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A new approach to Polioencephalomalacia (PEM)

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A New Approach to Polioencephalomalacia (PEM)

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Description of the Problem

Polioencephalomalacia (PEM) is a disorder of the ruminant central nervous system characterized by sudden onset and rapid death. At autopsy, the brain may be swollen and cerebral cortex degenerated. Early symptoms may include disorientation and muscular incoordination. Affected animals may push against fences or other objects with their heads. More commonly, they are found dead or in a coma. If central nervous system damage is not excessive, animals with early symptoms respond to massive injections of thiamine, but may not recover coordination.

Its rapid and generally fatal course likely explains why little research has been done on the disease. The response to thiamine has raised many questions. Rumen nutritionists have long assumed that rumen microorganisms synthesized enough thiamine to meet an animal's needs. Even if none were produced in the rumen, normal diets should furnish enough thiamine.

Discovery of an Experimental Model

The Department of Animal Science and Industry established a project in 1969 to study control feed intake of ruminants--to find out how much feed an animal can metabolize, and the metabolic and biochemical limits on feed consumption. Sheep were fitted with rumen fistulas, and liquid diets were continuously pumped into their rumens. The diets were a suspension of starch, sugar, casein, vitamins A, D, E, and K, and all minerals known to be essential. Animals were on the diets for up to 21 days, and then died suddenly after body temperature and heart rate increased.

Autopsy showed no cause of death. However, young animals and those on high energy intakes died sooner than their counterparts. Serum minerals (sodium, potassium, calcium, phosphorus, magnesium, zinc, and copper), hemoglobin, hematocrit, blood carbon dioxide, oxygen, and pH, and serum protein and urea all were normal.

Two 79-pound lambs were started on infusion at a level designed to supply 100% of the maintenance requirement. To attempt to re-create the symptoms of previous animals, energy intake was increased to 125% of maintenance the second day. On day four, one was in a coma, with a rectal temperature of 106°F and a heart rate of 188. Thiamine (200 mg) was given in the jugular vein and 200 mg intramuscularly. In two hours, rectal temperature was 104°F and the heart rate, 165. In three hours, the animal could stand. However, it relapsed and died about 10 hours after thiamine injection. At autopsy, the brain was removed for detailed microscopic study. The cerebral cortex was degenerated. Apparently, brain degeneration had proceeded too far for recovery.

The second animal of the pair showed symptoms on day six of infusion, and was immediately treated with thiamine. Recovery was dramatic. Twenty-eight hours later, symptoms recurred, but again the animal responded to thiamine injection. It entered and recovered from thiamine deficiency a third time, and was removed from the experiment. Five months later, it developed urinary calculi and was autopsied. Classical brain changes of PEM were seen.

Another lamb was started on infusion, with 150 mg thiamine per day added to the liquid diet. After four weeks of infusion, thiamine was removed from the diet; 48 hours later, heart rate increased from 100 to 180 beats per minute, and three days later, to 220. The animal died in thiamine deficiency nine days after thiamine was removed.

To facilitate pumping, the liquid diet was modified using corn sugar instead of starch (table 42). It produced the same deficiency symptoms observed earlier.

Apparently, infusion of the semi-purified diet (table 42) into fistulated lambs serves as an animal model to study PEM. Since the syndrome can be re-created, research on it should continue.

Our results suggest that thiamine nutrition of ruminants should be reexamined. Nutritionists have long assumed that ruminants synthesize enough B vitamins to meet their needs.

Simple thiamine deficiency is unlikely for two reasons: 1. Most diets, particularly those high in concentrates, should contain enough thiamine; 2. the PEM syndrome develops much more rapidly than does thiamine deficiency in monogastric animals

The Thiaminase Hypothesis

We hypothesize that PEM is caused by the production of an enzyme, thiaminase, probably in the rumen. At least two types of thiaminase are known. One breaks the thiamine molecule to two parts. The other creates a molecule with a shape and formula similar (but not identical) to thiamine that acts as a thiamine antagonist. Ampolium, an effective coccidiostat, functions as a thiamine antagonist, and large doses have caused PEM in cattle.

The brain, in contrast to other body tissues, can obtain energy only from metabolized glucose. As glucose metabolism requires thiamine, production of thiaminase and a subsequent thiamine antagonist could explain the severe symptoms involving the central nervous system.

Summary

An experimental model for production of polioencephalomalacia (PEM) has been developed. We postulate that in PEM, the rumen microorganisms produce an enzyme, thiaminase, that either destroys thiamine or produces a thiamine antagonist. The antagonist could explain the lesions and symptoms involving the central nervous system. Further research is underway to examine: (1) the thiaminase and thiamine antagonist hypothesis, and (2) possible remedies.

Table 42. Liquid ration composition, maintenance, 100-lb. lamb.

Source	Amounts ¹	Source	Amounts ¹
Corn Sugar	735.8	NaCl	10.000
Casein	64.1	CoCl ₂ ·6H ₂ O	0.806 ²
MnSO ₄ ·H ₂ O	137.070 ²	CuCl ₂	33.884 ²
K ₂ CO ₃ ·1 1/2 H ₂ O	37.396 ²	KI	0.980 ²
ZnSO ₄ ·7H ₂ O	391.304 ²	CrK(SO ₄) ₂ ·12H ₂ O	1.440 ²
Na ₂ MoO ₄ ·2H ₂ O	6.944 ²	FeCl ₂ ·4H ₂ O	1.100
CaCl ₂ ·2H ₂ O	11.715	Vitamin A	2200.000 ³
NaH PO ₄ ·H ₂ O	11.586	Vitamin D	500.000 ³
MgSO ₄ ·7H ₂ O	10.761	Vitamin E	11.000 ³
MgCl ₂ ·6H ₂ O	4.068		

¹ Expressed in grams per day unless indicated.

² Milligrams per day.

³ Expressed as international units per day.