Taurine deficiency in Newfoundlands fed commercially available complete and balanced diets

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Objective—To determine taurine status in a large group of Newfoundlands related by environment, diet, or breeding to a dog with dilated cardiomyopathy and taurine deficiency.

Design—Prospective study.

Animals—19 privately owned Newfoundlands between 5 months and 11.5 years old that had been fed commercial dry diets meeting established nutrient recommendations.

Procedure—Diet histories were obtained, and blood, plasma, and urine taurine concentrations and plasma methionine and cysteine concentrations were measured. In 8 dogs, taurine concentrations were measured before and after supplementation with methionine for 30 days. Ophthalmic examinations were performed in 16 dogs; echocardiography was performed in 6 dogs that were taurine deficient.

Results—Plasma taurine concentrations ranged from 3 to 228 nmol/mL. Twelve dogs had concentrations < 40 nmol/mL and were considered taurine deficient. For dogs with plasma concentrations < 40 nmol/mL, there was a significant linear correlation between plasma and blood taurine concentrations. For dogs with plasma concentrations > 40 nmol/mL, blood taurine concentrations did not vary substantially. Taurine-deficient dogs had been fed lamb meal and rice diets. Retinal degeneration, dilated cardiomyopathy, and cystinuria were not found in any dog examined for these conditions. The taurine deficiency was reversed by a change in diet or methionine supplementation.

Conclusions and Clinical Relevance—Results indicate a high prevalence of taurine deficiency among an environmentally and genetically related cohort of Newfoundlands fed apparently complete and balanced diets. Blood taurine concentrations indicative of taurine deficiency in Newfoundlands may be substantially less than concentrations indicative of a deficiency in cats. (*J Am Vet Med Assoc* 2003;223:1130–1136)

Dilated cardiomyopathy (DCM) is 1 of the most common acquired cardiovascular diseases of dogs. The underlying cause of the condition is unknown, but several contributing factors have been identified. For instance, the high prevalence of DCM in certain families of dogs and in specific breeds suggests there may be a genetic basis with possibly many pathways leading to a common end point. Viral infection, immune-mediated disorders, arrhythmias, toxins, and nutritional deficiencies are also cited as possible causes of DCM.[1]

Of the nutritional deficiencies cited as possible causes of DCM, taurine deficiency seems to be the most likely. Inadequate provision of dietary taurine results in DCM in cats,[2] but dogs are generally not recognized as having a need for dietary taurine, because dogs are able to synthesize taurine from the dietary sulfur amino acids cysteine and methionine.[3] Nevertheless, the authors have found evidence of taurine deficiency in dogs, some of which also had DCM. For instance, we have examined 2 unrelated dogs fed a tofu-based diet that was low in protein yet met published protein and other nutrient requirements.[4] Taurine deficiency in these dogs was attributed to the fact that the primary dietary protein source was soybean curd. Soybean protein is known to be low in sulfur amino acids relative to meat proteins and has been shown to accelerate loss of bile acids.[5] In dogs, as in cats, bile acids are conjugated exclusively with taurine.[6]

Similarly, Sanderson et al[7] found a substantial decrease in plasma taurine concentrations in Beagles fed an energy-dense, protein-restricted (10% protein on a dry matter basis) diet for 48 months,[8] and 1 of these dogs developed DCM. Feeding a commercial therapeutic diet similar to that used by Sanderson et al[7] may have induced DCM in Dalmatians, but although DCM was identified, diet-induced taurine deficiency was not clearly identified. American Cocker Spaniels are reported to develop taurine-deficiency DCM,[9] but oral treatment with taurine does not appear to reverse the condition as completely as it does in cats.

Cardiologists in private practice and at the University of California, Davis, Veterinary Medical Teaching Hospital have recently brought to our attention several dogs with low blood taurine concentrations and DCM in which large body size and diet

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appeared to be contributing to the taurine deficiency.[10] One of these was a neutered male adult Newfoundland, and the purpose of the study reported here was to determine taurine status in a large group of Newfoundlands related by environment, diet, or breeding to this dog.

