FECAL THIAMINASE IN FEEDLOT CATTLE

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Summary

Fecal thiaminase was measured on 152 feedlot cattle at three locations and on a variety of rations. No animals showed signs of polioencephalomalacia. Thiaminase activities ranged from 0.6 to 430 μmol thiamin destroyed per minute per liter of feces (μmol/min/l). Eighty-two percent of the thiaminase activities were below 20 μmol/min/l, and only 3 percent were less than 2 μmol/min/l. High levels of fecal thiaminase were apparently not related to ration. Thiaminase was detected in all animals studied, but one location had only minimal levels. When high levels of thiaminase were found, the samples were re-assayed, and the enzyme was confirmed to be thiaminase type I. Polioencephalomalacia, a central nervous system disease in ruminants, involves gastrointestinal destruction of thiamin, and the creation, through the action of thiaminase I and a cosubstrate, of a thiamin analog that inhibits thiamin-requiring metabolic reactions. Our data suggest that substantial numbers of feedlot cattle have the enzyme in their gastrointestinal tracts, but do not develop polioencephalomalacia because the appropriate cosubstrate is absent.

Introduction

Polioencephalomalacia (PEM) is a central nervous system disease of feedlot cattle that responds dramatically to large doses of injected thiamin, provided the treatment is given early. This response to thiamin seems peculiar, since the ruminant animal receives thiamin as a natural component of feed, and it is synthesized by rumen bacteria. Research in the United States, Great Britain, and Australia has shown that one factor in the etiology of PEM is an enzyme, thiaminase I, that breaks the bond between thiamin's two organic rings and replaces the thiazole ring with a nitrogen-containing "cosubstrate." A number of compounds will fill the cosubstrate role, but the specific cosubstrate involved with PEM has not been identified. Research at Kansas State has shown that the rate at which thiaminase operates is controlled largely by the amount of cosubstrate.

British researchers have shown that when thiaminase I is present in the rumen, it is also present in the feces, even from "normal" animals. The data reported here were collected in an attempt to find out how prevalent thiaminase I is in the gastrointestinal tracts of feedlot cattle.

Experimental Procedures

Our fecal thiaminase I assay utilized small amounts of radioactive thiamin and high levels of both thiamin (the primary substrate) and aniline, a cosubstrate. *Clostridium sporogenes*, a

bacterium known to produce large amounts of thiaminase I, was included in each sample set to verify that the assay was working properly.

Fecal grab samples were collected at random, diluted 1:1 with distilled water, squeezed through cheesecloth, and assayed. Samples were collected at the KSU Beef Research Center, the Fort Hays Branch Experiment Station, and at Ellis County Feeders, Hays, KS. All cattle appeared normal.

No information is available as to what constitutes "normal" versus "abnormal" fecal thiaminase activities. Based on examination of our data, a level that destroyed 20 μmol thiamin per minute per liter fresh feces (μmol/min/l) was arbitrarily chosen as the breaking point. Thiaminase activities over 100 μmol/min/l were designated as "high".

Animal and ration details are shown in Table 31.1.

Results and Discussion

Examination of Table 31.1 shows that there is little, if any pattern to the occurrence of "abnormal" or "high" ruminal thiaminase. Although at the KSU Beef Research Unit, all pens fed high levels of milo had animals with high thiaminase levels, a similar diet yielded the lowest thiaminase levels at Fort Hays. High levels of thiaminase occurred frequently in steers fed steam-flaked corn at the Fort Hays Branch Station; however, just a few miles away at Ellis County Feeders, animals fed cracked corn showed only low thiaminase levels.

Thiaminase I activities varied, from 0.6 to 430 μmol thiamin/min/l. All fecal samples showed at least some thiaminase I activity. If one sorts the thiaminase I levels from the entire experiment from low to high activity, then plots the ranking of each sample versus its thiaminase I activities (Figure 31.1), a fairly logical break is found at an activity of about 20 μmol/min/l; 82 percent of the samples had lower and 18 percent had greater concentrations.

Whenever an abnormal level of thiaminase activity was found, the sample was reassayed without added cosubstrate. In nearly all cases, little activity was found, indicating that no native cosubstrate was present in the sample. However, absence of cosubstrate in the feces may not indicate absence in other regions of the gastrointestinal tract. The known cosubstrates of thiaminase I are relatively small molecules and should be absorbed as they pass down the tract.

The assay we used is unable to discriminate directly between enzymatic and chemical thiamin destruction. In the rumen, the sulfate ion is converted to sulfide. However, sulfite is an intermediate in the process, and the sulfite ion is a potent destroyer of thiamin. PEM-like signs have been caused by high levels of sulfate in water or feeding gypsum (calcium sulfate) as a feed intake regulator. Chemical breakdown of thiamin by the sulfite ion was not a factor in our data because reassay of our high-activity samples in the absence of cosubstrate showed little, if any, activity. If sulfite had been present, thiamin destruction would have taken place, even in the absence of thiaminase I.

