RUMINANT THIAMINE REQUIREMENT IN PERSPECTIVE

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ABSTRACT


Thiaminases play an important role in the aetiology of CCN being responsible for the state of thiamine-deficiency which is an essential feature of the disease, evidence for which is presented here. These studies have led to a greater appreciation of the role of thiamine and thiaminases in ruminant nutrition especially as ruminal thiaminase activity is not confined to clinically affected animals but is of wider distribution. The importance of thiaminases in intensive beef production and the possibility of the need for thiamine supplementation in the form of a thiaminase resistant derivative is discussed.

Thiamine Deficiency and Cerebrocortical Necrosis

Thiamine is an obligatory nutritional requirement for many forms of animal and plant life. In tissues it occurs mainly as its diphosphate (TPP), and is a necessary coenzyme for several reactions in carbohydrate metabolism. In nervous tissue about 80% of thiamine is in this form, 5-15% as the triphosphate and the rest as the monophosphate and free thiamine. Deficiency of thiamine will lead to an impairment in function of the TPP-dependent enzyme systems and in nerve conduction.

It has always been thought that the ruminant is independent of an extraneous supply of thiamine as adequate amounts would be synthesised in the rumen. However during the last two decades a ruminant disease called cerebrocortical necrosis (CCN) or polioencephalomalacia (PEM) in which thiamine-deficiency plays an essential role, has been studied in several laboratories. The aetiology of CCN and its possible implications in intensive production are discussed here.

Cerebrocortical necrosis has been described in many parts of the world. Besides cattle and sheep it has been reported in other ruminant species such as the goat, deer and antelope. In cattle and sheep there is no breed or sex preference; the only uniformity being the age of incidence. Young animals between the ages of 2 to 7 months appear to be most susceptible, although cases outside this range occur.
The clinical signs of the disease involve impairment of the central nervous system and may include some of the following: aimless wandering, disorientation, blindness, recumbency, hyperaesthesia with clonic extensor spasms and opisthotonus. A detailed description of the disease is given by Terlecki and Markson (1961). The clinical signs last from 2-6 days and if not treated will usually end in death. Few spontaneous recoveries have been reported. At necropsy the only visible abnormality appears in the brain where disseminated areas of yellowish discoloration can be seen throughout the cerebral cortex. The brain may be oedematous with many swollen gyri. In some cases the intracranial pressure is so great that the cerebellum is herniated into the foramen magnum. The yellow areas appear fluorescent under ultra-violet light (Ca 365 nm) and on sectioning, clearly marked greenish yellow fluorescence may be seen confined to the outer layers of the cortical grey matter. Histopathology shows the predominant abnormality to be a focal or laminar necrosis of the cerebral cortex and its neurons suggesting localised anoxia (Pill et al, 1966; Jubb and Kennedy, 1970.)

Affected animals respond, often dramatically, to treatment with thiamine. Biochemical studies of the disease show the affected animal to be very deficient in thiamine.

The evidence for thiamine-deficiency is based on four practical considerations:

1. Response to thiamine treatment. Whilst this is not specific it is a valuable piece of evidence when considered in conjunction with the other criteria.

2. Tissue levels of thiamine are significantly lower than in healthy animals. When thiamine-deficiency is induced in monogastric animals by dietary means, the vitamin becomes depleted at different rates in different tissues (Lowry, 1952). Levels in liver fall rapidly whereas in brain the fall is much slower. McCandless et al (1968) found that in rats thiamine levels had to fall below 20% of normal values before onset of nervous symptoms. The fall in tissue and brain thiamine in CCN is of this order (Pill, 1967; Behrens and Holler, 1977; Edwin et al, 1979).

3. In thiamine deficiency intermediates of the tricarboxylic acid cycle such as pyruvate and lactate tend to accumulate in tissues and blood. During the early work on thiamine metabolism (Peters, 1963), the rise of pyruvate in blood and tissue in pigeons and rats was taken to indicate thiamine-deficiency. Besides pyruvate, accumulation of other ketoacids such as α-ketoglutarate, glyoxylate, α-ketoisocaproate, hydroxyphenylpyruvate and phenylpyruvate has been recorded in our laboratory (details to be published elsewhere). Here also the relevant dehydrogenases need TPP as a