



Differences in Taurine Synthesis Rate among Dogs Relate to Differences in Their Maintenance Energy Requirement^{1–3}

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Abstract

Diet-induced (taurine deficiency) dilated cardiomyopathy is reported more in large than small dogs possibly because taurine biosynthesis rate (TBR) is lower in large than small dogs. The TBR in 6 mongrels (37.9 ± 2.1 kg) and 6 beagles (12.8 ± 0.4 kg) was determined from the fractional dilution rate of urinary [1,2-²H₂]-taurine, (d4-tau). All dogs were given a 15.6% protein, 0.60% sulfur amino acid (SAA) diet in amounts to maintain an ideal body condition score. After 3 mo, 14.6 mg/kg body weight of d4-tau was given orally and TBR determined from d4-tau to taurine ratio in urine collected each d for 6 d. Enrichments of d4-tau were determined by GC-MS. Thereafter, mongrels and beagles were paired by ranking of SAA intake per metabolic body weight per kg^{0.75}. Each pair received the same amount of diet/kg^{0.75} for 2 wk, then TBR was again determined. Concentrations of taurine in plasma, blood, and urine and concentrations of plasma thiols were measured during each TBR determination. In Expt. 1, TBR and taurine concentrations in plasma and urine of mongrels were lower ($P < 0.05$) than those of beagles. In Expt. 2, TBR and taurine concentrations in blood and plasma of mongrels were lower ($P < 0.05$) than beagles. Together, the results support the hypothesis that large compared with small dogs have lower TBR when fed diets near-limiting in dietary SAA, but adequate to maintain ideal body condition. J. Nutr. 137: 1171–1175, 2007.

Introduction

Dilated cardiomyopathy (DCM)⁷ is a disease of the myocardium with impaired systolic pumping function in the ventricles of the heart. Approximately 0.5% of dogs are diagnosed for DCM among all of the dogs admitted to veterinary teaching hospitals (1). Interestingly, it has been reported that large breed dogs are predisposed to developing DCM (2). The etiology for DCM has not been clearly elucidated; however, genetic predisposition, viral infection, immune-mediated disorders, toxin, arrhythmias, and nutritional deficiencies such as taurine deficiency or L-carnitine deficiency have been suggested as possible causes (3). Of the nutritional factors, taurine deficiency has gained attention because taurine deficiency in cats was shown to directly cause a DCM that was reversible by taurine supplementation (4).

Taurine (2-aminoethanesulfonic acid) is a beta, sulfur-containing, amino acid ubiquitously found in animals and reported in especially high concentrations in heart, brain, central nervous system, retina, olfactory bulb, and white blood cells (5). The physiological function of taurine in heart is not fully understood. Proposed mechanisms include osmoregulation, calcium regulation, and inactivation of free-radicals (6).

Taurine is synthesized from the sulfur amino acids, methionine and cyst(e)ine (7), by the activities of the enzymes, cysteine dioxygenase (EC 1.13.11.20) and cysteine sulfinic acid decarboxylase (EC 4.1.1.29) in animals, excluding most carnivores (8). Because of this, taurine is not considered as an essential nutrient in many species. However, it is known that generally, carnivores have a dietary requirement for taurine, and there is evidence that under certain dietary conditions dogs require dietary taurine. Sanderson et al. (9) found a significant decrease in plasma taurine concentration in healthy beagles fed a high-fat, protein-restricted (10% dry matter basis) diet that exceeded the NRC minimum protein requirement of maintenance in adult dogs (10). After feeding the diet for 48 mo, these investigators found 1 dog developed DCM. This indicated that prolonged provision of a protein-restricted diet, although above the minimum protein requirement, could result in taurine deficiency in dogs. More recently, Fascetti et al. (11) reported 12 cases of low blood taurine concentration and DCM in large-breed dogs given apparently nutritionally complete and balanced commercial diets. They suggested that body size may be a factor contributing to development of taurine deficiency in dogs.

¹ Supported by Center for Companion Animal Health (CACH), School of Veterinary Medicine, University of California, Davis and by Royal Canin, Aimargues, France.

² Author disclosures: K. S. Ko, no conflicts of interest; R. C. Backus, no conflicts of interest; J. R. Berg, no conflicts of interest; M. W. Lame, no conflicts of interest; and Q. R. Rogers, no conflicts of interest.

³ Supplemental Tables 1 and 2 and Supplemental Figures 1 and 2 are available with the online posting of this paper at jn.nutrition.org.

