. The phosphorus concentrations in herbage plants fall with increasing maturity and with the shedding of seed. In any area, the relative lengths of the dry, mature period (low herbage phosphorus) and of the green, growing period (high herbage phosphorus) are determined largely by incidence of rainfall. Climatic or seasonal conditions thus influence the occurrence of phosphorus deficiency in grazing stock where appropriate remedial measures are not imposed. Heavy rainfall, resulting in waterlogging, also increases the availability of some soil minerals to plants, notably cobalt and molybdenum, so affecting the concentrations of those elements in the grazed herbage. These aspects are considered more appropriately in later chapters.

8.3. The Functions of Minerals

Four broad types of function for minerals exist: -

1. Structural function
2. Physiological function
3. catalytic and function
4. Regulatory function – although they are not exclusive to particular elements and many may be discharged by the same element in the same individual.

1. Structural function: - minerals can form structural components of body organs and tissues, exemplified by minerals such as calcium, phosphorus, magnesium, fluorine and silicon in bones and teeth and phosphorus and sulphur in muscle proteins. Minerals such as zinc and phosphorus can also contribute structural stability to the molecules and membranes of which they are part.

2. Physiological function: - minerals occur in body fluids and tissues as electrolytes, concerned with the maintenance of osmotic pressure, acid–base balance, membrane permeability and tissue irritability; sodium, potassium, chloride, calcium and magnesium in blood, cerebrospinal fluid and gastric juice provide examples of such functions.

3. Catalytic function: - minerals can act as catalysts in enzyme and hormone systems, as integral and specific components of the structure of metalloenzymes or as less specific activators within those systems. The number and variety of metalloenzymes that have been identified have increased greatly during the last two decades.

4. Regulatory function: - in recent years, minerals have been found to regulate cell replication and differentiation. Calcium, for example, influences signal transduction and zinc influences transcription, adding to long-established regulatory roles, such as that of the element iodine as a constituent of thyroxine.

In metalloenzymes, the metal is firmly attached to the protein moiety, with a fixed number of metal atoms per mole of protein. The metal cannot be removed without loss of enzyme activity and usually cannot be replaced by any other metal. However, the native zinc atoms in several zinc enzymes can be substituted by cobalt and cadmium without complete loss of activity and individual metalloenzymes are not always the domain of a single metal. The concentrations and activities of mineral–enzyme associations in particular cells and tissues have, in some instances, been related to the manifestations of deficiency and toxicity of those elements in the animal body.

In some cases, serious clinical and pathological disorders arise as a consequence of dietary mineral abnormalities which cannot as yet be explained in such biochemical terms. Two of the mineral elements, iodine and cobalt, are remarkable because, on present evidence, their entire functional significance can be accounted for by their presence in single compounds, thyroxine and vitamin B12, respectively; both compounds, and therefore iodine and cobalt, are nevertheless involved in a range of metabolic processes.

The functions of calcium and phosphorus are dominated so quantitatively by their requirements for the mineral base of the skeletal tissues that their manifold activities in the soft tissues and fluids of the body have been neglected by nutritionists concerned with dietary needs. These two minerals combine in the bones to provide strength, shape and rigidity, protecting the soft tissues and giving attachment to the muscles, while simultaneously forming a storage depot from which they can be mobilized for nutritional and metabolic emergencies, regulating the amounts in the blood and buffering against prolonged dietary inadequacies.

Regulation is vital, because calcium plays an important intracellular role involving cell signalling and an extracellular role in transmitting nerve impulses. Phosphorus participates in a wide range of metabolic reactions involving energy transfer. Every physiological event involving gain or loss of energy and almost every form of energy exchange in the cell include the making or breaking of high-energy phosphate bonds. In addition, phosphorus is an integral part of protein molecules and of the nucleic acids and their derivatives that are vitally concerned in cell replication and the transmission of the genetic code. The functions performed by minerals can only be fulfilled if the finite amounts ingested are sufficient to keep pace with the growth and development of the body and the reproduction of the species and to replace minerals that are ‘lost’, either as ‘harvested’ products or insidiously during the process of living.

8.4. Net Mineral Needs (Mineral Needs for Maintenance)

The basic maintenance requirement comprises the nutrients needed to keep intact the tissues of an animal which is not growing, working, reproducing or yielding any product. Body maintenance involves the performance of internal work in digestion, circulation, respiration and other vital processes, together with some external work in the ordinary movements of the animal. There is, in addition, a ‘productivity increment’, which contributes to the total maintenance requirement (M) because growth, work, reproduction or yield of a product increases the amount of food eaten and hence the amount of internal work done in utilizing the additional nutrients.

The difference between organic and inorganic nutrients in respect of maintenance needs stems from the different metabolic fates which the two types of nutrients experience. Organic nutrients enter metabolic pools, from which they are lost as heat, or converted to end-products of metabolism, simpler in form, which are excreted through the normal channels, i.e. no longer available as sources of energy or protein. In contrast, inorganic ions liberated in the course of metabolism are not changed and remain as available for the re-formation of their functional combinations as are the inorganic ions absorbed from the alimentary tract. Recycling is not complete, however, and there is an inescapable 'leakage' of minerals through the kidneys, intestinal mucosa, digestive glands and skin, which must be replaced.

