the amount of bacterial cells is less. However, the production of VFAs, methane and heat increases (Y_{atp} =8). The incremental heat reduction that occurs with increasing efficiency of microbial production has a positive effect on feed intake intake. Not only is heat production lower but the increased P/E signals a more balanced rumen, resulting in increased digestibility of the fibrous material (see Fig. 20).The protein to energy ratio is higher with higher Y_{atp} values that occur in a balanced rumen. Conversely in the unbalanced rumen due to low rumen ammonia concentration, Y_{atp} values are low as is the P/E ratio (Preston and Leng, 1987).

The implications for production of cattle in the hotter and or humid areas are profound because, without supplements, the total dry matter intake may also be decreased as a thermo-regulatory mechanism to lessen the effect of environmental heat. This may also have implications for high producing cattle such as dairy cows, in hot/humid areas under reproductive or lactation stress, grazing fibrous pastures.

The amount of energy produced by hind gut fermentation can be considerable and should not be under-rated but is not considered here.

6 PROTEIN – NITROGEN METABOLISM

6.1 Overview

Protein ingestion and availability to the animal is intimately associated with carbohydrate fermentation in the rumen but, for the purpose of discussion, it will be examined separately.

True protein when ingested by the ruminant is initially subjected in the rumen to microbial extra-cellular protease and peptidase action. This produces peptides, some of which are degraded further to amino acids in the bacteria. These are either used as such for microbial protein synthesis or are fermented to ammonia and VFAs. The VFAs are mainly discharged from the bacteria as with the ammonia, but some ammonia is used intra-cellularly to synthesise new amino acids that are then used by the bacteria. The ammonia released from some microbes into the rumen fluid may be taken up by others and converted into bacterial protein. When the bacteria or the undegraded dietary protein pass down into the abomasum

further enzymatic digestion of the protein takes place, splitting it into its component peptides and amino acids. Absorption of the amino acids takes place in the small intestine.

The reactions that take place in rumen and involving the micro-organisms, enable the animal to be given a source of nitrogen other than protein which when converted to ammonia is further transformed into bacterial protein and this then can be utilised in the small intestine as true protein.

6.2 Non-protein nitrogen

An extensive review of the historical use of non protein nitrogen (NPN) use in ruminants has been published in 'Nonprotein Nitrogen in The Nutrition of Ruminants', Loosli and McDonald (1968). The suggested use of NPN as food sources dates back to 1891 when Zuntz suggested that rumen microflora were able to breakdown cellulose as a source of energy and convert NPN into true protein. World War 1 caused a scarcity of vegetable proteins for animal feed in Germany and stimulated research into urea synthesis and its use in ruminant feeds. Krebs reviewed this work in 1937. Research in the United Kingdom and the USA around this time also supported the idea that urea could effectively replace some of the protein in diets for ruminants (Hart et al. 1939; Owen et al. 1943; Rupel et al. 1943).

A critical shortage of vegetable protein during World War 2, in the USA for feeding to ruminants, prompted the widespread use of urea as a feed for cattle and sheep. However, because of the toxicity problems encountered it was not until the 1950's that it became a widely accepted ingredient of cattle feed (Loosli and McDonald 1968).

The use of urea in Australia dates back to the early 1950's and is well established now as a inexpensive non-protein nitrogen supplement in cattle and sheep fed low protein diets.

6.3 Synthesis of microbial protein

The main feature of digestion in the rumen is the extensive breakdown of protein by the microorganisms and *de novo* synthesis of new microbial protein, a process that requires large amounts of energy from ATP (Czerkawski, 1986).

The microorganisms in the rumen break down dietary protein, first to amino acids, then to ammonia and various carbon compounds. The rate of breakdown of protein depends on its solubility. However, particulate protein is degraded more slowly. Although this process is apparently wasteful, some plant proteins cannot be readily digested by the ruminant and are often low in essential amino acids. The bacterial population can synthesise microbial protein, containing all the essential amino acids, forming a protein of quite high biological value. This enables the ruminant to survive on a protein source initially of lower biological value. This also has the potential of providing the animal with the majority of its amino acid requirements (Nolan, 1993). The synthesis of protein in turn also effectively means the synthesis of microbial enzymes that further are involved in the fermentation reaction.

The fermentation of the feed constituents, sugars, polysaccharides and proteins, by the enzymes contained in the bacteria, fungi and protozoa provide the organisms and the animal with essential elements for each other's survival. The microorganisms are provided with energy, in the form of ATP, needed for growth and maintenance. Endogenous use of recycled urea, excreted into the salivary secretions, is a further adaptation in the ruminant to low protein diets, 'protein conservation'. The urea, when it reaches to rumen is hydrolysed to ammonia and reconstituted into bacterial protein.

