Article

Thiamine status of feedlot cattle fed a high-concentrate diet

Tolga Karapinar, Murat Dabak, Omer Kizil

Abstract – As thiamine status of ruminants is adversely affected by rumen acidity, this study investigated whether or not thiamine deficiency occurs in feedlot cattle fed a high concentrate diet. Fifty 1- to 2-year-old feedlot cattle fed a high concentrate diet (75% barley) for at least 3 mo (high concentrate diet group) and 15 healthy feedlot cattle of similar ages (control group) that were fed a low concentrate diet (30% barley) were used. Rumen fluid samples were obtained by rumenocentesis and their pH was determined with a portable pH meter. Blood samples taken from all animals from a jugular vein were used to determine erythrocyte transketolase enzyme activity, and hence thiamine pyrophosphate (TPP) effect. Odor and mean pH values of ruminal fluid samples from the high concentrate diet group (47.2 ± 3.2) was significantly higher than in the control group (19.53 ± 2.5) (P < 0.001). The study provides evidence of a TPP effect in feedlot cattle fed a high concentrate diet.

Résumé – Statut de la thiamine de bovins d'engraissement consommant une diète à concentration élevée. Vu que le statut de la thiamine des ruminants est affecté négativement par l'acidité du rumen, une enquête a été réalisée pour déterminer si une carence en thiamine se produit ou non chez le bétail nourri à l'aide d'une diète à concentration élevée. Cinquante bovins âgés de 1 ou de 2 ans nourris d'une diète à concentration élevée (75 % d'orge) pendant au moins 3 mois (groupe de la diète à concentration élevée) et 15 bovins d'engraissement en santé d'âge semblable (groupe témoin) nourris à l'aide d'une diète à faible concentration (30 % d'orge) ont été utilisés. Des échantillons du liquide du rumen ont été obtenus par ruminocentèse et leur pH a été déterminé avec un compteur de pH portable. Des échantillons sanguins prélevés de tous les animaux à partir d'une veine jugulaire ont été utilisés pour déterminer l'activité des enzymes transcétolases érythrocytaires et donc l'effet du pyrophosphate de thiamine. L'odeur et les valeurs moyennes de pH des échantillons de liquides du rumen provenant de la diète à concentration élevée et du groupe témoin étaient acides (pH 5,3) et aromatiques (pH 6,1), respectivement. Le pourcentage de l'effet du pyrophosphate de thiamine moyen dans le groupe de la diète à concentration élevée (47,2 \pm 3.2) était considérablement supérieur à celui du groupe témoin (19,53 \pm 2,5) (P < 0,001). L'étude a fourni la preuve d'un effet de pyrophosphate de thiamine chez les bovins d'engraissement consommant une diète à concentration élevée.

(Traduit par Isabelle Vallières)

Can Vet J 2010;51:1251-1253

Introduction

R uminants with a functional rumen are considered to have no dietary thiamine requirements due to extensive ruminal thiamine synthesis by rumen microbes (1,2). Whole grain feeds, widely used in ruminant nutrition, are also important thiamine sources (3). However, thiamine deficiencies do develop in

Department of Internal Medicine, Veterinary Faculty, Firat University, 23119 Elazig, Turkey.

Address all correspondence to Dr. Tolga Karapinar; e-mail: tolgakarapinar@yahoo.com

Use of this article is limited to a single copy for personal study. Anyone interested in obtaining reprints should contact the CVMA office (hbroughton@cvma-acmv.org) for additional copies or permission to use this material elsewhere. ruminants under certain conditions such as ruminal fermentative disorders, ruminal microbial thiaminase production, intake of inactive thiamine analogs, and consumption of excessive sulfur from feedstuffs or water (4–9). There is a growing awareness that high performing cattle may have greater needs of B vitamins than can be ruminally produced (10).