Maintenance requirements vary appreciably for different elements. Endogenous losses of calcium and phosphorus, for instance, are substantial in the adult ruminant (approximately 6 g of each mineral for a 500 kg cow). On the other hand, sodium and chlorine are efficiently conserved, so that the adult maintenance requirements for these elements are usually extremely small, except in conditions of excessive sweating. Though small in amount, the maintenance requirement for manganese is probably of a similar order to that for growth at certain stages of development.

8.5. Faecal endogenous losses

For most elements and most situations, the major component of M is the endogenous loss of minerals via the faeces (FE). The amounts that are unavoidably lost are hard to measure if the faeces either serves as a route of excretion for a mineral absorbed in excess of need, as it does for phosphorus and manganese for example, or as a means of conserving a mineral during deficiency, as it does for sodium and iron. An early approach was to determine 'minimum endogenous loss' from the intercept of the regression of faecal excretion against intake for a given mineral, i.e. the faecal loss at zero mineral intake. Estimates of the maintenance requirement of ruminants for phosphorus (P) were reduced from 40 (ARC, 1965) to 12 mg P kg^{-1} live weight (LW) by the Agricultural Research Council (ARC, 1980) by adopting the minimum FE approach. Since no animal can survive for long without an essential mineral, animals need to do more than replace the minimum FE. However, failure to acknowledge that it is unnecessary to replace the entire FE, regardless of intake, leads to an overestimation of M. The most useful value is that FE which occurs at the minimum mineral intake needed to sustain optimal (usually maximal) production or zero balance in the case of non-producing livestock. Given that replenishment of skeletal phosphorus reserves during the non-productive dry period is vital for sustaining the next lactation, it is important to define M accurately for this element. Furthermore, the maximal efficiency with which a mineral can be absorbed from the gut cannot be ascertained unless the flow of mineral in the opposite direction, i.e. FE, is known.

8.6. Influence of minerals on food intake

Maintenance requirements were expressed on a body-weight basis, but there is now evidence that, for calcium and phosphorus at least, M for ruminants is a function of food intake (AFRC, 1991). The relationship with food intake is to be expected, given that FE is derived from sloughed mucosal cells and unresorbed digestive secretions, and it would be surprising if food intake does not also affect M for most minerals. Relationship to food intake rather than body weight will mean that M is relatively high at times of high food intake, such as lactation, and when diets of low digestibility are fed: in contrast, M should fall at times of low food intake, except for periods of in appetence caused by heat stress, when more minerals are lost in sweat.

8.7. Mineral Needs for Work

With grazing stock, movement may raise maintenance needs for energy by 10 – 20% or more above those of the animal at rest. With working animals, such as horses or bullocks used for draught or transport purposes, the energy requirements can be increased several times above maintenance. The extra food required to meet these requirements will usually supply the animal with sufficient additional minerals, even the cow, which is increasingly used for draught purposes, except perhaps for sodium. Hard physical work, especially in hot conditions, greatly increases sweating and therefore losses of sodium and potassium; the dietary requirement for salt is thereby raised. No other specific mineral requirements for physical work have been reported. No significant change in calcium or phosphorus balance or in losses of these minerals from the body was observed in horses performing light, medium or hard farm work, relative to those of the same horses performing no such work. However, there is a growing belief that increased consumption of oxygen during exercise leads to increased requirements for elements, such as selenium, which are involved in antioxidant defense.

8.8. Net Mineral Requirements for Reproduction

The mineral requirements for reproduction (R) in mammals are usually equated to the mineral content of the fetus and products of conception (placenta, uterus and fetal fluids) and therefore increase exponentially to reach a peak in late gestation. For the twin-bearing ewe, the calcium requirement in late gestation actually exceeds that of lactation, leading to important contrasts in the period of vulnerability to calcium deprivation when compared with the dairy cow. There is also a small additional requirement for growth of mammary tissue and the accumulation of colostrum prior to parturition.

8.9. Net Mineral Requirements for Production

The net mineral requirements for production (P) are given by the mineral content of each unit of production, such as weight gain (WG), milk yield (L) or fleece growth (F), and are usually taken to remain constant. However, for elements such as calcium and phosphorus, which are far richer in bone than in soft tissue, requirements for growth diminish as animals mature, because bone makes a progressively smaller contribution to each unit of live-weight gain. Mineral requirements for production are affected by the species or breed of animal, the intensity or rate of production permitted by other constituents of the diet – notably energy – and by the environment.

Chicks and weaned pigs consume similar types of diet, but the faster-growing broiler chick requires nearly twice the dietary concentrations of calcium and manganese required by pigs. High-yielding dairy cows obviously require much more dietary calcium and phosphorus than low-yielding cows, because of the richness of milk in these elements. However, the levels necessary in the dry ration do not rise, because total dry matter (DM) intakes increase with rising productivity of the cow as rapidly as do mineral requirements. The phosphorus requirements of hens tend to follow a similar pattern with onset of egg production, remaining a constant proportion of the diet, but calcium requirements greatly increase. A non-laying hen can normally meet its calcium (Ca) needs from a diet containing 5 g Ca kg^{-1} DM, whereas some eight to ten times this concentration is necessary for a hen laying one egg per day. Mineral intakes must ideally be sufficient to ensure the maintenance of adequate mineral reserves of the body tissues and adequate amounts in edible products.

