

GASTROINTESTINAL THIAMINASE VS. RATION CHANGES

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Summary

High levels of the thiamin-destroying enzyme, thiaminase I, were found in the feces of 3 of 50 apparently healthy dairy cows. All high fecal thiaminase I levels returned to normal within 3 weeks, indicating that thiaminase I occurs in "spikes" rather than continuing at elevated levels. All cows sampled had some thiaminase I, but the upper end of the "normal" range in feces was about 3.5 $\mu\text{mol}/\text{min}/\text{l}$. Thiaminase I levels were higher in the first than in subsequent lactations. When spikes in thiaminase I activity occurred, they were concentrated within about 20 days of calving and of the associated change to a high concentrate diet. Lactating cows fed a high concentrate post-calving diet had more thiaminase I than parturient cows fed a lower energy diet.

(Key Words: Polioencephalomalacia, Thiamin, Thiaminase.)

Introduction

Polioencephalomalacia is a central nervous disorder in ruminants. Affected animals respond rapidly to large thiamin injections even though thiamin intake and ruminal thiamin synthesis should be more than adequate. Although other factors are involved, the main cause appears to be microbial synthesis in the gastrointestinal tract of an enzyme that destroys thiamin. That enzyme, designated thiaminase I, splits the thiamin molecule between its two rings and substitutes a nitrogen-containing base (cosubstrate) for thiazole. Thus, for thiamin to be destroyed, three factors must be present; thiaminase I, thiamin (the primary substrate), and the cosubstrate. British researchers have shown that fecal thiaminase can serve as a

marker for the presence of thiaminase in the gastrointestinal tract and also that high levels of fecal thiaminase may be present in apparently healthy animals. Little is known as to whether or not those high levels persist. Hayes and Brent (KSU Cattlemen's Day, 1989) found a high level of fecal thiaminase I in a substantial number of feedlot cattle and suggested that polioencephalomalacia failed to develop because the appropriate co-enzyme was not present.

Several researchers have suggested that high levels of thiaminase I develop in response to ration changes. Dairy cattle offer a unique opportunity to study thiaminase because they are switched from a high roughage diet to a high energy diet at each calving.

Experimental Procedures

In our first experiment, fecal thiaminase was measured in 50 dairy cows that were from 8 to 139 days in milk. Three cows were found with fecal thiaminase levels above 3.5 $\mu\text{mol}/\text{min}/\text{l}$. Those cows were resampled weekly for 4 weeks. At the end of 4 weeks, all cows were re-sampled.

In a second experiment, five dairy cows were selected at random and sampled while on their precalving diet (roughage plus 14 lb/hd/day of concentrate) for 3 weeks prior to calving. At calving, the cows were switched to roughage plus 32 lb/hd/day of concentrate and were sampled weekly for an additional 3 weeks.

Fecal samples were diluted 1:1 with distilled water, and strained through cheesecloth,

and the resulting solution was assayed for thiaminase I. Each assay included a small amount of radioactive thiamin (labeled in carbon 1 of the thiazole ring), excess nonradioactive thiamin, and aniline as a cosubstrate. As radioactive thiamin was decomposed, its radioactivity was extracted into ethyl acetate. Activity was expressed as micromols of thiaminase destroyed per minute per liter of fresh feces ($\mu\text{mol}/\text{min}/\text{l}$). All samples that showed high levels of thiaminase were reassayed without aniline to confirm that the thiamin was being decomposed by thiaminase I and not by simple chemical activity.

Results and Discussion

Thiaminase I has been present in the feces of all cattle feces we have assayed. However, the levels are normally low. We defined the upper limit of normal thiaminase I activity as the point at which samples exceeded the cumulative average plus two standard deviations. That technique estimated $3.5 \mu\text{mol}/\text{min}/\text{l}$ as the upper normal thiaminase level.

In the first experiment, three animals were found with thiaminase I levels greater than $3.5 \mu\text{mol}/\text{min}/\text{l}$. When reassayed at weekly intervals, all had returned to normal levels before the third weekly sample (Figure 1). That strongly suggests that when an animal has a high level of gastrointestinal thiaminase, the problem persists for a relatively short time. This may help explain why very few animals develop polioencephalomalacia, even though thiaminase I is high. When the entire group of 50 cows was reassayed 4 weeks after the initial assay, no abnormally high thiaminase I levels were found.

When the dairy cows calved, their concentrate intake was increased from 14 to 32 lb/hd/day. Figure 2 shows the relationship between thiaminase levels and days after the concentrate increase. The three highest thiaminase values were within 20 days after calving. We have previously observed that polioencephalomalacia is most likely about 3 weeks after a ration change. Our data suggest that thiaminase production in the gastrointestinal tract may coincide with adaptation to a new diet.

Figure 3 shows the relationship between thiaminase I activity and lactation number. Thiaminase levels were higher ($P < .05$) during the first lactation than subsequent lactations. Cows in the first lactation are experiencing their first substantial ration change. Behavioral and/or physiological differences between the first and subsequent calvings may account for differences in thiaminase levels.

