

Taurine deficiency in dogs with dilated cardiomyopathy: 12 cases (1997–2001)

Andrea J. Fascetti, VMD, PhD, DACVN, DACVIM; John R. Reed, DVM, MS, DACVIM; Quinton R. Rogers, PhD, DACVN; Robert C. Backus, DVM, PhD

Objective—To determine signalment, history, clinical signs, blood and plasma taurine concentrations, electrocardiographic and echocardiographic findings, treatment, and outcome of dogs with low blood or plasma taurine concentrations and dilated cardiomyopathy (DCM).

Design—Retrospective study.

Animals—12 client-owned dogs with low blood or plasma taurine concentrations and DCM.

Procedure—Medical records were reviewed, and clinical data were obtained.

Results—All 12 dogs were being fed a commercial dry diet containing lamb meal, rice, or both as primary ingredients. Cardiac function and plasma taurine concentration improved with treatment and taurine supplementation. Seven of the 12 dogs that were still alive at the time of the study were receiving no cardiac medications except taurine.

Conclusions and Clinical Relevance—Results suggest that consumption of certain commercial diets may be associated with low blood or plasma taurine concentrations and DCM in dogs. Taurine supplementation may result in prolonged survival times in these dogs, which is not typical for dogs with DCM. Samples should be submitted for measurement of blood and plasma taurine concentrations in dogs with DCM, and taurine supplementation is recommended while results of these analyses are pending. (*J Am Vet Med Assoc* 2003;223:1137–1141)

Large-breed dogs, especially males, are predisposed to developing dilated cardiomyopathy (DCM).¹ Because the long-term prognosis for dogs with this disease is poor, methods for preventing the disease would be beneficial. However, in most affected dogs, the underlying cause is unknown.

In 1987, Pion et al² reported an association between low plasma taurine concentrations and DCM in cats. Oral supplementation of affected cats with taurine sig-

From the Departments of Molecular Biosciences (Fascetti, Rogers, Backus) and Surgery and Radiological Sciences (Reed), School of Veterinary Medicine, University of California, Davis, CA 95616-8741; and the Highlands Veterinary Hospital, 3451 Elkhorn Blvd, North Highlands, CA 95660 (Reed).

Supported in part by a grant from the Center of Companion Animal Health, School of Veterinary Medicine, University of California, Davis. Presented in part at the 18th Annual Veterinary Medical Forum of the American College of Veterinary Internal Medicine, Seattle, May 2000.

The authors thank Drs. Sean Delaney, Melanie Morgan, and Lorie Siemens for their assistance.

Address correspondence to Dr. Fascetti.

nificantly improved clinical signs, restored myocardial function, and improved survival times.³ Since then, the addition of taurine to commercial diets for cats has resulted in a marked decrease in the number of cats developing this disease.

Traditionally, dogs have not been recognized as having a dietary need for taurine, because they are able to synthesize taurine from the dietary sulfur amino acids methionine and cysteine.⁴ Recently, however, a cardiologist in private practice (JRR) brought to the attention of the authors 4 unrelated, large-breed dogs with DCM. At the time of initial examination, all 4 dogs were found to have low blood taurine concentrations. One common factor among the dogs was consumption of the same lamb meal and rice commercial dry diet. Later, a Border Collie with DCM and low blood taurine concentrations was brought to our attention by a second local cardiologist in private practice. This dog was also consuming a lamb meal and rice diet, but one produced by another manufacturer. The common diet history for these 5 dogs suggested that diet may have had a role in the development of low blood taurine concentrations and DCM in these dogs. The purpose of the study reported here was to determine the signalment, history (including diet history), clinical signs, blood and plasma taurine concentrations, electrocardiographic and echocardiographic findings, treatment, and outcome of dogs with low blood or plasma taurine concentrations and DCM. In addition, we wanted to determine whether diet may have had any role in the development of DCM.

Criteria for Selection of Cases

The cardiology database at the Veterinary Medical Teaching Hospital of the University of California, Davis, was searched for dogs examined between October 1997 and August 2001 in which a diagnosis of DCM had been made. Dogs were included in the study only if DCM had been diagnosed by a veterinary cardiologist; the diagnosis had been confirmed by means of echocardiography; samples had been submitted to the Amino Acid Laboratory at the University of California, Davis, and blood or plasma taurine concentration had been found to be low; and a complete diet history was available. In addition, the 5 dogs brought to the authors' attention by local cardiologists were included in the study.

Procedures

Information collected for all dogs included signalment, history, diet history, initial clinical signs, electrocardiographic and echocardiographic findings, blood and

plasma taurine concentrations, and treatment. Follow-up information was obtained by reviewing medical records or through telephone conversations with the attending cardiologist or primary care veterinarian.