6.4 The effect of diet

It has been discussed previously in the ruminant, because of the microbial synthesis of protein, along with the recycling (conservation) of urea and the ability to use non protein nitrogen sources from either the plants, or urea, ruminants have been farmed in areas with particular pasture characteristics. The characteristic pastures in these areas are low protein levels for most if not all of the growing cycle. However, as the expectations of livestock production increases for animals to reach their genetic potential to produce protein rich products (milk, meat), the primary goal of production from these pastures is to ensure that microbial fermentation and growth in the rumen are efficient. This is in order to maximise microbial protein yield from the rumen because it is the cheapest form of protein available to the animal and also because of the effects of microbial fermentation on digestibility (Nolan

1993). The significance of the protein to energy,(P:E) ratio on microbial numbers has been discussed in Section 4.2.

By recycling purine metabolites, mucoproteins and other nitrogen compounds including ammonia in the saliva, the requirements for nitrogen can be further minimised in ruminants, allowing them to survive undernourishment for extended periods. However, the amount of 'conserved' nitrogen is not inexhaustible and a fall in the number of microorganisms in the rumen may be caused by a lack of nitrogen and also cellulose, minerals and trace elements.

Cereal diets contain amounts of crude protein of 10% or less and about 40% are totally degraded in the rumen to ammonia, amino acids and peptides to meet microbial demands.

Provided sufficient cellulose is available, ammonia production in the rumen limits microbial growth along with any mineral deficiencies occurring in the pasture. These constituents become the limiting factor for microbial growth. Supplementation therefore has to provide for protein and mineral deficiencies also in the pasture (see Section 4.2). All living cells, microbial and tissue, need the following to survive, energy, protein, minerals and vitamins. Once a deficiency in any one class is satisfied the next limiting class becomes the limit to survival or production (Virtanen,1966).

6.5 Nitrogen transactions in the rumen

The reactions that occur involving nitrogen transformation in the rumen from dry pastures are summarised below:

1. Soluble protein is degraded to peptides and then amino acids and the latter are de-aminated producing ammonia and various carbon skeleton compounds. The proteases and peptidases, usually occur on the surface of the bacteria (Preston and Leng, 1987) whereas the deaminases are intracellular. The carbon skeletons can undergo fermentation to give ATP and produce VFAs.

2. Ammonia and suitable carbon skeletons are assimilated into bacterial protein.

3. Ammonia absorption occurs across the ruminal wall into the blood stream and travels to the liver, where it is either recycled via the saliva or intestinal secretions. Excess ammonia is

changed to urea in the liver, where it is transported via the blood to all parts of the body – including to the kidney, where a variable amount is excreted in the urine.

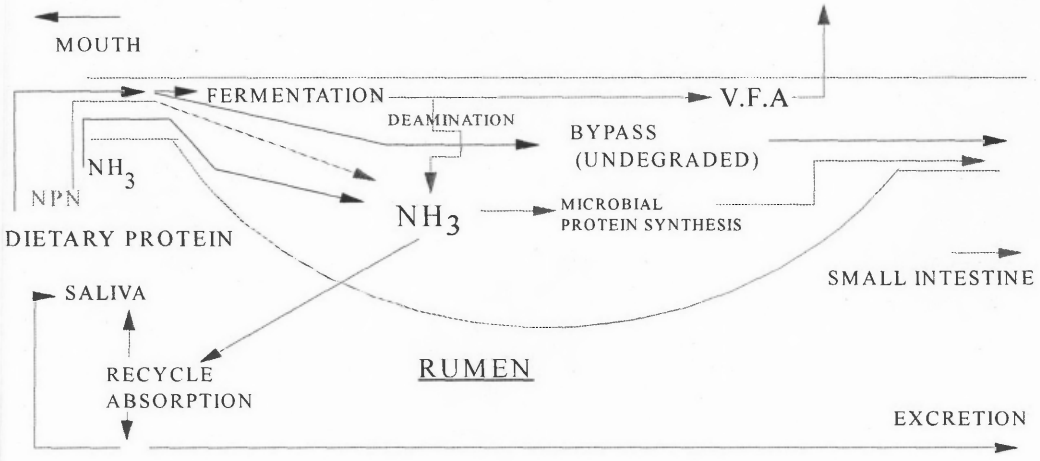
4. True protein escaping degradation in the rumen passes into the abomasum and is subjected to enzymatic digestion in the abomasum and is further digested and absorbed in the small intestine (as small peptides and amino acids).

5. Microbial protein from ruminal organisms flow from the rumen to the abomasum where they are killed by the acidity and are enzymatically degraded prior to absorption from the small intestine.

6. Non-protein nitrogen sources (such as urea and ammonium sulphate and some plant sources), also generate ammonia in the rumen for incorporation into bacterial protein, e.g.

$$N_2H_4CO + H_2O \xrightarrow{\text{Urease}} 2NH_3 + CO_2$$

Figure 18 Nitrogen reactions in the rumen



6.6 Soluble protein and undegraded (bypass or escape) protein

The majority of the soluble protein in feeds, especially in green feed, is fermented to ammonia in the rumen. Protein that is not degraded because of alterations in the structure of the protein

(partial denaturation), escapes to the small intestine where digestive enzymes dissolve it to its simpler amino acids prior to adsorption.

If after microbial synthesis is maximised, the P:E ratio is still not adequate to meet the animals' demands for production, a secondary strategy for augmenting the P : E ratio involves supplementing the diet with an undegraded (by-pass) source.

