**Diseases associated with deficiencies of water soluble vitamins**

Water-soluble vitamins, including vitamin C and the B complex, are of minor importance in ruminants (except for vitamin B complex ,because of their synthesis in the alimentary tract of these animals.

Vitamin requirements were defined as the smallest dietary amount necessary to prevent clinical signs of deficiency.

It was demonstrated that even when fed a vitamin-free diet, the synthesis of B complex vitamins by the rumen microflora was sufficient to avoid deficiencies. Consequently, it was concluded that a dietary supply of B-complex vitamins was unnecessary in ruminants.

Niacin requirements per feed unit are higher with high-energy feeds. Niacin increases the number of ruminal protozoa and in cows with clinical or subclinical ketosis, repeated doses of niacin lead to a rapid decrease of non -esterified fatty , acids. Dietary supplementation of niacin at 6 mg/d, have improved milk production.

Biotin supplementation of 20 mg/head per day in early lactation can result in improved hoof horn health.

Folic acid is essential for cell division and growth for protein synthesis and may increase milk .

Vitamin B12 requirements are usually met by ruminal microflora synthesis if the dietary supply cobalt is adequate. High concentrate diets can modify bacterial synthesis of the vitamin and metabolic utilization of propionate increases the demand for Vitamin B12.

Thiamin, nicotinic acid, riboflavin, pantothenic acid, pyridoxine, biotin, and folic acid are all synthesized by microbial activity.

Nicotinic acid and vitamin C are synthesized by other means. The young

calf or lamb, in the period before ruminal activity begins, is likely to receive inadequate supplies of these vitamins and deficiency states can be produced experimentally.

In the pre-ruminant stage, colostrum and milk are good sources of the water-soluble vitamins, ewes' milk being much richer than cows' milk. The production of signs of deficiency of the B vitamins in horses by the feeding of deficient diets has raised some doubts as to the availability of the B vitamins synthesized in the large intestine in this species.

Vitamin C is synthesized by all species and is not an important dietary essential in any of the domestic animals. Synthesis occurs in tissues and, although blood levels fall after birth, in the newborn calf they begin to rise again at about 3 weeks of age.

A dermatosis of young calves has been associated with low levels of ascorbic acid in their plasma and responds to a single injection of 3 g of ascorbic acid. A heavy dandruff, followed by a waxy crust, alopecia and dermatitis commences on the ears and spreads over the cheeks, down the crest of the neck and over the shoulders. Some deaths have been recorded, but spontaneous recovery is more usual.

**Thiamin deficiency**

**(Hypothiminosis)**

The disease caused by deficiency of thiamin in tissues is characterized chiefly by nervous signs.

**Etiology**

Thiamin deficiency may be primary, due to deficiency of the vitamin in the diet, or secondary, because of destruction of the vitamin in the diet by thiaminase.

A primary deficiency is unlikely under natural conditions because most plants, especially , seeds, yeast, and milk contain adequate amounts.

Thiamin is normally synthesized in adequate quantities in the rumen of cattle and sheep on a well-balanced roughage diet. The degree of synthesis is governed to some extent by the composition of the ration, a sufficiency of readily fermentable carbohydrate causing an increase of synthesis of most vitamins of the B complex and a high intake in the diet reducing synthesis.

Microbial synthesis of thiamin also occurs in the alimentary tract of monogastric animals and in young calves and lambs, but not in sufficient quantities to avoid the necessity for a dietary supply, so that

deficiency states can be readily induced in these animals with experimental diets.

**Epidemiology**

One of the best examples of secondary thiamin deficiency is inclusion of excess raw fish in the diet of carnivores, resulting in destruction of thiamin because of the high content of thiaminase in the fish.

A thiaminase-induced subclinical thiamin deficiency causing sub optimal growth rate of weaner lambs has been described.

Higher levels of thiaminase activity were present in the feces and rumen contents of lambs with poor growth rate compared with normal lambs .

The evidence indicates that the thiamin deficiency is a primalY one associated with deprivation of feed during transportation to the preembarkation feedlots. The low feed intake and failure of the ruminal microbes to adapt, thrive and synthesize a net surplus of thiamin during

**Pathogenesis**

The only known function of thiamin is its activity as a cocarboxylase in the metabolism of fats, carbohydrates and proteins and a deficiency of the vitamin leads to the accumulation of endogenous pyruvates.

Although the brain is known to depend largely on carbohydrate as a source of energy, there is no obvious relationship between a deficiency of thiamin and the development of the nervous signs which characterize it.

There are other prodromal indications of deficiency disease. For example, there is a decrease in erythrocyte precursors and in erythrocyte transketolase.