⁷ Abbreviations used: BCS, body condition score; BFM, body fat mass; BW, body weight; d4-tau, [1,2-²H₂]-taurine; DCM, dilated cardiomyopathy; LBM, lean body mass; MBW, metabolic body weight; MLBM, metabolic lean body mass; RLW, relative liver weight; SAA, sulfur amino acid; TBR, taurine biosynthesis rate; TTR, tracer to tracee ratio.

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Our research group recently found that plasma taurine concentration and taurine biosynthesis rate (TBR) in Newfoundland dogs, a giant dog breed, are substantially lower than those in beagles when both breeds are fed the same diet (12). We hypothesize that the greater incidence of taurine-deficiency DCM reported in large relative to small dogs is the result of lower TBR in large dogs. In the present study, we compare the abilities of large and small dogs to synthesize taurine when intake of diet is controlled to maintain ideal body condition and when intake is controlled to provide similar dietary sulfur amino acid (SAA) intake on a metabolic body weight (MBW, $\text{kg}^{0.75}$) basis.

Materials and Methods

Animals and diet. Husbandry and treatment of the dogs were in compliance with the NRC Guide for Laboratory Animals (13), and were approved by the Animal Use and Care Administrative Advisory Committee at University of California, Davis. Six sexually intact male beagles (12.8 ± 0.4 kg, 5–7 y) and 6 male mongrels (37.9 ± 2.1 kg, 5 intact and 1 neutered, 6–8 y), owned by the University, were designated small dogs and large dogs, respectively. The dogs were individually housed simultaneously in semi-open runs in the same building, and they received an allotment of diet each day that was completely consumed by the following day. Body weights (BW) and body condition scores (BCS) were determined each week.

All dogs were given the same, nutritionally complete and balanced, extruded dry-type diet produced for the study (Royal Canin). Dietary protein was limited to 15.6% to provide adequate but not excessive SAA to maintain nitrogen balance and provide for taurine biosynthesis (Table 1). The dietary protein content exceeded recommended allowance for maintenance of adult dogs (10% for 16.7 kJ/g metabolizable energy in the diet, dry matter basis) (10,14). Sulfur amino acid bioavailability of the diet was estimated by cecectomized rooster assay (15).

Expt. 1. For 3 mo, the amount of diet given to each dog was adjusted each wk, as needed, to achieve and maintain ideal BCS (5 on a 9 point scale) (16). After 2 mo, baseline venous blood and urine samples were collected, body composition determined, and 14.6 mg/kg BW of 99 atom % deuterated taurine ($[1,2\text{-}^2\text{H}_2]$ -taurine, d4-tau, CDN Isotopes) was given per os in a gelatin capsule wrapped in a marshmallow. Urine collection was repeated each morning before feeding for 5 d after administration of d4-tau. Concentrations of taurine in blood, plasma, and urine, and concentrations of total glutathione (reduced + oxidized), total cyst(e)ine (free plus bound to protein via a sulfhydryl bond), cysteinyl-glycine and homocysteine in plasma, and complete amino acid profiles (including cysteine and cystine not bound to protein) in plasma were determined as previously described (12). Urinary tracer (d4-tau) to tracee (taurine) ratio (TTR) for calculation of TBR of the dogs was determined by GC-MS.

TABLE 1 Composition and properties of the experimental diet¹

Crude protein, %	15.6
Acid hydrolyzed fat, %	23.2
Crude fiber, %	1.9
Ash, %	8.3
Metabolizable energy, ² kJ/g	18.0
Metabolizable energy from protein, %	12.7
Sulfur amino acid, %	0.60
Methionine	0.34
Cyst(e)ine	0.24
Taurine, mg/kg	280.0
Sulfur amino acid digestibility, %	77.9

¹ Ingredient list as provided by the diet manufacturer, Royal Canin: brewer's rice, rice bran, lamb-meal, poultry fat, lamb digest, and proprietary vitamin and mineral mixtures.

² Calculated using modified Atwater coefficients in the equation; metabolizable energy_{kJ/g} = [(crude protein_g × 3.5) + (ether extract_g × 8.5) + (nitrogen free extract_g × 3.5) + (crude fiber_g × 0)] × 4.19 (15).