The animal body can sometimes adjust to suboptimal intakes by reducing the amount of a mineral in its products. Thus quality, such as the shell strength of eggs and the tensile strength of wool fibres, may be reduced in order to maintain other more essential functions. This is clearly undesirable, so that assessment of mineral needs usually includes determination of the minerals in the tissues, fluids and products, as well as such gross criteria as weight gains, milk yields and so on. Milk is an exception, in that normal mineral concentrations are usually maintained during deficiency, priority being given to the mineral nutriture of the new generation at the expense of the mother. Where the provision of excess mineral increases concentrations in milk (e.g. iodine), it is unnecessary to allow for replacement of the entire secreted mineral.

8.10. Gross Mineral Requirements

Net mineral requirements underestimate the dietary needs of livestock for minerals, because ingested minerals are incompletely utilized, due to limits upon their absorption from the gut. A basic or minimum requirement for any mineral can be conceived as one in which all the dietary conditions affecting that mineral are optimum. Since these exacting conditions rarely apply, there can be no single requirement but rather a series of requirements, depending on the extent to which ‘conditioning’ factors are present in a particular grazing or ration. By the same reasoning, there must be a series of maximum ‘safe’ dietary levels, depending on the extent to which other minerals or compounds are affecting the absorption, retention and excretion of a mineral consumed in excess of need.

The chemical form of the mineral acquires particular nutritional significance for non-ruminant livestock, such as pigs and poultry. For example, phytate phosphorus and zinc bound to phytate are poorly absorbed in non-ruminants but are well used by sheep and cattle. Physical form may be important for mineral supplements, such as magnesium oxide, which can vary in particle size and hence in nutritive value.

Ideally, mineral supplements should be used only when requirements cannot be met with adequacy and safety by the judicious selection and combination of available feeds alone. However, this requires knowledge of the mineral composition of feeds, and success depends heavily on the appropriate database being available. The addition of protein concentrates to a grain mixture raises its content of such minerals as calcium, phosphorus, zinc and iodine. Bran is freely available on many farms and an excellent source of phosphorus for ruminants. While the substitution of a plant protein source for animal products, such as meat-meal or fish-meal, can result in lower availability of some minerals for pigs and poultry, notably of zinc and phosphorus, because of the presence of fibre and phytate, bran may be beneficial because it contains phytases.

In practice, mineral supplements are added routinely to home mixed and commercially compounded rations as an insurance against the inclusion of components which deviate from the norm of mineral composition. In some circumstances, mineral supplements are always necessary, because the pastures or feeds are abnormal in mineral composition as a consequence of local soil and climatic effects. A wide range of inorganic mineral supplements, covering all essential minerals are now available and are increasingly used to fortify rations because of increasing rates of animal production, decreasing availability and acceptability of animal by-products in feed formulation and increasing use of industrial products, such as urea, which replaces protein in feeds for ruminants without providing minerals. Mineral supplementation is an essential adjunct to urea supplementation and plays a vital role in increasing the nutritive value of low-quality roughages and crop by-products in developing countries.

Minerals in general are classified into two major groups depending on the concentration in living organisms. Some minerals required (present in the living organism) in a relatively higher concentrations and known as major or macro minerals. Some others which are required in trace amounts are referred to as micro minerals (micro elements). Those once which are expressed in percentages are called macro or major (0.01-2% - 5%. On the other hand, trace minerals those that are found in very low concentration and their unit is ppm, mg/kg proportions relative to million units. e.g. 10 ppm is equal to 10 in one million.

- ✓ **Major minerals →** Ca, P, Na, K, Cl, Mg, and S.
- ✓ **Trace elements →** Fe, Zn, Mn, Cu, Co, Mo, Se, I, F, Cr.

Both of them are important but some are more important in practical conditions. Ca, P, Na, Mg, and Cl.

Calcium is a predominant constituent of skeleton and teeth and 99 % of Ca in the body is found in skeletal system and teeth. It is also essential for the transition of nerve impulses and for the contractile properties of muscles. In addition to this it is involved in the coagulation of blood, plays part in cell membrane permeability. The Ca level in blood is fairly constant in farm animals and it ranges from 90 - 120mg/lt of blood. Conditions that can cause Ca deficiency are: -

1. Cereal and root crops are poor sources of Ca.
2. Some mineral does also interact with Ca rendering it the potential not to be available easily e.g. phosphorus.
3. Deficiency of vitamin D can also cause Ca deficiency.
4. High milk production in dairy cows in the early lactation and in late pregnancy periods. The cows tend to give priority to the formation of the fetus in late pregnancy and to the production of milk to the early lactation (after parturition). This causes health problem known as **milk fever**. This disease can occur in high milk producing cows the 1st few days after parturition. This problem is characterized by muscular weakness paralysis and unconsciousness. Rickets and osteomalecia can also occur. Sources of Ca are **milk, grain feeds, bone meal and lard.**

8.10.1. Phosphorus (P)

It is important in bone formation and about 88% of P is found in the skeletal system. They are components of phosphor - proteins, nucleic acids, phospholipids, and also important component of AMP, ADP, and ATP. Some of the conditions that cause deficiency problems of P in grasses of tropical countries are the formation of **Phytic acid** that forms complexes with **Ca and Mg.** This reaction makes /gives /or renders P to be un available to the animals. Milk, cereal grains, fish meal, and meat meals are good sources of P. The deficiency of P can adversely affect appetite causing abnormal appetite. Animals **chewing/ gnaw** bones, woods, rags, are signs of deficiency.