Additional clinical signs also in the circulatory and alimentary systems, but their pathogenesis cannot be clearly related to the known functions of thiamin. Subclinical thiamin deficiency due to thiaminases in the alimentary tract is associated with low erythrocyte transketolase activities and elevated thiamin pyrophosphate effects, which may explain the poor growth rate .

**Clinical findings**

Bracken fern (Pteridium aquilinum) and horsetail (Equisetum arvense) poisoning in the horse Incoordination and falling and bradycardia due to cardiac irregularity, are the cardinal clinical signs of bracken fern poisoning in the horse.

These signs disappear after the parenteral administration of thiamin. Similar clinical effects occur with horse, tail swaying from side to side occurs first, followed by pronounced incoordination, including crossing of the forelegs and Wide action in the hind legs.

When standing, the legs are placed well apart and crouching and arching of the back are evident. Muscle tremor develops and eventually the horse is unable to rise.

Clonic convulsions and opisthotonos are the terminal stage.

Appetite is good until late in the disease when somnolence prevents eating.

Temperatures are normal and the heart rate slow until the terminal

period when both rise to above normal levels.

The greatest danger is when the immature plants are cut and preserved in meadow hay.

Lambs 1-3 days old placed on a thiamin -deficient diet show signs after 3 weeks.

Somnolence, anorexia, and loss of condition occur first, followed by tetanic convulsions.

**Treatment**

In clinical cases the injection of a solution of the vitamin produces dramatic results (5 mg/kg BW given every 3 h) . The initial dose is usually given IV followed by 1M injections for 2-4 days. An oral source of thiamin should be given daily for 10 days and any dietary abnormalities corrected.

CONTROL

The daily requirement of thiamin for mono gastric animals is, in general,

30-60 µg/kg BW. The addition of yeast, cereals, grains, liver, and meat meal to the ration usually provides adequate thiamin.

**Biotin (Vitamin H) Deficiency**

**(Hypobiotinosis)**

Biotin or vitamin H, has several important biochemical functions. It is a cofactor in several enzyme systems involved in carboxylation and trans carboxylation reactions and consequently has a significant effect on carbohydrate metabolism, fatty acid synthesis, amino acid deamination,

purine synthesis, and nucleic acid metabolism. Biotin is found in almost all plant and animal materials and, being required in very small quantities, is unlikely to be deficient in diets under natural conditions,

especially as microbial synthesis occurs in the alimentary tract.

**Cattle**

Biotin is now considered a significant factor in lameness of cattle. Biotin is important for the differentiation of epidermal cells which are required for normal production of keratin and hoof horn tissue. Biotin also acts as a co-factor in carboxylase enzymes and is an important factor in both gluconeogenesis and fatty acid synthesis. Significant differences in the

fatty acid profile of horn tissue of cattle with claw lesions have been observed.

Biotin supplementation reduces clinical white line disease, reduces horn lesions, and improves horn quality by strengthening the intercellular cementing material between keratinocytes. Improved hoof integrity in intensively managed dairy cows has occurred following biotin supplementation.

A long period of supplementation is required before the

effect of the vitamin on hoof health care is expressed. In addition, there may be improved milk production, milk composition, and cow fertility with biotin Supplementation .

Skin lesions also develop in affected gilts and sows. There is gradual alopecia, particularly over the back, the base of the tail, and the hindquarters. The hairs are more bristly than normal and break easily.

The alopecia is accompanied by a dryness of the skin.

**Folic acid deficiency**

**(Hypofolicosis)**

Folic acid (pteroylglutamic acid) is necessary for nucleic acid metabolism and its deficiency in humans leads to the development of pernicious anemia. A dietary source is necessary to all species and an adequate intake is provided by pasture.

Although naturally occurring deficiencies have not been diagnosed positively in domestic animals, folic acid has numerous and complex interrelationships with other nutrients and the possibility of a

deficiency playing a part in inferior animal performance should not be

overlooked. The vitamin has a particular interest for equine nutritionists.

**Vitamin B12 deficiency**

**(Hypocyanocobal).**

Vitamin B12 deficiency is unlikely to occur under natural conditions other than because of a primary dietary deficiency of cobalt, which is an important disease in many countries of the world.

Although microbial synthesis of the vitamin occurs in the rumen in the presence of adequate cobalt and in the intestines of other herbivores such as the horse, it is probably a dietary essential in the pig and young calf. Animal protein is a good source. A deficiency syndrome has been produced in young calves on a synthetic ration. Signs include anorexia,

cessation of growth, loss of condition, and muscular weakness.

The daily requirement under these conditions is 20-40 flg of vitamin B12.