There has been little published about the factors affecting the extent of ruminal degradation of the proteins in food particles. However, time of passage through the rumen affects fermentation of the particulate proteins. In addition to chemical factors affecting cross-linking and the number of accessible sites,(to hydrolytic enzymes), in the protein molecules, physical characteristics also affect degradability in the rumen, (Preston and Leng 1987).

Using protein sources of zein and casein, McDonald and Hall (1957) showed that degradability was positively related to solubility. 'More soluble', however is not always synonymous with 'more degradable'. Soluble albumins are relatively slowly degraded, indicating that the structure, particularly disulphide bonding, as well as the solubility determines degradability (Annison, 1956).

Proteins from animal products are generally more rapidly degraded than those from plant products, however the animal proteins are seldom completely degradable. Plant proteins are more completely degradable but are lysed more slowly (Nolan, 1993). Fishmeal is a useful 'by-pass' protein supplement for ruminants as some always escapes fermentation even after a prolonged time in the rumen.

Attempts to modify proteins so that they are refractory to microbial degradation have largely been related to their physical alteration by either heat or the chemical treatment, e.g. using formaldehyde (Ferguson, 1975) prior to feeding. Beever et al. (1976), using a range of different drying techniques applied to fresh forage, observed a strong inverse relationship between the solubility of the forage protein and the flow of non–ammonium nitrogen (NAN) to the small intestine.

Faichney (1974) noted an increase in duodenal NAN flow and Ferguson (1975) increased wool growth by giving formaldehyde–treated casein to sheep.

The nutritional value of undigested protein sources such as casein reaching the small intestine and also bacterial protein depends on the amino acids they contain.

6.7 Summary of protein and the ruminant

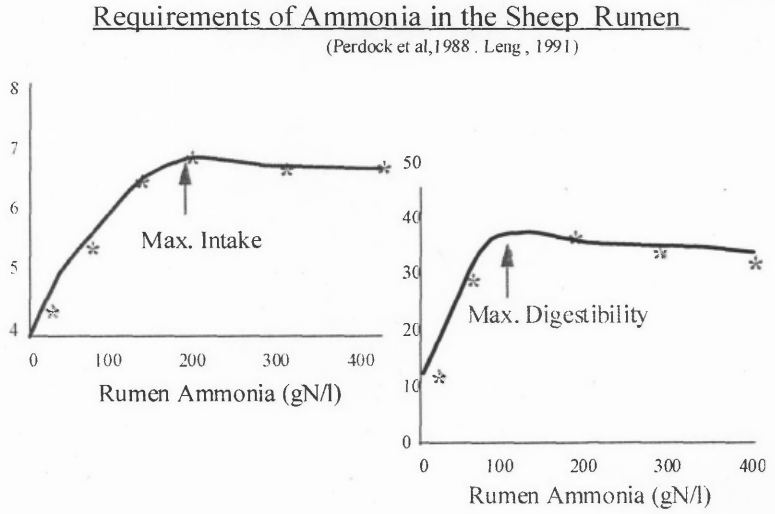
The rumen, with its microbial population has allowed the ruminant to exist and produce at moderate rates on diets lacking, or at least low, in true protein (Virtanen 1966).

Ruminants grazing the pastures on the Barkly Tableland have to exist on dry pastures that are deficient in protein for at least six months of the year (see *Introduction*).

The mechanisms available for supplementation of cattle in these areas, depend on supplying a NPN source to increase the rumen ammonia levels, when pastures are low in protein and supplying any minerals that are deficient. This strategy then supports improved digestibility of the pastures because of increased rumen bacterial numbers. When the the animal's requirements for protein (and also glucose), are highest (*viz.* last trimester of pregnancy, first 12 weeks of lactation and in growing cattle), the provision of small amounts of supplementary by-pass protein should have a positive effect on production. The improvement in milk production, growth rates in calves, fertility in males and females will consequently affect the viability of production in these areas.

The concentration of rumen ammonia levels from nitrogenous sources in the forage-based diet and their effect on the digestion of fibrous feeds levels have been examined by Perdock et al. (1988) and Leng (1991), *e.g.* Fig. 19.

Figure 19 Microbial requirements for ammonia for maximum intake (kg/d) or maximum digestibility (%)



With this experiment the maximum feed intake occured with a concentration of 200 mg ammonia-N/L of ruminal fluid; however, maximum digestibility of that feed occurred at lower concentrations (100 mg N/L). Thus the level of nitrogenous precursors for rumen ammonia does not have to be as high for maximum digestibility as for maximum intake, but maximum intake occurs at higher levels (200 mg N/L). In addition, the efficiency of microbial synthesis is not constant and will vary according to the nature and amount of diet ingested by the animal (Preston and Leng, 1987; McAllan et al. 1987).

These observations tend to confirm earlier *in vitro* studies that demonstrated that Y_{atp} (the net yield of microbial dry matter per mole of ATP produced) could vary between 10 and 20 (Harrison and McAllan 1980). Beever (1993) states that this discredits the acceptance of a constant efficiency of microbial synthesis.