Ruminal acidosis, an important ruminal fermentative disorder, varies from peracute life-threatening forms to chronic illness (11,12). Acute ruminal acidosis develops following excessive consumption of readily fermentable carbohydrates over a short period, resulting in rapid fermentation with production of large amounts of lactic acid. It is characterized by a dramatic reduction in ruminal pH (\leq 5), metabolic lactic acidosis, and overt clinical signs. Chronic ruminal acidosis (CRA) may occur when ruminants are fed excessive quantities of concentrate over a prolonged period of time, rather than being suddenly

exposed without adequate adaptation. In this condition, the rumen microbial population adapts to the high-grain ration (11). Ingested feedstuffs are fermented rapidly and volatile fatty acid concentrations are increased in the rumen. Due to the high concentrations of volatile fatty acids, the rumen fluid pH usually ranges from 5.0 to 5.5. The buffering action provided by chewing and saliva production is decreased because the diet is lower in fiber and higher in concentrate (11,13). However, lactic acid does not accumulate because it is metabolized by the bacteria. The major clinical signs of CRA are reduced feed intake and rumen hypomotility; therefore, clinical diagnosis of CRA is overlooked. Chronic ruminal acidosis, however, may lead to severe pathological conditions such as liver abscesses, chronic laminitis, and cerebrocortical necrosis (CCN) as a result of rumenitis, increased histamine levels, and thiamine deficiency, respectively (13-16). Although there have been several studies on liver abscesses and chronic laminitis, data on thiamine deficiency due to CRA are lacking. The aim of this study was to investigate whether or not thiamine deficiency takes place in feedlot cattle fed high concentrate diets.

Materials and methods

Animals

Fifty 1- to 2-year-old feedlot cattle fed a high concentrate diet (HCD group) and 15 age-matched healthy feedlot cattle on a low concentrate diet (control group) were used. The cattle in the HCD group had been fed a diet of 75% cracked barley, 8% bran, 7% cotton seed meal, and 10% straw for at least 3 mo. The cattle in the control group had been fed a diet of 30% cracked barley, 10% bran, 10% sugar beet pulp, 10% cotton seed meal and 40% straw.

Rumen fluid collection and examination

Rumen fluid samples were obtained by rumenocentesis. Ruminal fluid pH was determined with a portable pH meter (pH 340i/ SET; WTW Wissenchaftlich, Germany). Odor of the samples was classified as aromatic (normal) or acidic (abnormal).

Blood sample collection and erythrocyte transketolase test

Blood samples were collected from a jugular vein into heparincontaining tubes. To hemolyze the cells, 3 mL double distilled water was added to 1 mL of blood and mixed by vortex for 1 min. The hemolysates were stored at -20° C until they were analyzed. Erythrocyte transketolase enzyme activity, and hence thiamine pyrophosphate (TPP) effect, were determined according to Clausen's colorimetric method (17).

Statistical analyses

Statistical analyses were performed using SPSS for Windows (SPSS, version 10; Microsoft, Chicago, Illinois, USA). Group means were compared using Student's *t*-test.

Results

Ruminal fluid samples in the HCD and control groups were acidic (pH 5.3) and aromatic (pH 6.1), respectively. The mean TPP effect in the HCD and control groups is given in Table 1.

The mean TPP effect in the HCD group (47.2% \pm 3.2) was significantly higher than in the control group (19.5% \pm 2.5) (*P* < 0.01).

Discussion

Thiamine plays a central role in energy-yielding metabolism, especially the metabolism of carbohydrate. The principal function of thiamine in all cells is as coenzyme cocarboxylase or TPP. Thiamine pyrophosphate is the coenzyme for 3 important enzyme complexes: pyruvate dehydrogenase in carbohydrate metabolism; α-ketoglutarate dehydrogenase in the citric acid cycle; and the branched-chain keto-acid dehydrogenase involved in the metabolism of leucine, isoleucine, and valine. It is also a coenzyme for transketolase, in the pentose phosphate pathway (18). Independent of its coenzyme function, thiamine also has specific roles in neurophysiology. Thiamine deficiency results in a wide range of clinical effects from anorexia to CCN (1,3). Although there has been no difficulty in diagnosis of CCN based on clinical and necropsy findings, determination of mild thiamine deficiencies causing only anorexia (an important economic loss in the feedlot industry) may present a diagnostic challenge.