Materials and Methods

Dogs—Eight male (5 neutered) and 11 female (6 spayed) privately owned Newfoundlands were included in the study. Dogs weighed between 30 and 59 kg (66 and 130 lb) and were between 5 months and 11.5 years old. All dogs were related by environment, diet, or breeding to a 10-year-old 47-kg (103-lb) neutered male Newfoundland with DCM and a low plasma taurine concentration (24 nmol/mL).

Evaluation of taurine status—For 15 of the 19 dogs, blood samples were collected for determination of blood and plasma taurine concentrations initially and again 18 weeks later. Blood samples were collected from the remaining 4 dogs only once. On days blood samples were collected, dogs were weighed, and a diet history, including the name and amount of the commercial diet and any supplements or treats that were fed, was obtained.

Once taurine deficiencies were identified, owners were requested to submit free-catch urine samples for analysis prior to instituting any diet change. Owners of 15 dogs submitted urine samples. Owners of 4 dogs changed their dogs' diet to a different commercial dry diet between the time of initial and follow-up blood sample collections because of concerns that the original diet may have been causing a taurine deficiency. Owners of 2 of these dogs also administered taurine to their dogs.

Laboratory analyses—For determination of blood and plasma taurine concentrations, cephalic vein blood samples (3 mL) were collected and immediately placed on ice. Plasma was obtained from a portion (2 mL) of each sample within 3 hours by centrifugation for 10 minutes at 1,200 × *g*. Free-catch urine samples were stored frozen until submitted for analysis.

Prior to analysis, freshly separated plasma samples were extracted with 6% sulfosalicylic acid to reduce binding of free cysteine with protein.[11] Blood samples were diluted 1:3 with water, and resulting solutions were frozen and thawed twice. Diluted blood samples were then extracted with 6% sulfosalicylic acid, as described for plasma samples. Urine samples were thawed and extracted with 6% sulfosalicylic acid also.

Taurine concentrations in blood, plasma, and urine samples were determined with automated amino acid analyzers that incorporated cation-exchange high-pressure liquid chromatography separation and ninhydrin-reactive colorimetric detection.[a] Plasma taurine concentrations < 40 nmol/mL were considered evidence of a taurine deficiency; this concentration was lower than the lowest concentration reported in a previous study[12] of healthy dogs. Concentrations of methionine, cystine, and other amino acids in selected plasma and urine samples were determined along with taurine concentration. Because cysteine in plasma samples was assumed to be oxidized, cysteine concentrations were extrapolated from half-cystine concentrations. The concentration of creatinine in urine samples was determined with a commercial kit.[b]

Blood samples anticoagulated with EDTA were submitted to the Josephine Deubler Genetic Disease Testing Laboratory for testing for a cystinuria-associated mutation identified in Newfoundlands.[13]

Cardiac and ophthalmic examinations—Two-dimensional and M-mode echocardiography were performed in 2 males and 4 females with initial plasma taurine concentrations < 40 nmol/mL (mean ± SD, 9 ± 1 nmol/mL). Median age was 5 years (range, 10 months to 8 years), and median body weight was 49 kg (108 lb; range, 30 to 59 kg). The remaining dogs were not available for echocardiographic examinations.

Complete ophthalmic examinations, including slit lamp biomicroscopy, direct and indirect ophthalmoscopy, **Schirmer tear tests (STTs)**,[c] and applanation tonometry,[d] were performed in 6 male and 10 female dogs. Median age was 4.7 years (range, 1.9 to 9.5 years). The remaining dogs were not available for ophthalmic examinations.

Methionine supplementation—Eight dogs with plasma taurine concentration < 40 nmol/mL but without clinical (n = 8) or echocardiographic (3) signs of myocardial failure were treated with methionine. Methionine was administered once daily for 30 days at a rate of 3 g of methionine/kg of commercial dry diet fed (approx 0.7 g of methionine/1,000 kcal fed). Owners were instructed to dissolve crystalline L-methionine[e] in warm water (0.25 teaspoon per cup of water) and mix the solution with food (1 cup of solution per cup of food) so that the methionine was absorbed in a portion of the diet. Owners reported that all food wetted with the solution was ingested. Owner compliance was evaluated by measuring the amount of methionine returned by the owners after the 30-day supplementation period. Cephalic vein blood samples for amino acid analysis were collected within 1 week before and on the last day of the supplementation period.