8.10.2. Sodium (Na), Potassium (K), and Chlorine (Cl)

The functions of these substances are closely related. They are involved in the regulation of osmotic pressure of body fluids, maintain acid-base balance. Na and K are important in nerve and muscle excitability and Cl is required for HCl synthesis in the digestive system. Na and Cl are not adequate in most food stuffs. So we have to add certain proportion of salts usually up to 1%, Whereas, K is excess in most of the food stuffs. In the cases of Cl and Na deficiencies appetite may be lost and the animal become weak and unhealthy. On the other hand, excess amount of salt can be toxic.

8.10.3. Sulfur (S)

Sulfur is not a problem in most of the feed stuffs because it is found in amino acids such as methionine and cystine.

8.10.4. Magnesium (Mg)

70% of magnesium is found in the skeletal system.The rest of it is distributed to the soft tissue of the body. Besides the constituents of bone and teeth it is a common enzyme activator. The condition that may increase Mg deficiency is known to be the application of high level of nitrogen or fertilization of range lands. This deficiency is known as ***hypomagnesaemia*** or ***hypomagnesium tetani.*** This problem causes inadequate absorption of Ca in the digestive

tract. ***hypomagnesium tetani*** is characterized by nervousness, twitching of the facial muscles, staggering gait, and unconsciousness. Death may occur due to this problem.

Critical Minor Minerals (trace elements)

8.10.5. Iron (Fe)

Most of the Fe in the body found bound with proteins, such as blood proteins like hemoglobin and cytochromes in the electron transport chain also contain Fe. Most of the function of Fe is related to respiration. A condition that may cause Fe deficiency in young animals is due to the low content of Fe in milk. The deficiency problem of Fe is anemia and related conditions.

8.16.6. Copper (Cu)

Copper is required for the formation of haemoglobin. It is the component of plasma proteins that is responsible for the release of Fe from different cells in to the plasma. It is also important component of erythrocoprine protein which is a part of red blood cells or erythrocytes. And it is a component of cytochrome oxidase. The function of Cu and Fe is required for the pigmentation of hair, fur, and wool. Deficiency symptoms are depigmentation and anemia, bone disorders are seen, loss of motion, and infertility can also resulted. Cu deficiency can be observed in two conditions.

1. If the soil is low in Cu content.
2. When the areas are adequate in Cu content but contain excess amount of molybdenum and/or sulfur, because excess of these minerals form complexes with Cu and depresses its availability. The areas that can be taken as an example of these problems are Debre Zeit and Nazareth.

8.10.7. Cobalt (Co)

It is important because **anemia** and **emaciation** in cattle and sheep have been observed for many years due to the deficiency of these minerals. They are sometimes called as:

- ✓ 'Pining' $\Longrightarrow$ Whipe-worm - animals were excessively emaciated.
- ✓ Or 'wasting' disease $\Longrightarrow$ this shows that it can cause heavy weight loss.
- ✓ 'Salt sick' $\Longrightarrow$ when salt is provided, animals recover due to the presence of some amount of Co in salt. Co is required by microorganisms in the rumen for the synthesis of (B_{12}) cyanocobalamine.

8.10.8. Iodine (I)

It is required for the synthesis of thyroxin. Thyroxin is a thyroid hormone that is involved in enhancing basal metabolism and accelerating growth and also increases respiratory process. The deficiency cause of iodine is **goiter**,

and affects **normal birth of kids**. A characteristic of goiter is swelling of the neck, reproductive failure, and birth of hairless offspring / weak or dead fetal in animals. Thyroxin is also used in metabolic activities.

8.10.9. Conditions of deficiency:

Iodine deficiency is seen in areas a bit far from seas. Deficiency could be due to dietary deficiency or due to goitrogenic compounds suppressing the functions of iodine. The functions that suppress iodine are foods of brassica family – kale, cabbage, rape, soybean, linseed, peas, and ground nut. Excess amounts of iodine can also cause goiter.

8.10.10. Manganese (Mn)

It is an important enzyme activator. Deficiency can result in fertility problems, can induce skeletal abnormalities. It can cause perosis in chicks slipped tendon. The condition that causes this problem is that maize is low in this element and most foods of animal origin are poor sources. Green plants and bran are good sources.

8.10.11. Zinc (Zn)

Zinc is used as enzyme activator. The deficiency can cause infertility particularly in human beings. Dermatitis is caused by parakeratosis => characterized by reddening of the skin. Skin lesion can be observed if people have fair red color and reddening of the skin can be seen.

$\Longrightarrow$A kinds of dry diet or lack of water could cause Zn deficiency and can be aggravated by high level of Ca in the diet; otherwise it is widely distributed in nature.

8.10.12. Expression of Requirements

The requirements of animals for minerals can be expressed in several ways:

1. in amounts per day or per unit of product, such as milk, eggs or
2. weight gain; in proportions, e.g. percentage, parts per million (ppm), mass mass^{-1} (e.g. mg kg^{-1}) or moles (sometimes micro - or millimoles) kg^{-1} DM of the whole diet.