The transketolase test is the most reliable indicator for assessment of active thiamine status (1,19). The activation of apo-transketolase in erythrocyte lysate by thiamine diphosphate added in vitro has been accepted as an index of the thiamine status (18). When the transketolase test is used as the indirect variant, the parameter is the reactivation effect, that is, the TPP effect (1). Thiamine deficiency causes a decrease in transketolase activity and an increase in the TPP effect in the erythrocytes. The mean TPP effect of clinically healthy cattle has been reported as 15.5%, 19%, 23.2%, and 25%, by Clausen (17), Dabak and Gul (20), Bogin et al (21), and Rehm et al (22), respectively. Thiamine pyrophosphate effect values of more than 45% may be associated with thiamine deficiency (22). Bogin et al (21) reported that the mean TPP effect was 25% for normal sheep and 71% in sheep with suspected CCN. Jackman (19) concluded that the TPP effect was 25% in healthy sheep and 122% in sheep with CCN.

Chronic ruminal acidosis is a common condition in feedlot cattle especially those fed a high-grain ration and may establish intraruminal changes that lead to thiamine deficiency. Varying degrees of thiamine deficiency have been reported in sheep with acute ruminal acidosis and chronic ruminal acidosis (23,24). Mean values of 46.7% and 109.3% TPP effect have been reported in cattle and sheep with clinically acute ruminal acidosis, respectively (20,24). Dabak and Gul (23) reported that sheep with CRA had a mean 59.4% TPP effect. In the present study, the increased TPP effect (47.2 \pm 3.2%) in cattle fed the high concentrate diet might indicate that these animals suffered thiamine deficiency. Thiamine deficiency in both CRA and acute ruminal lactic acidosis may occur because of inadequate synthesis of thiamine (25), the bacterial production of thiaminase in the acidic ruminal fluid (4,5,15,25), or impaired absorption or increased metabolic demand for thiamine (as a co-factor of enzymes) (4). In addition, thiamine is destroyed or inactivated by thiaminases and a decrease in ruminal pH may result in the release of bacterial thiaminases (27). There is

Table 1. Mean levels of thiamine pyrophosphate (TPP) effect (%) with a high-concentrate diet (HCD) and healthy cattle (control group)

	HCD group $(n = 50)$	Range	Control group ($n = 15$)	Range	Р
TPP Effect (%)	47.2 ± 3.2	20.39-112.58	19.5 ± 2.5	4.56-33.28	< 0.01

a positive correlation between fecal thiaminase and TPP effect (28). Ruminal thiaminase activity and ruminal thiamine concentration were not determined in the present study.

Thiamine supplementation of ruminants fed under intensive fattening regimens has been one method to control the potential for subclinical thiamine deficiency (3). It has been reported that dietary thiamine supplementation in dairy cattle tended to increase milk and component production when dietary concentrations of neutral and acid detergent fiber were lower and nonfiber carbohydrate was higher than recommended (29). The effect of dietary thiamine supplementation on fattening performance in feedlot cattle fed a high-concentrate diet should be investigated.

In conclusion, the study has shown that the TPP effect is higher in feedlot cattle fed a high-concentrate diet compared with cattle fed a low-concentrate ration.

Acknowledgment

This research was supported by The Scientific and Technological Research Council of Turkey (TUBITAK) project number 106O519. cvj

References

- Braunlich K, Zintzen H. Vitamin B₁ in animal nutrition. Basel, Hoffmann-La Roche 1976; Number: 1593.
- Ewan RC. Vitamins. In: Swenson MJ, Reece WO, eds. Duke's Physiology of Domestic Animals. London: Cornell Univer Pr, 1993: 503–516.
- 3. McDowell LR. Vitamins in Animal Nutrition. San Diego: Academic Pr, 1989:155–183.
- 4. Edwin EE, Jackman R. Thiaminase I in the development of cerebrocortical necrosis in sheep and cattle. Nature 1970;228:772–774.
- Loew FM, Dunlop RH. Induction of thiamine inadequacy and polioencephalomalacia in adult sheep with amprolium. Am J Vet Res 1972; 33:2195–2205.
- Sheereve JE, Edwin EE. Thiaminase-producing strains of Cl. Sporogenes associated with outbreaks of cerebrocortical necrosis. Vet Rec 1974; 94:330.
- Gooneratne SR, Olkowski AA, Klemmer RG, Kessler GA, Christensen DA. High sulfur related thiamine deficiency in cattle: A field study. Can Vet J 1989;30:139–146.
- Dabak M, Gul Y. Investigation on the thiamine deficiency in cattle with dietary indigestion. F U Saglik Bil Dergisi 1996;10:345–352.
- 9. Gould DH. Polioencephalomalacia. J Anim Sci 1998;76:309-314.