Expt. 2. Control of diet presentation to maintain ideal BCS was continued after Expt. 1 so that SAA intake per MBW could be calculated for each dog. The dogs were then ranked from least to highest SAA intake per MBW, and large and small dogs were paired by rank of SAA intake per MBW to make 6 experimental pairs. The mean SAA intake per MBW for each pair was determined, and the quantity of diet it represented was given to the pairs each d for 2 wk. After 1 wk, blood, plasma, and urine were sampled, body composition determined, d4-tau administration and urine collection repeated, and biochemical analyses conducted, as described in Expt. 1.

Sample collection, processing, and analysis. Blood (~5 mL) was collected from the cephalic vein by venipuncture into heparinized syringes (~20 μL of sodium heparin solution, 1000 USP kU/L, Baxter Health Care). Urine (≥ 5 mL) was collected by free-catch before feeding.

Taurine concentrations in blood, plasma, and urine were determined by the method of Kim et al. (17) using an amino acid analyzer (12). To normalize urinary taurine concentration, urinary creatinine concentrations were determined with a commercial kit (Cold Stable, Pointe Scientific).

The GC-MS analysis and calculation of TBR from enrichment of TTR in urine were conducted using a modification (12) of the method of Fay et al. (18). MS of the deuterated taurine derivative revealed a unique fragment of 241 m/z, which was assumed to be an M+3 rather than the expected M+4 fragment. Deuterium on carbon adjacent to the sulfonate group of the taurine label was assumed to exchange with available protium during the derivatization step. Use of the M+3 fragment was justified because its fractional abundance increased linearly with increasing enrichment of the M+4 tracer in standards.

TBR was calculated using equations,

$$TBR = \frac{D}{TTR(t_0)} \times (-K)$$

$$TTR = \left(\frac{P_{M+3}}{P_M} \right)_{\text{sample}} - \left(\frac{P_{M+3}}{P_M} \right)_{\text{nature}} \times \left(\frac{1}{1+A} \right),$$

where D is the dose in mg given, $TTR(t_0)$ is the TTR at time 0 as interpolated from TTR in d 1–5 urine samples, K is the rate constant, P_{M+3} and P_M are areas of peaks corresponding to ions of tracer and tracee, respectively, and A is natural abundance of the isotope used ($^2\text{H} = 0.00015$).

Body composition. Lean body mass (LBM) and body fat mass (BFM) was estimated by isotopic water dilution (19,20). For this, sterile filtered (0.2 μm /25 mm Anotop, Whatman), salinated (90 g/L NaCl), deuterated-water (99.9%, Sigma-Aldrich) was subcutaneously injected (0.4 g/kg), and after 4.5 h, cephalic venous blood (2 mL) was collected by venipuncture. Deuterium enrichment in serum water was measured as previously described (20).

Statistical analysis. Effect of body-size (large and small) and means of food intake control (that supporting ideal body condition [Expt. 1] and that supporting similar SAA intakes between large and small dog pairs [Expt. 2]) on food and SAA intakes, TBR, BW, LBM, BFM, and circulating amino acid and thiol concentrations were evaluated using mixed-model ANOVA (PROC MIXED, version 9.1, SAS Institute). Body-size and means of food intake control were assigned as fixed and random effects, respectively, and Tukey multiple comparisons adjustment were used in post-hoc analyses. Significance of correlations between food intake and taurine entry on taurine concentrations in blood, plasma, and urine were determined by regression analysis. Percentage data were transformed [$2 \times \arcsin \times (\text{observation})^{-1/2}$] prior to analyses. Differences were considered significant at $P \leq 0.05$ or a noteworthy trend at $P > 0.05$ and < 0.10 . Results are expressed as means \pm SEM unless otherwise stated.

Results

Clearly, BW and food intake in large dogs were greater ($P < 0.01$) than those in small dogs in Expt. 1 and Expt. 2 (Table 2). However, mean SAA intake per MBW of large dogs was 23.2%

TABLE 2 Body composition and food intake of dogs given enough diet to maintain ideal body condition (Expt. 1) or similar amounts per kg MBW between small and large dog pairs (Expt. 2)¹