Dietary change—After results of the methionine trial were evaluated, owners with dogs that had plasma taurine concentrations < 40 nmol/mL were contacted, and a dietary change was recommended. Owners were asked to substitute their current commercial dry diet with a commercial dry diet identified in the study to be consistently associated with plasma taurine concentrations > 40 nmol/mL.

Statistical analyses—Paired *t* tests were used to compare plasma taurine, methionine, and cysteine concentrations obtained before and after methionine supplementation and before and after a dietary change. Logarithmic transformation of urine taurine-to-creatinine ratio data was conducted prior to analysis with Student *t* tests and correlation analyses.

Results

Taurine status—Initial plasma taurine concentrations for the 19 dogs ranged from 3 to 228 nmol/mL (**Fig 1**). Twelve dogs had plasma taurine concentrations < 40 nmol/mL. The 2 dogs with the highest plasma taurine concentrations (228 and 166 nmol/mL) were receiving taurine (1 g/d, PO) at the time blood samples were collected. Taurine administration had been prescribed for treatment of DCM in 1 of these dogs; in the other dog, taurine was given by the owner as a perceived beneficial gestational supplement.

Initial blood taurine concentrations in the 19 dogs ranged from 45 to 400 nmol/mL. Plotting blood taurine concentrations against plasma taurine concentrations revealed a curvilinear relationship between blood and plasma taurine concentrations (**Fig 2**). For plasma concentrations < 40 nmol/mL, blood taurine concentration increased as plasma taurine concentration increased. For plasma concentrations > 40 nmol/mL, blood taurine concentration increased, but less so, as plasma concentration increased.

The range for plasma taurine concentration was wider than ranges for plasma methionine (34 to 69 nmol/mL) and cysteine (35 to 88 nmol/mL) con-

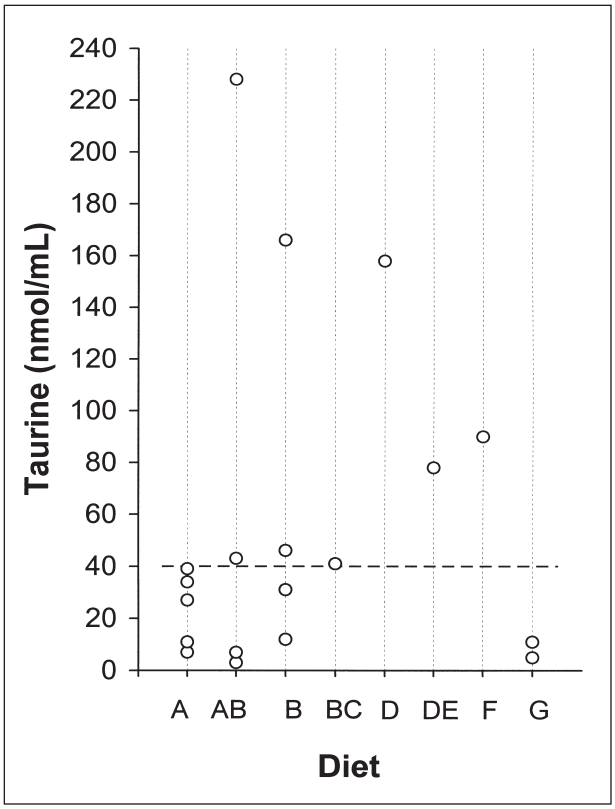

Figure 1—Scatterplot of plasma taurine concentrations in 19 Newfoundlands fed 1 of 7 commercially available complete and balanced diets (or a combination of 2 diets). The horizontal, dashed line represents the concentration below which dogs were considered to have a taurine deficiency.