It would seem reasonable to conclude that there is considerable potential to increase the ultimate supply of total protein to the animal through stimulation of microbial protein synthesis (Preston and Leng, 1987).

7 MINERALS

There are 22 mineral elements that are essential for human life (Howell, 1983). These consist of the macro-minerals, calcium, phosphorus, potassium, sodium, chlorine, magnesium and sulphur and the micro minerals iron, zinc, copper, manganese, cobalt, molybdenum, selenium, chromium, tin, vanadium, fluorine, silicon, nickel and arsenic.

The micro-minerals have to be supplied in small concentrations for the integrity of the cells and tissues to be maintained and so they were designated as 'essential'. The ruminant is more complex than the monogastric because of the microbial population in the forestomachs. This population also has needs that may be different to the host and they can supply the host with by-products *i.e.* Vitamin B12. The pastures and soils of the different regions vary all over Australia and so the requirements for minerals in supplements will differ.

Mineral supply for the micro-organism is important for their well being: bacteria need phosphorus, sodium, magnesium, copper, cobalt and zinc as well as other minerals for involvement in enzymes and other processes to participate in their intermediary metabolism (Leng 1991). While the anaerobic fungi are highly dependent on sulphur for their growth (Atkin et al. 1983), the rumen micro-organisms convert sulphate to hydrogen suphide that is used to synthesise methionine and cysteine for microbial cell growth (Preston and Leng, 1987).

Most of the minerals found in animal tissues have been ingested with and in food and there is a differing mineral composition in plants, both between species, within species and at various stages of growth. Plant trace element uptake is very dependent on soil pH, decreasing with increasing acidity (Howell, 1983). Trace elements are not uniformly distributed within plants. Copper may be held in the roots of some plants and not be transported to the shoots even under conditions of severe copper deficiency. Old green leaves may retain copper and not transport it to the young leaves and meristems (Loneragan, (1981).

Animals grazing pasture are liable to ingest soil together with the pasture plants especially in an ove-grazed area and the soil becomes a source of minerals.

The leaching with rain and moist conditions of minerals in dry standing feed contributes to the deterioration of this feed source and increases the minerals needed for supplementation of the feed.

7.1 Phosphorus

McCosker and Winks (1994) claimed that phosphorus deficiency was a major problem faced by cattle producers in the northern parts of Australia.This region is deficient in phosphorus all year round, but during the wet season this deficiency increases as the dry matter of the plant decreases. This occurs during the plants growing phase diluting the available phosphorus. The low level of phosphorus in the soils is because they have originated from parent material low in phosphorus (Skerman, 1983). Further consequences for the grazing animals are that the pasture on these deficient soils contain fewer legumes and have a low crude protein and digestibility. The animal absorbs phosphorus more efficiently from the diet if adequate protein

is also available. Thus signs of phosphorus deficiency in northern Australia are confounded with concurrent deficiencies of protein inherent with the vegetation and climate. The cattle exhibit depraved appetites, eating sticks, stones and carcasses. Bone chewing often results in the clostridial infection, 'botulism' due to unvaccinated cattle eating bones infected with the *C. Botulinum* bacterium. A significant number of animals get bone fragments caught in their mouths or in the oesophagus and develop the condition known as 'choke'. Phosphorus deficiency results in retarded growth, lowered milk yields, reduced fertility and increased bone fragility.

The clinical signs of phosphorus deficiency also are manifested in females that exhibit signs of decreased fertility and decreased cyclic oestrus activity.

Table 8 Effect of supplementation with high-phosphorus blocks* on cycling frequency in heifers and on fertility of cows near Tully, Queensland

Percentage of maiden heifers cycling (n=180)	
Before supplementation	After supplementation for14 d
0.5-1.1	10-11

Percentage of cows calving				
Unsupplemented			Supplemented	
1991	1992	1993	1994	1995
40	40	45	97	96

*MEGAPHOS molasses-based block with 10% urea (Olsson Industries)

Phosphorus and urea supplementation had a direct effect at this property. The property experienced an annual average rainfall of 2.42 m (80 inches) and the natural pasture species were typical of high rainfall areas and contained low crude protein concentrations. The effect of phosphorus deficiency on the heifers had been decreased growth rate and delayed oestrous cycles affecting the number of heifers coming into heat. The number of heifers cycling within

14 days of phosphorus supplementation increased from 1.1% to 10% per day. The effect after 1993 of phosphorus and urea supplements was an increase in fertility of 52% the following year.

Reduced growth rates resulting from phosphorus deficiency combined with a protein deficiency in both males and females, affects time to achieve mature weight and indirectly affects lifetime fertility because of the longer time it takes to achieve critical mating weight in both sexes (see Table 9).