	Expt. 1		Expt. 2	
	Small dogs	Large dogs	Small dogs	Large dogs
Body weight, kg	12.7 ± 0.3 ^a	36.3 ± 1.9 ^b	13.1 ± 0.4 ^a	38.3 ± 2.7 ^b
Lean body mass, kg	9.8 ± 0.5 ^a	28.5 ± 1.3 ^b	8.9 ± 0.4 ^a	27.9 ± 2.1 ^b
Body fat mass, kg	2.9 ± 0.3 ^a	8.1 ± 0.7 ^b	4.2 ± 0.3 ^a	10.5 ± 0.5 ^c
Body fat mass, %	23.2 ± 2.9 ^a	21.9 ± 1.6 ^a	32.0 ± 2.5 ^b	27.5 ± 1.2 ^{ab}
Food intake, g/d	208 ± 12 ^a	347 ± 24 ^b	257 ± 16 ^a	570 ± 21 ^c
SAA intake, g · kg ^{-0.75} · d ⁻¹	0.19 ± 0.01 ^{ab}	0.14 ± 0.01 ^a	0.22 ± 0.01 ^b	0.22 ± 0.01 ^b

¹ Observations are before d4-tau administration and represent mean ± SEM, *n* = 6. Values in rows with superscripts without a common letter differ, *P* ≤ 0.05.

less (*P* < 0.05) than that of small dogs in Expt. 1 and exactly the same for large and small dogs in Expt. 2 because SAA intake was intentionally controlled for each pair of dogs in Expt. 2 to provide the same amount of the precursor for taurine synthesis (Table 2).

In Expt. 1, plasma (*P* < 0.06) and urine (*P* < 0.07) taurine concentrations tended to be lower in large than in small dogs (Table 3). Blood taurine and plasma glutathione, cyst(e)ine, cysteinyl-glycine and homocysteine in Expt. 1 did not differ between large and small dogs (*P* > 0.05). Plasma and blood taurine concentrations in Expt. 2 were 110 and 54% greater in small dogs, respectively, than in large dogs (*P* < 0.05). In contrast, concentrations of urine taurine and plasma glutathione and cyst(e)ine did not differ (*P* > 0.05) between small and large dogs in Expt. 2.

Due to limited sample volume, only 5 plasma samples could be submitted for complete amino acid profile analysis in large dogs for Expt. 1. In Expt. 1 but not Expt. 2, plasma concentrations of glycine (*P* < 0.01) and serine (*P* < 0.02) were greater in large than in small dogs (Supplemental Table 1). In Expt. 2 but not Expt. 1, plasma concentrations of tryptophan were less (*P* < 0.05) in large than small dogs. Hydroxyproline was less (*P* < 0.04) in small than in large dogs in Expt. 2. All other plasma amino acid concentrations were not significantly (*P* > 0.05) different between small and large dogs in either experiment.

TABLE 3 Taurine and thiol concentrations in dogs given enough diet to maintain ideal body condition (Expt. 1) or similar amounts per kg MBW between small and large dog pairs (Expt. 2)¹

	Expt. 1		Expt. 2	
	Small dogs	Large dogs	Small dogs	Large dogs
Taurine concentration				
Plasma, μmol/L	69 ± 10 ^{ab}	40 ± 6 ^a	86 ± 11 ^b	41 ± 15 ^b
Blood, μmol/L	198 ± 18	157 ± 20	232 ± 30	151 ± 21
Urine, μmol · L ⁻¹ · mg creatinine ⁻¹	13 ± 4.5	1.8 ± 0.9	10.0 ± 3.4	3.6 ± 3.3
Thiol concentration in plasma, μmol/L				
Glutathione	13 ± 1.3 ^a	15 ± 1.2 ^{ab}	16 ± 1.1 ^b	17 ± 1.0 ^b
Cyst(e)ine	182 ± 23	200 ± 10	164 ± 10	170 ± 12
Cysteinyl-glycine	14 ± 1.0	20 ± 3.0	nd ²	nd
Homocysteine	10 ± 0.9	10 ± 1.9	nd	nd

¹ Observations are before d4-tau administration and represent mean ± SEM, *n* = 6. Values in rows with superscripts without a common letter differ, *P* ≤ 0.05.

² nd, not determined.

The TBR were normalized to BW, MBW, relative liver weight (RLW, kg^{0.87}), LBM, and metabolic LBM (MLBM, LMB kg^{0.75}) (Table 4). TBR was normalized to RLW for comparison between large and small dogs because the liver is the major organ of taurine biosynthesis in dogs (21). For Expt. 1, all normalized TBR were lower (*P* < 0.05) in large compared with small dogs, where the per MBW, LBM, MLBM, and RLW TBR were lower by 49, 37, 48, and 43%, respectively. In Expt. 2, TBR/LBM was lower (*P* < 0.03) and TBR/BW tended to be lower (*P* = 0.06) in large than in small dogs.