Proportions have the merit of simplicity and have obvious practical advantages, so long as the total diet is palatable, but they are of limited value when the regular *daily* intake of large amounts of mineral is essential, as in the case of calcium for the laying hen. Whether expressed as amounts or concentrations, requirements can be greatly influenced by factors that limit the absorption and utilization of the minerals in question.

9. METABOLIC DISTURBANCES

They are diseases of livestock caused by productivity practices in which the normal metabolic balance is disturbed. These are important diseases wherever livestock are subjected to high levels of productivity. Thus they are of little or no importance for any farmers in tropical countries.

9.1. Pica/hypophosphatemia in the tropics

A dietary deficiency of phosphorus, if sufficiently severe or prolonged, leads to abnormalities of the bones and teeth, subnormal growth, milk yield and egg production reduction, depressed appetite, poor efficiency of feed use and the development of pica or deprived appetite and fertility may be impaired.

The loss of appetite caused by phosphorus deprivation is often paralleled by a craving/desire for and a consumption of abnormal materials, such as soil, wood, flesh and bones. The deprived appetite (pica) may either take a generalized form, known as **allotriophagia**, or it can be expressed more specifically as osteophagia (craving/desire for bones) and as **sarcophagia (craving for flesh**). These forms of pica are not specific to phosphorus deficiency, since they have been observed in animals suffering from lack of sodium and potassium and also in sheep receiving insufficient energy and protein under field conditions. Bone-chewing in cows has been induced by exteriorizing the parotid salivary duct and feeding a low-phosphorus diet, thus preventing the recycling of phosphorus. A complete blocking of the drive to eat bones was achieved within 1 h by intravenous infusion of sodium phosphate sufficient to raise serum P ion to normal.

Pica, whatever its cause may be, can be disastrous in areas where the carcasses are infected with *Clostridium botulinum*, and toxin formation during the process of putrefaction can cause botulism and death. Sheep and cattle from botulism (toxic paralysis) have been reported from several parts of the world, due to the widespread occurrence of *C. botulinum* and of dietary phosphorus deficiency. The animal can be protected against botulism by vaccination, but the problem of aphosphorosis remains unless remedial measures are taken.

9.2. Ketosis/Acetonemia

This occurs in high yielding cows within a few weeks of calving, usually in housed cows fed conserved fodder. Over several days there is loss of body condition and slight drop in milk yield followed by a sudden drop in appetite and milk yield. Loss of condition is accelerated, affected animals are depressed and the sweet smell of acetone can be detected on the animals breath or tasted in the milk. Feaces tend to be firm and covered in mucus, and some animals have variable nervous signs such as salivation, chewing, incoordination, blindness and aggression. As the underlying cause is high milk yield, the drop in milk yield results in eventual recovery from the clinical signs.

9.3. Hypocalcemia/milk fever

This disease occurs in high yielding dairy cows one or two days befor or after calving. The first sign is loss of appetite and a slight drop in temperature. Affected cows quickly become uncoordinated, fall over and, after attempts

to rise, stay seated often with the head resting on the shoulder. If untreated the cow becomes comatose and dies within one day of the first signs. The rumen stops functioning and blot may be complication and the final cause of death. The risk of milk fever increases with age, it is rare in calving heifers and uncommon in cows calving for the second time.

9.4. hypomagnesaemia (*grass staggers; grass tetany; milk tetany in calves*)

This occurs most commonly in high yielding dairy cows around peak lactation that are grazing lush grasses, and in calves reared predominantly on a diet of milk. Peracute causes suddenly develop staggers, fall over in convulsion, paddle their legs frantically and froth at the mouth. the heat pounds dramatically and death quickly follows. Potassium fertilizer should not be applied in the spring as it is associated with reduced magnesium availability to cattle, and may trigger hypomagnesaemia. It is needed less for grazed crops than silage, because of the return of potassium in excreta in the grazed sward. The low magnesium content of rapidly growing grass swards as well as the low availability of the magnesium often present difficulties on farms where the cows are susceptible to hypomagnesaemia.

10. OTHER METABOLIC DISORDERS

Changes to the reticulo-rumen environment can influence the metabolism of the micro-organisms present, resulting in disease conditions, such as acidosis, bloat and nitrate poisoning for the host.

10.1. Acidosis

Lactic acidosis is a common metabolic condition observed when ruminants are overfed large amounts of grain or other rapidly fermented carbohydrates which result in the excessive production and accumulation of lactic acid in the rumen. In conditions where there is an ample supply of highly fermentable carbohydrate, rumen pH continues to decline resulting in a change in the rumen microflora from predominantly gram-negative to gram-positive lactic acid-producing bacteria and also reduced rumen motility. One consequence of a decrease in motility is a decrease in rumination and less production of saliva leading to a reduction in the buffering capacity of the rumen. Eventually, the bacteria that utilize lactic acid are outnumbered by those that produce it which leads to the accumulation of lactic acid in the rumen. The bacteria mainly responsible for lactic acid production in the rumen are *Streptococcus bovis* and *Lactobacillus* Spp. Organisms like *Megasphaera elsdenii* and the *selenomonads* are major lactate utilizers and are inhibited by low pH. The change in microbial populations can be very rapid and occur within a 24-hour period. Several pathological changes in animals are associated with the onset of ruminal lactic acidosis and some of these changes are directly related to changes in the microbial population in the rumen. Excessive grain intakes increase the concentrations of endotoxins in the rumen due presumably to the disintegration of the gram-negative bacterial cells. The large amounts of lactic acid and VFA present in the rumen cause an osmotic flow of water from blood into the rumen, and affected animals are become dehydrated. The hypovolemia may subsequently lead to circulatory shock.