- Santschi DE, Berthiaume R, Matte JJ, Mustafa AF, Girard CL. Fate of supplementary B-vitamins in the gastrointestinal tract of dairy cows. J Dairy Sci 2005;88:2043–2054.
- Garry FB. Indigestion in ruminants. In: Smith BP, ed, Large Animal Internal Medicine. St. Louis, Missouri: Mosby, 2002:722–747.
- Kleen JL, Hooijer GA, Rehage J, Noordhuizen JP. Subacute ruminal acidosis (SARA): A review. J Vet Med A Physiol Pathol Clin Med 2003;50:406–414.
- Owens FN, Secrist DS, Hill WJ, Gill DR. Acidosis in cattle: A review. J Anim Sci 1998;76:275–286.
- Brent BE. Relationship of acidosis to other feedlot ailments. J Anim Sci 1976;43:930–935.
- Nocek JE. Bovine acidosis: Implications on laminitis. J Dairy Sci 1997;80:1005–1028.
- Randhawa SS, Ahuja AK, Rathor SS. Effect of lactic acidosis on histamine and thiamine levels in buffalo calves. Indian J Anim Sci 1988;58:1019–1023.
- Clausen HH. The transketolase test: A diagnostic means for the evaluation of subclinical and clinical thiamine deficiency states in cattle. Dtsch Tierar Wochenschr 1977;84:453–492.
- Bender DA, Mayes PA. Vitamins & Minerals. In: Murray RK, Granner DK, Mayes PA, Rodwell VW, eds, Harper's Biochemistry. New York: Lange Medical Books, 2003:481–498.
- Jackman R. The diagnosis of CCN and thiamine deficiency in ruminants. Vet Ann 1985;25:71–77.
- Dabak M, Gul Y. Investigation on the thiamine deficiency in cattle with dietary indigestion. F U Saglik Bil Dergisi 1996;10:345–352.
- Bogin E, Soback S, Immelman A. Transketolase activity in the blood of cattle and sheep in relation to thiamine deficiency. Zentralbl Veterinarmed A 1985;32:135–139.
- Rehm WF, Zerobin K, Christeller S, Kunovits G, Weiser H. Diagnosis of clinical vitamin B1 deficiency in cattle. Berl Münch Tiearar Wochenschr 1971;84:64–67.
- Dabak M, Gul Y. Thiamine deficiency in sheep with chronic rumen acidosis. Vet Rec 2004;154:58–59.
- Karapinar T, Dabak M, Kizil O, Balikci E. Severe thiamine deficiency in sheep with acute ruminal lactic acidosis. J Vet Intern Med 2008;22:662–665.
- 25. Pierson RE, Jensen R. Polioencephalomalacia in feedlot lambs. J Am Vet Med Assoc 1975;166:257–259.
- Dunlop RH. Polioencephalomalacia (cerebrocortical necrosis). In: Aiello SE, ed. Merck Veterinary Manual. Philadelphia: Merck & Co, 1998:960–963.
- Brent BE. Relationship of acidosis to other feedlot animals. J Anim Sci 1976;43:930–935.
- Ramos JJ, Marca C, Loste A, Garcia de Jalon JA, Fernandez A, Cubel T. Biochemical changes in apparently normal sheep from flocks affected by polioencephalomalacia. Vet Res Commun 2003;27:111–124.
- Shaver RD, Bal MA. Effect of dietary thiamin supplementation on milk production by dairy cows. J Dairy Sci 2000;83:2335–2340.