Taurine entry rate in dogs (taurine synthesized + ingested food each day) was determined for Expt. 1 and 2. The entry rates were then normalized by BW, MBW, RLW, LBM, and MLBM and regressed against the indicators of taurine status. Each of the normalized entry rates and taurine concentrations in blood and plasma were positively correlated (*P* < 0.05) in both experiments (Supplemental Table 2 and Supplemental Fig. 1). Relative to the blood and plasma correlations, correlations between urine taurine concentration and taurine entry rates were higher in Expt. 2 and lower or not significant (0.05 < *P* < 0.1) in Expt. 1.

With decreasing percentage of food intake relative to that expected based on BW (10,22), concentrations of taurine in Expt. 1 decreased (*P* < 0.05) in plasma, blood, and urine (Supplemental Table 2 and Supplemental Fig. 2). The same relations between food intake and indicators of taurine status were not significant (*P* > 0.33) in Expt. 2, where the range in food intake was substantially less (32–44 g/MBW) than that in Expt. 1 (17–35 g/MBW).

Discussion

The major difference between the 2 experiments of this study was the way in which food intake (i.e., SAA intake) was controlled. In Expt. 1, all dogs were given enough diet to maintain an ideal BCS for 3 mo including the period when TBR was determined. This feeding condition resulted in similar body fat percentages among the small and large dogs (Table 2) and body fat percentages consistent with previously reported ideals in dogs (16). Thus, in Expt. 1, TBR associated with maintenance energy intake of small and large dogs was determined. The most salient finding of Expt. 1 was that, although large dogs consumed 67% more diet than small dogs (Table 2), their TBR were similar to those of small dogs (Table 4), whereas there was a trend for lower plasma taurine concentrations (*P* = 0.06) than those of small dogs (Table 3). It is noteworthy in this context that mean plasma and blood taurine concentrations in the large, but not the small dogs, were indicative of marginal taurine status

TABLE 4 Taurine biosynthesis rate in dogs given enough diet to maintain ideal body condition (Expt. 1) or similar amounts per kg MBW between small and large dog pairs (Expt. 2)¹

Taurine biosynthesis rate	Expt. 1		Expt. 2	
	Small dogs	Large dogs	Small dogs	Large dogs
Total, mg/d	749 ± 53	816 ± 218	752 ± 97	1228 ± 315
BW, mg · kg ⁻¹ · d ⁻¹	59.1 ± 4.4 ^a	22.3 ± 6.0 ^b	58.4 ± 6.5 ^{bc}	31.6 ± 7.5 ^{bc}
MBW, mg · kg ^{-0.75} · d ⁻¹	112 ± 8 ^a	54.7 ± 15 ^b	111 ± 13 ^a	78.8 ± 19 ^{ab}
RLW, mg · kg ^{-0.87} · d ⁻¹	82.2 ± 6.1 ^a	35.5 ± 9.6 ^b	80.0 ± 8.9 ^a	50.4 ± 12 ^a
LBM, mg · kg ⁻¹ · d ⁻¹	77.0 ± 5.0 ^a	28.3 ± 7.3 ^b	83.7 ± 8.0 ^a	42.9 ± 9.9 ^b
MLBM, mg · kg ^{-0.75} · d ⁻¹	136 ± 8 ^a	65.6 ± 17.1 ^b	145 ± 15 ^a	99.0 ± 23.4 ^a

¹ Total and normalized taurine biosynthesis rates are expressed as mean ± SEM, *n* = 6. Values in rows with superscripts without a common letter differ, *P* ≤ 0.05.

(11). The plasma taurine concentration observed in 1 large dog (15 $\mu\text{mol/L}$) was similar to concentrations reported in dogs with DCM that was corrected by taurine supplementation (11,12). These results support the hypothesis that large compared with small dogs are at greater risk for development of taurine deficiency when dietary SAA concentrations are marginal.

Urine taurine concentration was determined because it reflects acute changes in taurine status as a result of renal homeostatic modulation of taurine excretion (23). Variances in urine taurine concentrations within dog groups were great compared with variances observed in blood and plasma taurine concentrations. Nonetheless, there was a trend ($P = 0.07$) for urine taurine concentrations to be lower in large compared with small dogs in Expt. 1 (Table 3). This finding is consistent with a trend of a lower taurine status in large compared with small dogs consuming the same diet.