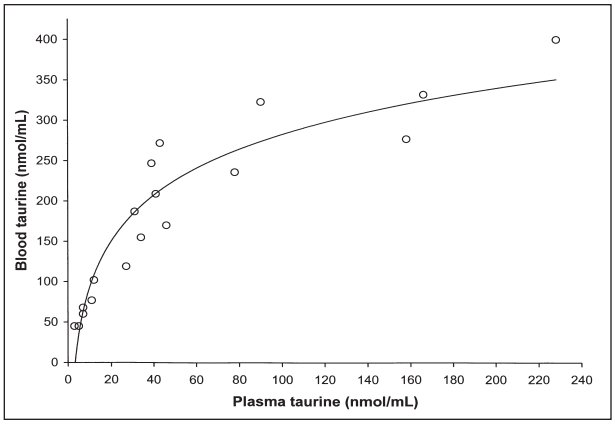

Figure 2—Scatterplot of blood versus plasma taurine concentrations in 19 Newfoundlands fed commercially available complete and balanced diets. The solid line represents the logarithmic, least-squares fit (blood taurine concentration = 83 ln [plasma taurine concentration] – 98; $r = 0.95$).

centrations. Plasma taurine concentrations were not significantly correlated with plasma methionine or cysteine concentrations. Mean ± SD plasma methionine concentration was 51 ± 8 nmol/mL; mean plasma cysteine concentration was 53 ± 13 nmol/mL.

Urine taurine concentration—Urine samples were obtained from 15 dogs prior to any changes in the dogs' diet. Plasma taurine concentrations for these dogs ranged from 2 to 53 nmol/mL (mean ± SD, 22 ± 17 nmol/mL). The urine taurine-to-creatinine ratio for these dogs ranged from 0.2 to 290 mmol of taurine/mol of creatinine. The ratio was not significantly correlated with plasma taurine concentration.

Diet history—Diet histories indicated that for all 19 dogs, > 90% of metabolic energy requirements were provided by commercial dry (extruded) diets. Seven commercial diets were fed to the dogs, and inspection of labels on bags of these diets indicated that taurine was not an added ingredient. Six diets were produced by 2 manufacturers with substantial market distribution. Most dogs had been fed these diets for long periods, ranging from 4 months to 4 years. The exceptions were 5 dogs whose owners had elected to switch diets within 2 weeks prior to collection of blood samples for determination of taurine status. Various mixtures of the new and former diets were fed during the switch, but in all dogs, the former diet had been fed for a long period. Bag labels indicated that the 7 diets met recommended nutrient profiles or passed minimum feeding protocol tests recommended by the Association of American Feed Control Officials (**Table 1**).[14]

Seven of the 19 dogs were principally fed 1 of 2 lamb and rice formulations (diets A and G) produced by a single manufacturer. All 7 of these dogs were found to have plasma taurine concentrations < 40 nmol/mL. Inspection of ingredient lists for these 2 diets indicated that lamb meal was the primary protein source in both. Four other dogs were fed a lamb and rice formulation produced by another manufacturer (diet B), but inspection of the ingredient list indicated that lamb, rather than lamb meal, was the primary protein source. Two of these 4 dogs had plasma taurine concentrations < 40 nmol/mL.

Plasma taurine concentration was determined twice, 18 weeks apart, in 11 of the 19 dogs. In 4 dogs being fed a lamb meal and rice diet (diet A), the diet was not changed between the initial and follow-up samples, and in 1 dog being fed a lamb meal and rice diet (diet A), the diet was changed to another lamb meal and rice diet (diet G). In the 6 other dogs, the diet was changed to another commercial diet. Plasma taurine concentrations in the 5 dogs that continued to receive lamb meal and rice diets did not change significantly (mean ± SD, 19 ± 14 vs 20 ± 13 nmol/mL; **Fig 3**). However, plasma taurine concentration was significantly ($P < 0.02$) increased in the 6 dogs in which the diet was changed (26 ± 18 vs 66 ± 13 nmol/mL). In these dogs, diet changes were not associated with significant changes in plasma concentrations of methionine or cysteine.