Table 9. The effect of supplementation with high-phosphorus blocks* on growth rate and fertility of cattle

	Year	Age (months)	Live weight (kg)	Notes
Females				
	1994	24-27	280-300	Not cycling
	1996	12-15	280-300	91% (137/150) pregnant after 1st artificial insemination
Bulls				
	1994	12-15	288-312	Growth rate 600-800 g/d
	1996	12-15	480-520	Growth rate 1100-1300 g/d

* MEGAPHOS molasses-based block with 10% urea (Olsson Industries)

Source: Backhouse (1996); unpublished

7.2 Delayed growth and puberty referable to phosphorus and protein deficiency

In a survey of northern properties (O'Rourke, 1990), 69 % of properties surveyed, managers stated that heifers gave birth to their first calf at 24 months of age (15 mo old at conception) whilst 31% calved first at 36 months of age (27 mo at conception). Venereal infections, such as vibriosis, are also given as causes for infertility but until the nutritional effect is fully understood and uniform nutritional regimes are initiated, the true effect of infective causes of infertility in these herds is open to conjecture. The concurrent problems of slow maturation in

males and females and low fertility rates in the females and difficulty in removing all bulls from the herd, have led to the common practice of allowing the bulls to run with the females 12 months of the year.

The practice of non-seasonal breeding in turn has an effect on the herd structure. This is because, in practice, there is no clear or intended breeding period and although a majority of calves may be born over the wet season, some calves are born throughout the year.

7.3 Effect of year-round reproduction

This '12 month birth spread' affects the uniformity of calf size and weight in northern herds. If the dry pasture occurs during the last trimester of pregnancy, or the first 3 months post calving, there will be a low protein level of the pasture. This will affect the birth size of the calf and result in a slower calf growth because of restricted maternal milk secretion in the early life of the calf and subsequently a decreased total lactation. If the restriction of protein is also accompanied by either a cellulose and or mineral deficiency, the cow and/or calf may die. The lack of uniform age and size also adversely affects herd fertility, because of the disparity in age at which the maiden heifers begin to breed in the next year. The calves born later in the year are too young to breed next year when pasture conditions are suitable and so are delayed another 12 months before breeding. The inter-calving interval (time from calf to calf) of the cows calving in the period of feed deficiency, is longer because of the ensuing seasonal anoestrus in these cows due to the lack of nutrients from the dry pastures.

7.4 Effect of the age of the cows on reproductive rates

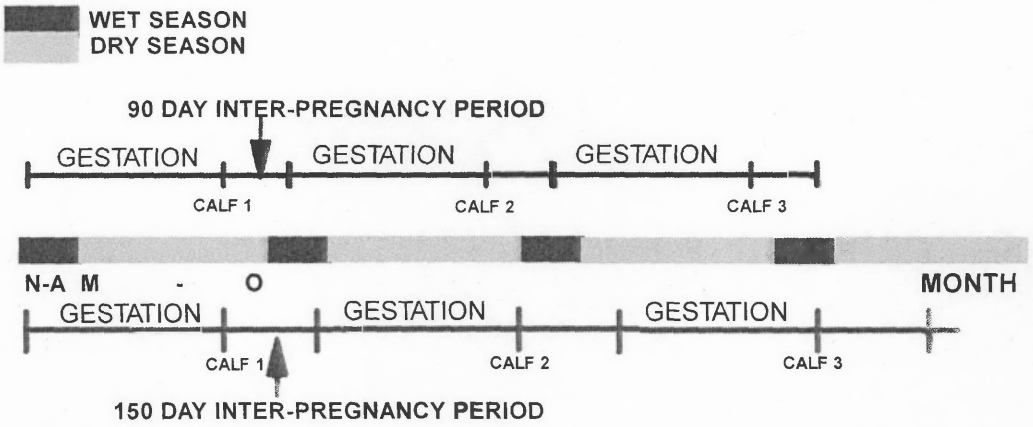
The ages of the females constituting the herd (herd structure) also has a very important effect on overall fertility of the herd. The fertility is affected in a herd consisting of older cows because a naturally occurring decreasing fertility occurs in older cows (>8 years old). Holroyd et al.(1990b) found that mean pregnancy rates of lactating cows were highest in 5-9 year olds. After 9 years, breeding cows suffered an increased mortality rate (O'Rourke et al. 1995a). It then is impossible to improve fertility to maximum levels if the herd has a majority of these cows, even with increased supplementation. In contrast, a herd of cows within the optimal age

for fertility, 3-9 years, the potential to reach its maximum physiological fertility levels is possible. Fordyce et al. (1993) found that calves of mature dams were 1.8 kg heavier at birth than those from immature dams and this resulted in calves at weaning 4-10 kg heavier if they were from mature dams. Provided they are have the nutritional capability of reaching their full breeding potential, they will have the capacity to produce more calves than a herd of older cows.