Adapting the rumen to increasing concentrations of grain can reduce the incidence of acidosis. Usually the numbers of bacteria which produce lactic acid increase with the introduction of concentrate into the diet. Simultaneously, the numbers of bacteria which metabolize lactic acid also increase and the accumulation of lactic acid in the rumen is avoided. With time, the numbers of lactic acid producing bacteria decrease and the rumen ecosystem returns to a stable condition. In other words, gradual adaptation allows the simultaneous growth of both lactate-producing and lactate-utilizing microbial populations. Generally, such conditions are largely avoided if coarse particle size concentrates are fed and microorganisms are given time to adapt to concentrate over a 3 to 4 week period during which increasing amounts of concentrates are substituted for forage at 5- to 7- day intervals.

The antibiotic Virginiamycin prevents lactic acid accumulation in both the rumen and hindgut, even when high starch diets are fed to animals without prior adaptation. It has been also reported that during a challenge with highly fermentable carbohydrates, addition of *Megasphaera elsdenii* B159 prevented an accumulation of lactic acid and shifted ruminal fermentation away from acetate and propionate towards butyrate and valerate. Addition of anti-microbial agents such as ionophores is also effective in limiting ruminal dysfunction associated with the proliferation of lactic-acid producing bacteria with high-grain diets. These compounds are believed to inhibit Gram-

positive lactate producing bacteria like *Streptococcus bovis.* Ruminal bacteria that synthesize propionate tend to be Gram-negative and resistant to ionophores. Accordingly, the reduction in lactate accumulation within the rumen results in increased pH and hence reduced the problems associated with acidosis.

Undoubtedly, genetic adaptation by rumen microorganisms is what enables ruminants to acquire increased tolerance to toxins. Presumably, microorganisms shift their metabolism from substrates that are commonly available in the rumen towards toxic substrates which are occasionally available for metabolism. The highly competitive nature of the rumen environment is such that organisms that fail to adapt to a particular environment are quickly overcome by those which are more "fit" for the digestive process. Only rarely, however, are these less "fit" microorganisms completely eliminated from the rumen. Thus, the genetic diversity of rumen populations is maintained in anticipation of future changes in the rumen environment.

10.2. Bloat

Bloat can be observed as an acute swelling between the last rib and the hip on the left side of the cattle, from behind. The cow is restless, finding lying uncomfortable and may eventually die of heart failure or suffocation as a result of inhaling rumen contents. Pasture bloat is caused by **stable foam** in the rumen caused by the rapid digestion of legumes, in particular, although young leafy grass that has recently **received nitrogen fertilizer** can also cause bloat. Lucerne is the most likely of all legumes to cause bloat, with cows sometimes dying within a few hours of entering a field for grazing.

Some legumes have developed a chemical, tannin, which reduces the speed of protein digestion and probably discourages animals from grazing it. They are present in sufficient quantities in birdsfoot trefoil to prevent the production of stable foam, and the content in white clover increases sufficiently at flowering to make it safe to graze. If a mixed grass and clover sward has enough clover to cause bloat (probably more than 50% of the herbage by mass), it should not be grazed for long periods, but should either be conserved if there is sufficient mass, or it should be rested for a few weeks until the clover inflorescences appear, after which it can be grazed or conserved.

Cows are most likely to become bloated in the late evening after a day's grazing and also after a wet period when they avidly/eagerly graze to make up for lost time. Wet grass reduces saliva production, and the saliva contains a mucin that disperses foam in the rumen. Herbage that has been frozen is particularly likely to cause bloat as the rupture of plant cell walls releases the solutes that contain much potassium. Potassium-rich feeds, such as molasses, are well known for causing bloat, whereas grasses rich in sodium appear to be less likely to cause bloat. The precise mechanism has not yet been determined but may relate to the stimulation of saliva production by sodium-rich feeds, and the foam-dispersing properties of the salivary mucin.

Forage supplements will usually slow down the rate of digestion and reduce bloat, but if there is adequate herbage, grazing supplements may not be eaten by some cows in sufficient quantities, particularly if they are based on straw

or other low quality forages. Mineral oils also help to disperse the foam and can be added to a concentrate feed or sprayed onto the pasture or the cows' flanks, to be licked off as needed. Linseed oil is often used. A proprietary product, **poloxalene**, also breaks up the foam and can be used as a drench for clinical cases or included in feed blocks as a preventative measure.

Often simply walking the cow from the field to the farm steading to receive medication will alleviate the swelling. It is important to keep a bloated cow on her feet if possible, as death can follow soon after recumbency. There is undoubtedly a genetic component in the susceptibility of cattle to bloat. There are reported breed differences in susceptibility. Jersey cows are particularly prone to the disorder. Cows can get used to feeds that are liable to make them bloat; this may be by altering their behavior to spread their meals out more evenly over the day. Lactating cows are particularly susceptible because of their high intakes.