Effect of methionine supplementation—For owners of the 8 dogs treated with methionine, measurement of the amount of methionine remaining after the 30-day supplementation period indicated that between 63 and 92% of the methionine prescribed had been used. Mean plasma taurine concentration was significantly ($P < 0.01$) increased, but the absolute increase varied among dogs. Mean ± SD plasma taurine concentration before supplementation was 13 ± 5 nmol/mL, and mean concentration following the 30-day supplementation period was 41 ±

Table 1—Proximate analysis of commercial dry diets fed to 19 Newfoundlands, 12 of which had taurine deficiency

	Diet						
Variable	**A**	**B**	**C**	**D**	**E**	**F**	**G**
Crude protein (%)	24	27	24	26	26	34	17
Crude fat (%)	14	18	12	17	19	23	7
NFE* (%)	48	48	NA	39	42	34	63
Crude fiber (%)	2	2	6	2	2	3	7
Ash (%)	10	6	NA	7	6	6	6
ME (kcal/g)	3.8	4.1	3.3	3.9	4.2	4.3	3.4
Taurine† (mg/kg)	269	425	625	ND	ND	344	81

Percentages were determined on a dry matter basis.
*Reported or calculated by difference. †Determined with an automated amino acid analyzer on water extracts of diets.
NFE = Nitrogen-free extract. NA = Not available. ME = Metabolizable energy. ND = Not determined.

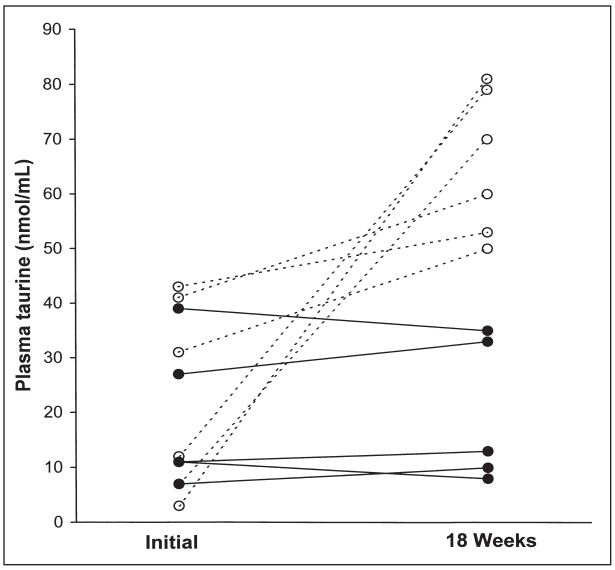

Figure 3—Plasma taurine concentrations in 11 Newfoundlands with taurine deficiency. Concentrations were measured before and 18 weeks after the diet was changed in 6 dogs (open circles); in the remaining 5 dogs, the diet was not changed (closed circles).

22 nmol/mL. Methionine supplementation also significantly ($P < 0.02$) increased plasma concentrations of methionine (56 ± 6 vs 77 ± 6 nmol/mL) and cysteine (50 ± 3 vs 61 ± 3 nmol/mL).

Cardiac examinations—None of the 6 dogs that underwent echocardiography had any clinical signs of cardiovascular disease, and results of echocardiography indicated that none of the dogs had DCM. In all dogs, wall thickness, left atrial, and aortic measurements were within reference limits. Five of the 6 dogs had left ventricular end diastolic and end systolic dimensions and shortening fractions within ranges expected for their body weights.[15] One dog, which was 6 years old, had a normal end diastolic dimension but high end systolic dimension. Shortening fraction for this dog was 22%, whereas shortening fractions for the other dogs ranged from 30 to 35%.

Ophthalmic examinations—Mean STT value (19.8 mm/min) and mean intraocular pressure (10.3 mm Hg) were within reference limits. None of the dogs had any fundic abnormalities, and ocular abnormalities that were seen appeared to be unrelated to taurine status. Ocular abnormalities included ectropion (4/16 dogs; 8/32 eyes), punctate spots of melanin on the posterior lens capsule (3/16 dogs; 5/32 eyes), prominent anterior vitreous strands (5/16 dogs; 10/32 eyes), and incipient cataracts (5/16 dogs; 10/32 eyes). Cataractous changes were localized to the anterior subcapsular, posterior subcapsular, or anterior cortical region. One dog, a 1-year-old sexually intact female, had bilateral posterior, polar, subcapsular, triangular cataracts. No age or sex predisposition was evident for any of the ocular abnormalities, except that all 4 dogs with ectropion were neutered females. Three dogs did not have any ocular abnormalities.