Similarly, a herd of young cows(<4 y.o.) has a lower potential reproductive capacity. Young cows grazing the northern type pastures have well recognised problems of a long intercalving after their first calf (McCLure,1994; Holroyd, 1987). O'Rourke et al. (1995c) in a review of wastage related time of calving and subsequent fertility to the age of first lactation. The latter found that Brahman cross heifers raised 4 calves in their life time up to 10 years old if they were lactating at 2–years of age, but if they were pregnant at 2-years of age, they raised 3.2 calves and if non-pregnant at this age raised only 2.6 calves. This effect can be related to the effect of birth weights and post weaning growth rates (Fordyce et al. 1993). The birth weight was affected by the nutrition of the dam in late pregnancy and, when the pasture-growing season commenced earlier, the growth rate of calves was higher (Fordyce et al. 1993). Calves from first-calf heifers are generally smaller because they have less milk available due to the immaturity of the mammary gland in the cow, but milk secretion increases in subsequent lactations. Further, because of the lack of milk production in their mothers, when weaned, they will consequently tend to be smaller (Fordyce et al. 1993). If the herd consists in any one year of a large proportion of first-calf cows, then the total number and size of saleable weaners will be less – therefore affecting profitability. If the majority of these weaners have to be retained as replacements, the physical size of the cows in the breeding herd becomes reduced. Thus small calve-size tends to be perpetuated in the herd from the smaller cows with the inter-calving interval remaining longer than the ideal.

Thus a number of the problems with herd structure can be avoided provided there are enough replacement cattle available at the optimum time each season. The number of replacement females is dependent on the inter-calving interval of the breeding females in the herd, which is in turn mainly dependent on the nutrient supply from the pasture (see Fig. 20).

Figure 20 Effect of intercalving interval



The effect of intercalving interval on fertility and subsequent first breeding age of heifers .
A. 12 month intercalving interval - cows are always breeding , lactating and ovulating at the time of protein availability and subsequent calves are always 24 months at the second following wet so will have reached critical mating weight .
B 15 month intercalving interval , calves born progressively later , by the third calf both cow and calf are at a period of protein lack at a time of greatest protein need .

In Fig. 20, it is assumed that all cows are starting their breeding life at the time of maximal nutritional value of the grass and over a breeding life of six years the cow with the inter-calving interval of 12 months provides 6 calves (top red scale). The cow that starts off as a 12-month breeder but on the second calf takes 15 months between calves (bottom blue scale) and stays on a 15-month inter-calving interval will only breed a maximum of five calves in this period. However, the situation will probably be worse than this because each year the cow is calving later in the grass cycle and by the third calf there is a lack in the quantity and nutritional quality of grass available. The result is that the inter-calving interval is more likely to become 18 months or more. Calves born at the time of nutrient deficiency will be affected by the problems associated with protein lack in the mother (small birth weight) and decreased milk availability: consequently if the calf survives, it will have a retarded growth rate and take longer to reach critical breeding weight. However, under these circumstances higher mortality in breeding cows is likely to occur due to the nutritional drain of minerals caused by reproduction (especially phosphorus). If orphaned calves are found in time, they will have to be hand-reared, but if not found in time will perish. The structure of the breeding herd is adversely affected further by this prolonged calving spread due to the rate at which

replacement female calves become available. This occurs because the cattle with inter-calving intervals of 15 months or greater provide subsequent female calves that may take 2 years to reach critical mating weight before they enter the breeding herd and the average age of the herd tends to increase. Identifying and removing the cows that have inter-calving intervals greater than 12 months and are not breeding from their daughters, if possible, will contribute to the overall improvement of herd fertility and a more stable age breeding herd. Reducing the spread of calving to only 4-5 months would produce a more uniform turnoff of calves for sale or breeding and reduce the cost of supplying supplementation. Further economy could occur because of the formulation of the supplement being used could be better suited to the cows' physiological needs.

A defined breeding period would make it easier to match the protein content of the supplement with the increasing stage of pregnancy. This would prevent the dearer protein supplements being eaten by the non-pregnant cows that do not need this level or type of protein. These issues are summarized in Fig. 20.

Niethe (1996) recognises the great loss in income in the extensive northern grazing areas due to the losses occurring from deaths in the breeder cattle. This was evident from the number of female replacements needed to keep stable breeding numbers causing a large disparity between the males and females available for sale. If cows have reached a stage in pregnancy greater than 170 days, any severe restriction in nutrients can be fatal. When studying the survivability of cattle during droughts, Tyler and Fordyce (1988) predicted that at the beginning of the drought period, cattle in poor condition, but empty, had a 50% chance of survival till the next wet. If the same cattle were at the beginning of pregnancy the survival rate was predicted to be 41%, but if they were in mid-pregnancy or late pregnancy, their survival rate was only 28% and 14%, respectively. Cow losses because of complications due to pregnancy have been historically quoted at between 5-8% (Niethe,1996). However, with the implementation of disease eradication schemes in the North the actual figure was found to be 13% in the Kimberly and Victoria River District (Schmidt and Yeates, 1982) and 11-28 % in the gulf district of Northern Territory (Brown, Schlink and Carpenter, 1994). A program of synchronisation of breeding times and strategic supplementation based around the occurrence of the maximal nutrient supply in the pasture would have a place in decreasing this very big economic loss.

Fordyce et al. (1997) estimated that post partum anoestrus extended for an average of 7 months, while Entwistle (1983) argued that high cow mortality rates, low weaning weights and post-partum anoestrus were the main contributing factors to low production in the northern herds.

7.5 Protein type and phosphorus supplementation

Supplementation with blocks in the extensive areas of cattle production in the northern territory could be a practical and feasible way of providing supplements to complement the pasture conditions, as long as sufficient animals eat enough of the supplements to improve their fertility.