Results of the second experiment are shown in Figure 4. Mean thiaminase I activity was lower ($P < .05$) on the precalving diet (14 lb/hd/day concentrate) than on the postcalving diet (32 lb/hd/day concentrate). The highest thiaminase I level in this experiment was $4.1 \mu\text{mol}/\text{min}/\text{l}$. The absence of thiaminase I spikes like those seen in the first experiment is not surprising because we were sampling only 5 cows, and high thiaminase levels were observed in only 3 cows out of 50 in the first experiment.

The effect of thiaminase spikes on animal performance is unknown and will be difficult to measure. Few animals are affected, and the performance depression, if it occurs, is probably transitory. Thiaminase I in feces may not mean that the thiamin status of the animal is severely compromised, because a cosubstrate must be present for thiamin to be destroyed. However, the presence of fecal thiaminase I casts doubt on the presumption that thiamin is always adequate in ruminants.

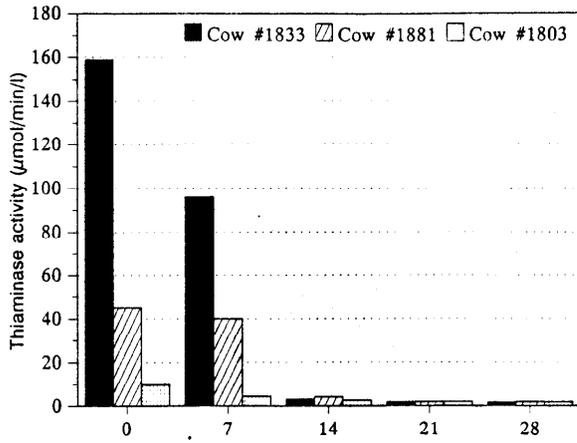


Figure 1. Decline of Fecal Thiaminase with Time in Three Dairy Cows Initially Found to Have Abnormally High Fecal Thiaminase

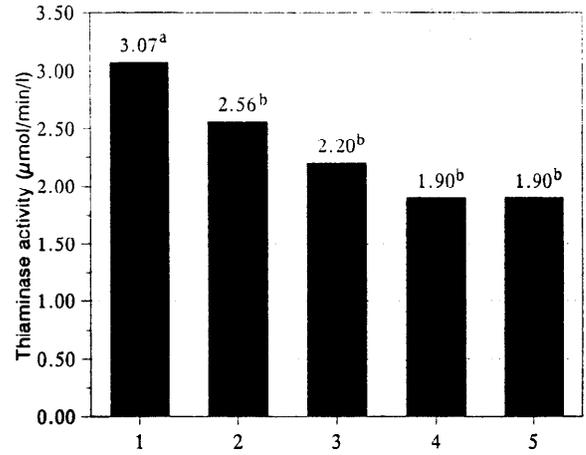


Figure 3. Effect of Lactation Number on Thiaminase Activity

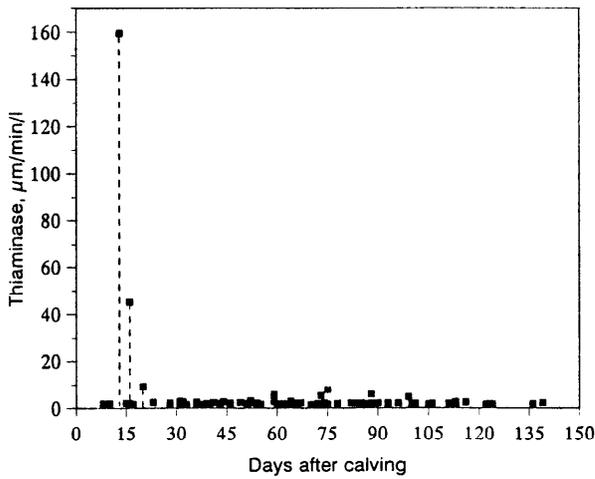


Figure 2. Fecal Thiaminase Levels vs. Time after Calving

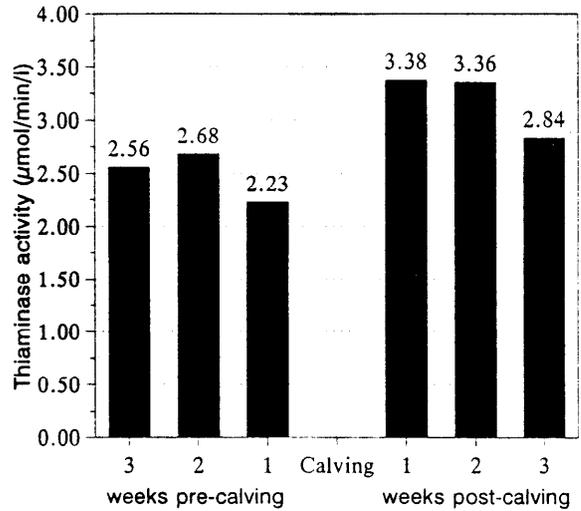


Figure 4. Effect of Increasing Concentrate at Calving on Fecal Thiaminase