Cystinuria screening—Blood samples from 8 dogs with plasma taurine concentrations < 40 nmol/mL were submitted for screening for the genetic mutation associated with cystinuria in Newfoundlands. The nonsense mutation in the *SLC3A1* gene of Newfoundlands shown to cause cystinuria was not found in any of the samples. The dibasic amino aciduria pattern characteristic of cystinuria was not observed in amino acid profiles of urine from these dogs.

Discussion

Taurine participates in many physiologic processes, including modulation of cellular calcium flux and neural excitability, osmoregulation, detoxification, membrane stabilization, and bile acid conjugation. Although taurine is clearly required for normal cardiac function,[2,16,17] the underlying mechanism for the requirement is unknown.[18] The most probable mechanisms for development of DCM in response to taurine deficiency involve disturbances in osmoregulation, calcium regulation, or free radical inactivation.[19] Taurine concentrations in tissues, such as the myocardium,[19,20] skeletal muscle,[21] and retinal epithelium,[22] decrease during periods of taurine deficiency, and the decrease is generally reflected in blood and plasma taurine concentrations. As a consequence, taurine deficiency may be conveniently detected through measurement of blood and plasma taurine concentrations.

Clinical surveys and prospective studies[23,24] in cats show that plasma taurine concentrations < 25 nmol/mL indicate taurine deficiency sufficient to cause DCM. Evaluation of plasma taurine concentrations in dogs with cardiac valvular and myocardial disease has led some investigators to suggest that plasma taurine concentrations ≤ 25 nmol/mL indicate taurine deficiency in dogs.[12] Plasma taurine concentrations in dogs in the present study varied greatly, ranging from 2 to 228 nmol/mL. Although 9 of the 19 dogs had plasma taurine concentrations < 25 nmol/mL, which could be considered indicative of a taurine deficiency, none had DCM and only 1 had echocardiographic evidence of reduced myocardial function.

In cats, it is not unusual to find low blood or plasma taurine concentrations in the absence of DCM. When taurine is excluded from the diet of cats, plasma taurine concentrations decrease so quickly that plasma taurine concentrations < 25 nmol/mL can be observed in as little

as 4 weeks after the dietary change.[23] However, development of DCM is typically delayed. Pion et al,[2] for instance, maintained 11 cats on a purified diet low in taurine for 4 years and found that only 2 developed echocardiographic evidence of myocardial failure. Similarly, Sanderson et al[7] found that only 2 of 10 Beagles depleted of taurine as a result of dietary protein restriction for more than 12 months had evidence of myocardial failure. The duration of taurine deficiency in the Newfoundlands in the present study was not known; however, if diet induced the taurine deficiencies, then exposure time, determined on the basis of diet history, ranged from 4 months to 4 years.

The low prevalence of cardiac dysfunction in these dogs may have reflected a lack of interaction with a necessary contributing factor.[24] Quantitative genetic evaluation of cats in a large cattery has shown a heritable susceptibility to DCM and, presumably, taurine-deficiency DCM.[25] Heritable limitations in taurine absorption, metabolism, and excretion are suggested to explain the genetic variation in development of DCM.

Another clinical consequence of taurine deficiency in cats is central retinal degeneration. Although taurine deficiency is known to cause central retinal degeneration and DCM in cats, both conditions are not always observed, and some taurine-deficient cats have both conditions, whereas some have only DCM and others have only central retinal degeneration. The variation in clinical manifestation of taurine deficiency is suggested to be a result of the variable presence of contributing factors.[25] Dietary zinc may be an example of these contributing factors. Disruption of retinal tissue associated with taurine deficiency is exacerbated by feeding rats diets low in zinc.[26]

Measurement of blood and plasma taurine concentrations is recommended for identification of taurine deficiency in cats,[27] as blood concentration may substantiate a finding of taurine deficiency when plasma concentration is equivocal. In taurine-replete animals, most of the taurine in the blood is in granulocytes and platelets,[28] with approximately a quarter of the taurine in blood in the plasma fraction.[29] Because of this, blood taurine concentration is only slightly altered after feeding, whereas changes in plasma taurine concentration may be substantial, particularly in taurine-depleted animals.[27] A variable amount of taurine leaks from the formed elements of the blood into plasma during clotting, and this leakage may substantially increase plasma taurine concentration and confound interpretation of results. Blood taurine concentrations are not subject to this bias.