The effect of strategic supplementation was demonstrated by Lindsay et al. (1982) who found that weight losses in weaner cattle grazing poor quality pastures (spear grass) could be reduced using various forms of protein supplementation. Their conclusions were that that there were more benefits to be gained from feeding a diet consisting of urea, cottonseed meal and molasses together where growth was occurring. This study provides an example of the supplement matching the physiological needs of the growing animal. The amount of phosphorus in the cottonseed meal, albeit small, might also be significant. This experiment clearly showed that animals in the growing phase, eating a low protein, fibrous hay could be supplemented with a NPN source (urea) and a by-pass protein source(cotton seed meal) containing phosphorus. These supplements, gave the best live weight gain when compared to feeding either cottonseed meal and hay, or urea and hay.

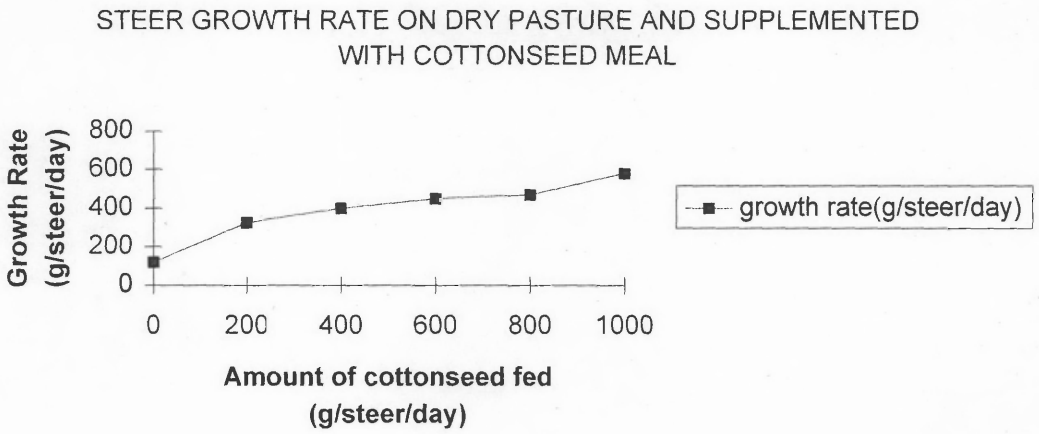
Lindsay 's group supplemented pregnant cows, fed on tropical forage diets, with protected protein. They found that pregnant cattle on spear grass hay lost 810 g/d on hay alone, 310 g/d on urea (10%)/sulphur blocks but gained 750 g/d when supplemented with 1 kg /d cottonseed meal. The cottonseed meal also contained significant levels of phosphorus.

Lindsay et al. (1982) also found that the birth weight of calves was greater (40%) from 22 kg to 31 kg) in those animals fed molasses/urea (10%) and dry spear grass pasture, in the last 3 months of pregnancy compared with those on mature dry pasture alone.

Fordyce (1995) recommends the provision of nitrogen and sulphur for most of the year to breeder in Northern Australia, but does not differentiate between different types of protein.

Smith and Warren (1986) found a linear response in growth rate in yearling steers grazing poor quality Mediterranean dry pasture when the steers were supplemented with cottonseed meal daily (see Fig. 21).

Fig. 21 Steer growth rate



Source: Smith and Warren (1986).

From the above experiments, it can be seen that supplements of urea, cottonseed and sulphur to steers grazing dry pasture resulted in increased growth rates. If both urea/molasses and by-pass protein are supplemented together when protein needs are high, then the response is even greater. This experiment, however, did not recognise the possible effect of a small amount of phosphorus from the cottonseed meal.

7.6 Cobalt

Underwood (1971) has reviewed the history of cobalt deficiency in Australia.

Cobalt deficiency is predominantly a disease of ruminants and has not been reported in monogastric species (Campbell, 1983). The inability to raise cattle in certain areas has been

associated with a chronic wasting disease and has been called 'coastal wasting disease', 'pine', 'salt sick', 'bush sickness' and 'wasting disease '. Cattle were restored to health by dosing with cobalt, top dressing the pastures with cobalt-containing fertiliser or with the insertion into the rumen of sustained release cobalt devices. Winter (1987) described cobalt deficiency in cattle in Northern Australia in cattle grazing green panic (*Panicum maximum*) and stylo (*Stylosanthes guianensis*) and showed that supplementation with intra-ruminal cobalt supplements gave increased live-weight gains.