The lower than expected TBR in large dogs appears to be at least partially a result of lower than expected SAA intake by large dogs. Although the large and small dogs were housed in the same environment during the experiments, large dogs consumed less diet (and therefore less SAA) on a MBW basis than small dogs to maintain ideal body condition (Table 2, Expt. 1). Energy intakes of small dogs [$555 \pm 29 \text{ kJ} \cdot \text{kg}^{-0.75} \cdot \text{d}^{-1}$] were very close to intakes that would be predicted from body weight using a well established allometric relation [$552 \text{ kJ} \cdot \text{kg}^{-0.75} \cdot \text{d}^{-1}$, (10)]. In contrast, energy intakes of large dogs were substantively less than those that would be predicted [$427 \pm 37 \text{ kJ} \cdot \text{kg}^{-0.75} \cdot \text{d}^{-1}$]. Variations in breed attributes other than body weight, such as conformation, hair coat, and physical activity, may account for deviations in scaling of maintenance energy intake (22,24). The observed positive correlations between taurine status indicators (blood, plasma, and urine taurine concentrations) and food intake (Supplemental Fig. 1 and Supplemental Table 2) indicates that food intake differences probably accounted for the observed size-effects on TBR and taurine status.

To the authors' knowledge, the scaling of taurine metabolism with body mass has not been reported. In Expt. 2, exactly the same amount of SAA per MBW was given to each pair of small and large dogs so that effect of metabolic body size on TBR could be evaluated when the same quantity of substrates of taurine metabolism is provided. It was presumed that taurine metabolism scales with MBW as is reported with metabolism of other nutrients (25,26). However, although food intake was controlled according to MBW in Expt. 2, correlations between taurine entry and indicators of taurine status were greatest with taurine entry rate normalization by BW and LBM (Supplemental Table 2). This may indicate taurine entry scales linearly rather than exponentially with body weight.

In Expt. 2, SAA intake relative to that in Expt. 1 was increased in both large and small dogs, but more so in large dogs (69 ± 15 vs. $24 \pm 4\%$). The TBR in large dogs tended to be lower than those in small dogs after normalization to MLBM ($P = 0.32$) and RLW ($P = 0.20$) (Table 4). These normalizations were used because most taurine synthesis occurs in the lean mass, especially liver (23), and the percentage body fat in small compared with large dogs tended to be greater, in Expt. 2 ($P = 0.31$) relative to Expt. 1 ($P = 0.99$) (Table 2). Together, findings of the experiments indicate that the observed body-size effect on TBR was primarily a result of size-related difference in SAA intake relative to expected energy needs.

Plasma thiol concentrations did not differ between large and small dogs but plasma cysteinyl-glycine tended ($P = 0.10$) to be higher in large dogs. However, a trend ($P < 0.10$) of higher cysteinyl-glycine concentration was found in large compared

with small dogs in Expt. 1. Lower intake of dietary SAA in large dogs relative to small dogs may result in lower γ -glutamyl transpeptidase (EC 2.3.2.2) and dipeptidase (EC 3.4.3.5) activities to hydrolyze plasma glutathione and cysteinyl-glycine (23). This should spare plasma glutathione and cysteinyl-glycine, maintaining homeostatic concentrations of these thiols.

Most of plasma amino acid concentrations (Supplemental Table 1) were similar to or greater than those in other reports with healthy dogs (27,28). The exceptions were proline, hydroxyproline and a few other dispensable amino acids. This indicates that the experimental diet and amount consumed were adequate for maintenance of protein and amino acid balance, with the exception of taurine (29). The low-normal plasma concentrations of free cyst(e)ine in dogs in this study are consistent with the experimental diet providing SAA sufficient for protein synthesis, but not sufficient for optimal taurine status in large dogs.

In summary when a low, but adequate, protein diet was given to dogs of varying body size to maintain ideal body condition, a trend of lower taurine concentrations in blood, plasma, and urine was found in large dogs, but not in small dogs. Some large dogs had taurine deficiency (plasma taurine $\leq 40 \mu\text{mol/L}$) such that, if continued for the long-term, would be at risk for development of taurine-deficiency DCM. Our results support the hypothesis that the rate of taurine synthesis in large dogs is lower than that in small dogs when taurine precursor SAA are not in excess. In general, large relative to small dogs appear to be at greater risk for taurine deficiency because they ingest less diet for their MBW than small dogs. We conclude that the SAA allowance should be increased enough for large-breed dogs and dogs with low maintenance energy requirement to enable them to maintain an optimal taurine status.

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