Extensive research on taurine deficiency in cats has shown that taurine deficiency is typically indicated when blood taurine concentration is < 250 nmol/mL.[18] This finding has led some investigators to suggest that the same limit might indicate taurine deficiency in dogs. However, results of the present study indicate that blood taurine concentrations may be as low as 200 nmol/mL in taurine-replete dogs. An inflection in the curvilinear relationship between plasma and blood taurine concentrations was found at a plasma concentration of approximately 40 nmol/mL. In dogs with plasma concentrations greater than this value, blood taurine concentrations increased little as plasma taurine concentration increased, and the variance among blood taurine concentrations was low. This inflection point may indicate when the formed elements of the blood are at their taurine-holding capacity, whereas at plasma taurine concentrations < 40 nmol/mL, the granulocytes, platelets, and other blood elements may be deficient in taurine. This inflection point is near to the lowest plasma taurine concentration (44 nmol/mL) observed by Kramer et al[12] in their survey of clinically normal dogs. Inserting a plasma taurine concentration of 40 nmol/mL into the regression equation calculated for the dogs in the present study yields a blood taurine concentration of 208 nmol/mL.

Two findings support the conclusion that taurine deficiencies in the dogs in the present study were induced by diet. First, 8 of the 9 dogs with plasma taurine concentrations < 25 nmol/mL had been fed 1 of 2 diets with lamb meal as the principal protein source for a prolonged period. Second, plasma taurine concentrations increased when the diet was changed but were not altered with continued feeding of the lamb meal diets. Low bioavailabilities of methionine and cysteine in the lamb meals were initially postulated as the cause of the taurine deficiencies in these dogs. Processing during the creation of meat meals variably reduces protein quality, and low ileal digestibility of protein (67%) and cysteine (29%) in dogs fed low-ash lamb meal has been reported.[30] If this were the case, however, we would have expected the low bioavailabilities of methionine and cysteine to be reflected in low plasma concentrations.[31] However, plasma cysteine and methionine concentrations did not appear to be low in taurine-deficient dogs in the present study, compared with published values for healthy dogs,[32] nor were they significantly different from concentrations observed in taurine-replete dogs. Hence, availability of the sulfur amino acid precursors for the synthesis of taurine appeared to be adequate.

Addition of methionine to the lamb meal diets appeared to result in a substantial improvement in the taurine status of taurine-deficient dogs in the present study. Because a placebo group was not included in evaluating the effects of methionine supplementation, it is possible that the improved taurine status resulted from coincidental environmental effects. The amount of crystalline methionine administered to these dogs was approximately 0.3%, and this level of supplementation was not considered excessive, given that the minimum recommendation for dietary methionine and cystine is 0.43% of dry matter.[14] The amount of supplemental methionine given to these dogs was roughly equivalent to what would have been provided by doubling the dietary protein concentration. Taurine status may have been improved by increasing the amount of precursor for taurine synthesis and stimulating the rate-controlling enzyme in the synthesis pathway (cysteine dioxygenase[33]), because observed increases in plasma concentrations of cysteine and taurine following methionine supplementation were consistent with an increase in the supply of methionine substrate. However, an enigmatic finding in the taurine-deficient dogs was that a dietary change improved taurine status

without significantly affecting plasma methionine or cysteine concentration. An explanation for this may be that the lamb meal and rice formulations were sufficient in methionine and cysteine, but other dietary factors precluded adequate synthesis of taurine. Dietary manipulations are reported to modulate activities of enzymes that compete for cysteine and its metabolites,[34] but none of these manipulations would appear to implicate a dietary factor that could clearly explain the present results.