Pasture bloat remains a serious problem for farmers in countries, where the cattle rely on pasture with little or no fertilizer applied and a high legume content. In Europe, the greater emphasis on controlling nitrogen emissions may encourage farmers to use high clover swards for their cattle, potentially leading to more serious problems with bloat.

10.3. Plant Aspects of Bloat

The compound in legumes which causes the foam build up is not known for certain. Many feel it is some type of soluble leaf protein but differs on which protein fraction is responsible. Other plant compounds which have been reported to influence bloat are saponins and pectins. Legume species vary in their ability to cause bloat. Alfalfa, ball clover, annual medics, white clover, and Persian clover are considered to have high bloat potential; red, crimson, and subterranean clovers have medium potential; and berseem clover and arrow-leaf clover have low bloat incidence.

A high tannin level in arrow leaf clover is thought to be responsible for its low incidence of low bloat. However, all can cause bloat and should be managed properly. Birds foot trefoil, sainfoin, crown vetch, and most tropical legumes are non-bloating legume species. Livestock are most likely to bloat on clover pastures in the early spring. One theory is that the warmer day temperatures increase photosynthesis or the synthesis of carbohydrates and proteins. However, the night temperatures at that time are still cold which slows the breakdown process of some of the carbohydrates and proteins which occurs at night. The net result is the buildup of carbohydrates and proteins, one of which is the soluble leaf protein which is believed to cause bloat.

10.4. Animal Aspects of Bloat

Cattle may bloat after grazing clovers for only 2 hours or for as long as 2 weeks. Within a given herd, some animals will bloat and some will not. Selecting against bloat susceptibility is possible because of bloat prone. Families of

dairy and beef cattle, differences in the bloating potential of cattle breeds, and transmittal of bloating tendencies to offspring. Many producers feel that Brahman cross cattle are less likely to bloat than non-Brahman cattle. Some individuals are chronic bloaters and should be culled. Possible reasons cited are differences in (1) salivary flow and composition, (2) grazing behavior, (3) feed intake, (4) lower rates of gas production in the rumen, (5) conditions in the rumen unfavorable to persistent foaming, (6) physiological responses to tactile stimulation or stretch of the reticulorumen walls, and (7) anatomy. Grazing studies in Louisiana have shown wide variations in bloat severity for the same animals from morning to afternoon grazing periods of the same day, as well as variations in bloat severity of the same animals after corresponding grazing periods from day to day. This further demonstrates that the cause of legume bloat is quite complex.

10.5. Bloat Prevention

Care must be taken when first turning cattle on to lush legume pastures in the early spring. The drastic change in diet from dry hay with 6 to 10% protein to young clover with over 25% protein (dry weight basis) and a moisture content of about 85% is a shock to the microflora and protozoa in the rumen. A transition period of 1 to 2 weeks where livestock have access to both hay and legume is helpful. This can be accomplished by allowing the animals to graze clover for an hour or two a day while receiving hay or providing hay on the clover pastures. In any case, animals should never be placed on lush legume pasture with an empty rumen.

Pasture management practices for reducing the incidence of bloat center on not allowing young succulent legumes to constitute the total diet of livestock. Utilization of grass-legume mixtures instead of pure legume pastures is the most desirable option in terms of cost and labor to reduce frothy bloat. Bloat is very rare on grass-legume pastures when legumes constitute 50% or less of the available forage. The most economical grass-legume mixtures are seeding from 15 to 20 pounds of ryegrass per acre with the legume in the late fall. Cost will be from $5 to $7 per acre for seed plus planting expenses. Besides preventing bloat, adding a grass will provide earlier grazing that will further reduce overwintering costs of the cow herd.

If clover constitutes the major portion of the available forage in a pasture, hay can be fed. An alternative is to limit graze the pasture several hours a day or use a portable electric fence to strip graze a small portion of the pasture each day. Here again, hay should be fed free choice.

10.6. Nitrate Toxicity/Poisoning

Nitrate itself is not overly toxic to animals; however, when nitrate is reduced to nitrite by rumen microflora, it becomes very toxic. Under normal conditions, nitrate ingested by ruminant livestock is converted to ammonia and then bacterial protein in the rumen by bacteria. Nitrate toxicity is a function of the amount and rate at which nitrate is consumed. Hence, when higher than normal amounts of nitrate are consumed, an accumulation of nitrite may

occur in the rumen. The main hazard to ruminants is ingestion of plants that have accumulated excessive amounts of nitrates or nitrites. Excess nitrite then will be absorbed and combined with blood hemoglobin to form met-hemoglobin, which impairs the oxygen-carrying capacity of red blood cells. In other words, the nitrite oxidizes iron in hemoglobin from the ferrous (+2) to ferric (+3) state. The resultant methemoglobin has a very poor affinity for oxygen which greatly reduces the oxygen-carrying capacity of red blood cells. Readily fermentable carbohydrates, lactate and hydrogen are sources of electrons for the reduction reactions and facilitate nitrite production. If the physiological capacity of the microbes to carry on the reduction reactions is not exceeded, nitrate and nitrite reductions increase simultaneously due to an adaptation of the rumen microorganisms to those diets. Drought conditions and the usage of manure as fertilizer heighten nitrate accumulation in cereal grasses found in pasture.