The clinical signs of anaemia are due to a secondary lack of vitamin B12 production from the rumen microorganisms. For ruminants cobalt must be given in the feed or as a drench,because it is utilised only in the rumen by the microorganisms. The microorganisms produce vitamin B12, which contains 4% cobalt by weight. Because the manufacture of the B12 by the organisms is secondary to its role in microbial health, intra-muscular injections of B12 are ineffective in the long term and a ruminal supply of cobalt is the primary goal. The marked loss of appetite, which is the main cause of the emaciation, is related to the ruminant's dependence on acetic, propionic and butyric acids as their main energy source. The loss of micro-organisms dependent on adequate cobalt causes the digestibility of the feed to drop and, in the intermediate step in the production of VFAs the conversion of methmalonyl-CoA to succinyl-CoA is dependent on a B12 containing enzyme, methylmalonyl-coA mutase. A deficiency of cobalt and therefore B12 interferes with the production of VFAs. Vitamin B12 also is involved in the reconversion of homocysteine to methionine via the enzyme 5-methyl-tetrahydrofolate homocysteine methyltransferase. Loss of methyltransferase activity also results in a deficiency of methionine – one of the essential amino acids – in the animal. Smith et al. (1974) suggested that both folate metabolism and lipid mobilisation in the liver are mediated via methionine. Thus, a decreasing movement of lipids out of the liver in cobalt-deficient animals, helps to explain the fatty liver found on post-mortem. The ruminant has a relatively high dietary requirement of cobalt. Lee and Marston (1969) quoted values in the range 0.07–0.1 ppm/d.

The non-specific clinical condition of ill-thrift can also be caused by a number of factors other than cobalt deficiency. Malnutrition, other trace element deficiencies, parasitism and other mineral deficiencies also cause ill thrift. The post-mortem picture with cobalt deficiency is of starvation with anaemia and fatty liver. An association of cobalt status and parasitism was

found in cattle that had been receiving cobalt supplemented in an area north of Bundaberg in Queensland. *Haemonchus contortus* and *Cooperia* worm burdens increased significantly in these animals suggesting that helminths as well as microorganisms require adequate cobalt for B12 synthesis (Skerman 1983).

7.7 Copper

Although copper is universally distributed in animals and plants, extensive investigations have failed to reveal the full biological significance of the element (Campbell, 1983). Bennetts and Chapman (1937) first reported the existence of copper deficiency following work on the cause of enzootic ataxia in lambs by Bennetts (1932).

Like sheep, cattle show some degree of pigmentation loss and changes in coat fibre character. Bennetts and Hall (1939) reported 'falling disease' in cattle caused by fibrosis of the myocardium and heart failure. They found this was due to severe copper deficiency, but Wassermann (1975) concluded that the condition was due to an active vitamin D-like substance in the plants the cattle were grazing, causing calcification of the myocardial muscles resulting in failure. The role of copper is further complicated by the interaction between copper and sulphur and molybdenum. The sulphur and molybdenum interfere with copper absorption (Dick,1956). Pastures high in molybdenum, but normal in copper, caused copper deficiency in animals grazing them because of the molybdenum interference (Dick and Bull, 1945; Cunningham, 1950). Sulphur and molybdenum are cumulative in their antagonism to copper, but injected copper overcame the antagonism of the sulphur and molybdenum, showing that the antagonism of these elements to copper took place in the rumen (Suttle and Field, 1974). The feeding of other transition elements, *i.e.* zinc, iron, molybdenum, cadmium, mercury and silver affects not only absorption but also subsequent levels in the tissues.

The most obvious clinical sign of copper deficiency in cattle, as well as low weight gain, is loss of pigmentation of the hair. This is striking in red-coated animals where the coat takes on a yellowish rusty color. Around the eyes, the pigmentation change has been referred to as 'copper spectacles'. There may also be anaemia and scouring, bone fragility and a poor conception rate.

7.8 Selenium

The main site for absorption appears to be the duodenum and monogastric animals apparently absorb more of the ingested selenium than ruminants. Absorbed selenium is carried to the plasma and the selenite form of selenium undergoes a chemical transformation in the erythrocytes and under the influence of the enzyme glutathione peroxidase, is expelled from the erythrocytes and taken up by the β-lipoproteins. Selenium enters all tissues, is highly labile and can be transferred across the placenta to the foetus. It is excreted in the faeces, urine and expired air (Howell, 1983). The main seleno-protein is glutathione peroxidase that is present in a wide range of body tissues and the level in red cells is measured as an indication of selenium status.

Selenium is necessary for growth and fertility in animals and is required to prevent liver necrosis and 'mulberry heart disease' in pigs, 'exadudative' diathesis and pancreatic fibrosis in poultry and 'white muscle disease' in lambs, calves and foals. 'Ill thrift ' occurs in lambs and calves. In dairy cows retained placenta and mastitis and conception rates are selenium responsive disorders (Lean, 1991).

The problem with mild selenium deficiency in the ruminant is that the clinical signs are non-specific. Ill-thrift in young animals is not specific for selenium and can be caused by parasites, protein deficiency and cobalt deficiency and while diarrhoea may occur in calves it is not seen in lambs (Campbell, 1983).

7.9 Vitamin E and selenium

Selenium and vitamin E play different but interdependent roles in metabolising toxic byproducts caused by oxidants and free radicals, which damage unsaturated phospholipids and critical sulphydryl groups of cell membranes. Selenium is an essential component of the enzyme glutathione peroxidase and lipid peroxides in cells. Failure to remove the oxidants results in the production of harmful free radicals.

Vitamin E has a protective function on the membranes of cells and its intracellular structures by trapping the free radicals formed that have escaped the enzyme removal system.(Caple, 1990).