A diet-induced, extraordinary loss of taurine also might explain the incongruity of finding taurine deficiency with normal plasma methionine and cysteine concentrations. Taurine is obligatorily lost in urine and in bile acids that are not recovered by means of enterohepatic circulation.[35] Urinary or gastrointestinal tract loss of taurine could theoretically be so great that synthesis of taurine would be insufficient to match losses, even when a complete and balanced diet was provided. Extraordinary urinary taurine losses cannot be ruled out as a cause for the present observations, in that as much as the urine taurine-to-creatinine ratio is an indicator of urine taurine loss,[35] this ratio appeared to be too high in some dogs in the present study, given their plasma taurine concentrations. Urine taurine-to-creatinine ratios in the present study were within the range of ratios reported for taurine-replete Beagles.[36,37] In taurine-deficient cats, urinary taurine losses are low because of the low concentration of taurine in plasma filtrate and the upregulation of renal tubular taurine transporters.[38] Thus, the cause for the higher-than-expected renal taurine losses in the present study was not apparent. Further investigation in which diet and husbandry are well controlled is needed before an importance can be assigned to urine taurine excretion in dogs with taurine deficiency.

Gastrointestinal tract loss of taurine is substantially affected by diet in cats.[39,40] This is of importance to taurine homeostasis in cats, because taurine balance studies show that taurine loss in the gastrointestinal tract is much greater than taurine loss in urine.[39,40] The magnitude of the gastrointestinal tract loss of taurine is reflected in the range (400 to 2,000 mg of taurine/kg of diet) of taurine requirements for feline diets.[41,42] If diet affects gastrointestinal tract loss of taurine in dogs as it does in cats, then the demand for taurine synthesis in dogs may vary greatly with diet, possibly by as much as 500%. It is conceivable that the diets associated with taurine deficiency in the Newfoundlands in the present study induced gastrointestinal tract loss of taurine that exceeded taurine synthesis. Factors that might have been responsible are rice bran and lamb meal used in the formulations. Recent research in cats has shown that the dietary taurine requirement is increased when either rice bran[43] or low-quality, heat-damaged protein[5,44,45] is added to purified diets. The effect in both cases appears to be a result of increased gastrointestinal tract taurine loss, the mechanism for which may be accelerated bile acid excretion caused by intestinal residue binding bile acids or promoting microbial destruction of taurine.

Results of this study indicate a high prevalence of taurine deficiency among an environmentally and genetically related cohort of Newfoundlands. Although DCM and retinal degeneration were not found, the absence of these signs does not mean the condition was without consequences. A finding of DCM and low plasma taurine concentration in a related Newfoundland prompted the present study, and poor reproduction of bitches, small litters, and short-statured puppies were anecdotally reported. Similar observations are associated with taurine deficiency in cats.[46] Blood taurine concentrations of dogs deemed taurine replete in the present study were > 250 nmol/mL, which is considerably less than blood taurine concentrations of taurine-replete cats. In the present study, the SD of blood taurine concentrations for dogs with plasma taurine concentrations > 40 nmol/mL was 7%. Subtracting 2 SDs from 208 nmol/mL (the blood taurine concentration calculated from the regression equation for a plasma concentration of 40 nmol/mL) yields 180 nmol/mL, and blood taurine concentrations less than this might be considered indicative of a taurine deficiency in dogs. Taurine deficiencies appeared to be induced by feeding lamb meal and rice diets in these dogs and were reversed by methionine supplementation, but the mechanism for taurine deficiency was not apparent. However, research on cats indicates that rice bran and lamb meal may have contributed to the deficiency by increasing gastrointestinal tract loss of taurine. Since these findings were first reported in an abstract, the manufacturers producing diets associated with taurine deficiency have added taurine to their formulations. This supplementation may prevent taurine deficiency, but not the disturbance that produced the deficiency. Thus, contributing dietary factors and the mechanisms of their effect should be determined.

[a]System 7300 and Model 121-M automated amino acid analyzers, Beckman Instruments Inc, Palo Alto, Calif.
[b]Diagnostic kit 555-A, Sigma Chemical Co, St Louis, Mo.
[c]Schirmer tear test, Schering-Plough Animal Health, Kenilworth, NY.
[d]Tono-Pen XL tonometer, Oculab, Glendale, Calif.
[e]Ajinomoto USA Inc, Raleigh, NC.

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