Although several nitrogen-containing organic bases can serve as thiaminase I cosubstrates, no one has yet established the specific cosubstrate responsible for PEM. For PEM to develop, thiaminase I must act on both thiamin and the specific "PEM cosubstrate" to create a specific

thiamin analog. Earlier studies at KSU have shown that there is probably always enough thiamin available to let the reaction operate at maximum speed. According to the results of the present experiment, a substantial number of feedlot cattle contain thiaminase I in their gastrointestinal tracts, some at high levels. The fact that so few animals develop PEM indicates that the limiting factor in the reaction is specific cosubstrate availability.

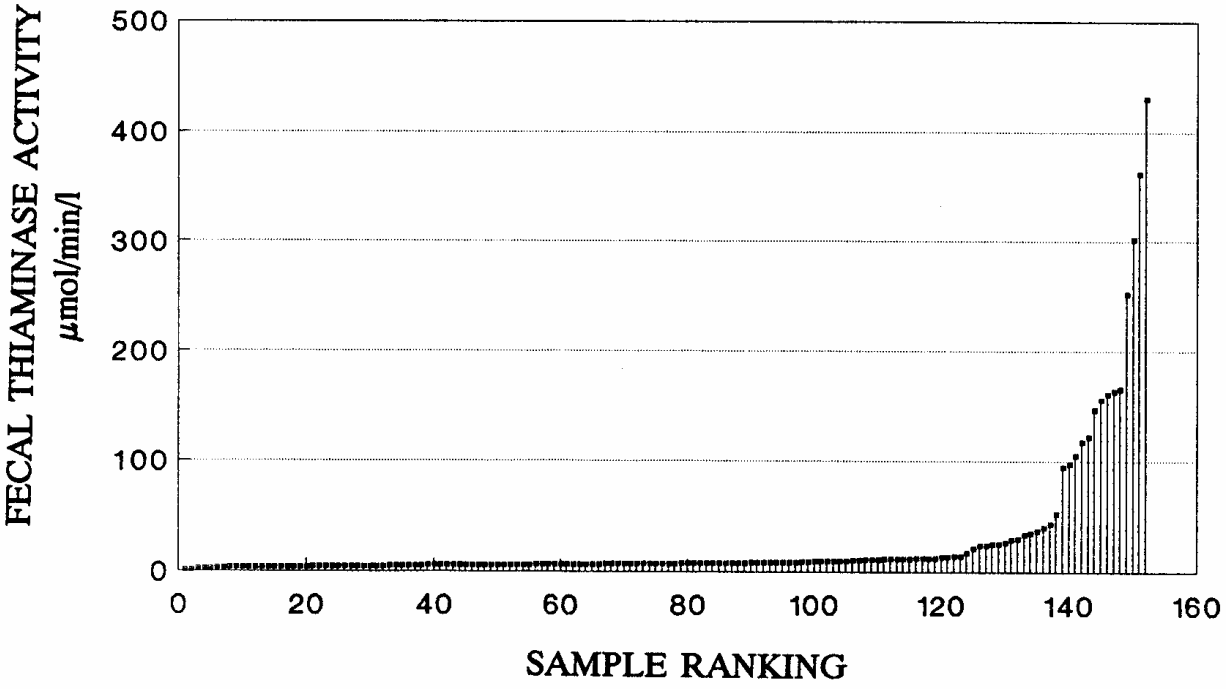

Figure 31.1. Plot of sorted fecal thiaminase activities.

Table 31.1. Numbers and Percentages of Cattle with Abnormal (over 20 μmol/min/l) Thiaminase I Activities and Maximum Activity

Description	No. Sampled	μmol Thiamin Destroyed/min/l 20-100	100 +	Max. Act.
KSU Beef Research Center				
780 lb steers fed an 84% milo diet with corn silage as the roughage. On the diet 17 days.	11	3[a]	2	161
545 lb heifers fed a 35% milo diet with equal parts of milo and corn silage. On the diet 11 days.	11	0	0	6
625 lb heifers fed an 84% milo diet with corn silage as the roughage. On the diet 14 days.	9	3	1	121
285 lb mixed calves fed a 75% milo diet with whole plant milo silage as the roughage. On the diet 7 days.	4	0	0	4
750 lb Holstein steers fed an 84% milo diet with corn silage as the roughage. On the diet 21 days.	2	1	0	52
Fort Hays Branch Experiment Station				
1200 lb steers finished on a high-concentrate diet based on steam-flaked corn.	22	3	7	361
1200 lb steers finished on a high-concentrate diet. Concentrate mix was 50% steam-flaked corn, 50% ground milo.	20	1	1	163
1200 lb steers finished on a high-concentrate diet based on ground milo.	24	2	0	24
650 to 725 lb heifers fed a high-concentrate diet based on ground milo.	9	1	0	28
575 lb heifers fed a high-concentrate diet based on ground milo.	7	1	1	430
Ellis County Feeders				
670 lb steers on a starting diet containing 10% cracked corn. Steers had arrived in the yard 4 days earlier.	18	1	0	23
800 to 850 lb steers on a finishing diet of 90% cracked corn. They had been on the diet 21 days.	15	0	0	9

[a]Number of cattle in each category.