10.7. Anti-nutritional factors/ poisonous plant problems

Plant secondary metabolites are a diverse group of molecules that are involved in the adaptation of plants to their environment but are not part of the primary biochemical pathways of cell growth and reproduction. In general, the terms *plant secondary compounds, phytochemicals, antinutritional factors*, and *plant xenobiotics* have been used in the literature to refer to this group of compounds. There are well over 24,000 structures, including many compounds that have antinutritional and toxic effects on mammals. This number does not include the oligomeric polyphenolic compounds (Proanthocyanidins and hydrolysable tannins) that are just now being more accurately described and will increase the number by several thousand. Some major plant secondary metabolites or phytochemicals that occur in plants include **protease inhibitors, lectins, alkaloids, nonprotein amino acids, cyanogenic glycosides, saponins, and tannins**. These compounds are involved in defense against herbivores and pathogens, regulation of symbiosis, control of seed germination, and chemical inhibition of competing plant species (allelopathy), and therefore are an integral part of the interactions of species in plant and animal communities and the adaptation of plants to their environment.

Toxic plant secondary metabolites are present in plants at low concentrations (generally less than 2% of the dry matter) and have negative physiological effects when absorbed, such as **neurological problems**, **reproductive failure**, **goiter**, **gangrene**, and **death**. Examples are **alkaloids**, **cyanogenic glycosides**, **toxic amino acids**, **saponins**, and many others. Nontoxic phytochemicals lower digestibility of nutrients and affect palatability. Higher concentrations (>2% of DM) of these compounds are required for inducing negative effects, and the primary site of activity is in the digestive tract or the sensory organs associated with feeding behavior. These plant secondary metabolites include **tannins, protease and amylase inhibitors**. Compounds that have a structural role in the plant (e.g., lignin, biogenic silica, and cutin) lower the extent of microbial digestion of cell wall polysaccharides.

This division between groups of plant secondary metabolites is not exclusive. For instance, hydrolysable tannins are potentially toxic to ruminants. The major lesions are hemorrhagic gastroenteritis, necrosis of the liver, and kidney damage with proximal tubular necrosis. Excessive and fast consumption of oaks and other tree species that contain

more than 5% hydrolysable tannins results in high mortality and morbidity in cattle and sheep. Contrary to this, plant secondary metabolites are also associated with improved nutritive value and may have beneficial effects on animal health.

Proanthocyanidins, more commonly called **condensed tannins** are associated with improved protein digestion and metabolism in ruminants and in protecting ruminants against legume bloat. Tannins may also protect ruminants against helminthiasis. Growing interest in the potential health-promoting effects of plant secondary metabolites in human foods has prompted research on their potential to prevent or **treat cancer**, **circulatory disease**, and **viral infection**. The mechanisms by which these substances have beneficial effects on health may also be related to their toxic effects, and the difference between toxicity and beneficial effects may be dose- and structure-dependent. However, mechanisms of toxicity and health-promoting effects of most of the plant secondary metabolites in human and animal diets are not well established.

Interest in plant secondary metabolites has risen dramatically in recent years among plant molecular biologists and plant breeders because of their diverse effects, which, in addition to those mentioned above, include **antioxidant**, **antiviral**, **antibacterial**, and **anticancer** effects. To name a few recent developments, molecular biologists have made genetic modifications in Proanthocyanidins biosynthesis in forage plants with the aim of eliminating bloat, improving the efficiency of conversion of plant protein into animal protein (increase rumen undegradable protein and thus increase protein availability postruminally), **reduce greenhouse gases** and **reduce gastrointestinal parasites**; and plant breeders have developed and commercialized rapeseeds (canola) with low levels of **glucosinolates** and **erucic acid**, and **cottonseed** with low **gossypol**.

Genetically, modified rice, which expressed insecticidal cowpea trypsin inhibitor, has also been produced. The emerging molecular genetic approaches have tremendous potential to unravel the regulatory genes that control plant secondary metabolite biosynthesis. This information, together with increased knowledge of the enzymes specific for the pathway, could facilitate the genetic engineering of plants.

Most of the plant resources, especially in the tropical regions, are rich in plant secondary metabolites, and the lack of information on the appropriate methods for their determination has been the main bottleneck in better understanding of the enzymes and biochemical pathways in their synthesis, the genes responsible for controlling major biochemical processes, and the physiological significance of plant secondary metabolites, and in exploiting the beneficial effects of these phytochemicals.

10.8. Toxins in animal feeds

The various feed ingredients should be analyzed for the toxins present in them which are otherwise injurious to the health of animals. The examples of toxins in the various feeds are given below.

1. Gossypol in cotton seed
2. Hemagglutinins in soybean and castor beans.

3. Glucosinolates in rape seed.
4. Tannins in sorghum, oil seed meals, mango seed kernels, mustard oil cakes and Lucerne meal.
5. Cyanogenic glycosides
6. Phytic acid in all cereals, oil seed meals.
7. Mycotoxins, primarily aflatoxins in maize, ground nut cake, etc.

Ultra violet screening is used whereby a greenish yellow fluorescence is observed when the sample is exposed to ultra violet light to detect mycotoxins. One should get from the best source of supply and one should have some idea of normal levels of toxicity which may be expected. Fish meal, meat meal and bone meal should be checked for pathogenic bacteria like Salmonella.