Measurement of blood and plasma taurine concentrations—In all dogs, blood and plasma taurine concentrations were determined by the Amino Acid Laboratory at the University of California, Davis. At least 1 mL of heparinized blood, plasma, or both was submitted for analysis. Blood and plasma taurine concentrations were determined as described⁵ with an automated amino acid analyzer.^a Plasma taurine concentrations < 40 nmol/mL and blood taurine concentrations < 150 nmol/mL were considered indicative of a taurine deficiency.

Statistical analyses—Descriptive statistics (mean, SD, median, and range) were calculated for taurine concentrations and echocardiographic findings. Paired *t* tests were used to compare taurine concentrations and echocardiographic findings obtained at the time of diagnosis of DCM with values obtained after treatment. All analyses were performed with commercial software^b; values of *P* ≤ 0.05 were considered significant.

Results

From October 1997 through August 2001, 64 dogs with DCM were evaluated at the University of California veterinary teaching hospital. Blood samples from 24 of these dogs were submitted for analysis of taurine concentration, and 14 of the dogs were classified as having a taurine deficiency on the basis of a low blood or plasma taurine concentration. Six of the 14 dogs were excluded from the study, because they were American Cocker Spaniels, a breed well documented to have taurine- and carnitine-responsive DCM.⁶ A seventh dog was excluded because of an inadequate diet history. Thus, 12 dogs were included in the study, including the 5 dogs brought to the authors' attention by local cardiologists and the 7 dogs in which DCM was diagnosed at the veterinary teaching hospital during the study period.

The 12 dogs included in the study consisted of 8 males (4 sexually intact) and 4 females (2 sexually intact). There were 3 English Setters, an Alaskan Malamute, a Border Collie, a German Shepherd Dog, a Golden Retriever, a Gordon Setter, a Great Pyrenees, a Labrador Retriever, a Newfoundland, and a Scottish Terrier. Dogs ranged from 4.5 to 11 years old at the time DCM was diagnosed (mean and median, 8.3 years).

Lethargy and anorexia were the 2 most common clinical abnormalities (7 dogs each). Other clinical signs included cough (*n* = 5), dyspnea (5), weight loss (3), trembling (1), and collapse (1). Three of the dogs were clinically normal at the time of initial examination. In 2 of these dogs, an arrhythmia was detected during a routine examination. The third dog was evaluated at the request of the owner, because 2 of her other dogs had recently been found to have low taurine concentrations and DCM.

Ten of the 12 dogs had ECG abnormalities. **Left ventricular enlargement (LVE)** was the most common abnormality (*n* = 7), followed by left atrial enlargement

(6), atrial fibrillation (3), left bundle branch block (2), and ventricular premature contractions (1). All of the dogs underwent echocardiography. Mean ± SD E-point to septal separation at the time of initial examination was 14.5 ± 5.6 mm (*n* = 10; median, 14.5 mm; range, 7.5 to 26 mm). Mean fractional shortening at the time of initial examination was 15.7 ± 7.6% (*n* = 12; median, 15.9%; range, 7.0 to 28.9%). Mean plasma taurine concentration at the time DCM was diagnosed was 16 ± 20 nmol/mL (*n* = 12; median, 7 nmol/mL; range, 2 to 64 nmol/mL). Blood taurine concentrations had also been measured in 8 dogs. Mean blood taurine concentration at the time DCM was diagnosed was 121 ± 76 nmol/mL (median, 135 nmol/mL; range, 8 to 229 nmol/mL).

All 12 dogs were consuming a commercial dry diet with lamb meal, rice, or both as primary ingredients. Eight of the 12 dogs were consuming the same commercial lamb meal and rice dry diet^c (diet A). The remaining 4 dogs were each consuming 1 of 4 other commercial diets^{d–g} (diets B through E). Nutrient composition of the 5 diets was determined from the manufacturers' reported data (Table 1).

All 12 dogs were treated with taurine (1,000 to 3,000 mg, PO, q 24 h) beginning at the time DCM was diagnosed or when the low blood or plasma taurine concentration was discovered. In addition, 8 dogs were treated with inotropic agents, 7 were treated with diuretics, 7 were treated with angiotensin-converting enzyme inhibitors, and 1 was treated with a calcium channel blocker to manage cardiac abnormalities. Eleven of the dogs were changed to a different commercial dry diet at the time DCM was diagnosed or when the low blood or plasma taurine concentration was discovered.

Dogs were reevaluated between 1 and 12 months after the time of initial diagnosis (mean ± SD, 4.3 ± 3.5 months; median, 3 months). Electrocardiography was repeated on 4 dogs. Results were normal for 1 of the 4 dogs; abnormalities identified in the other 3 included left ventricular enlargement (*n* = 1), atrial fibrillation (2), and ventricular premature contractions (1). Echocardiography was repeated on 9 dogs. Mean E-point to septal separation was 8.9 ± 4.1 mm (*n* = 8; median, 8.3 mm; range, 2.9 to 17 mm). This was significantly (*P* = 0.002) lower than the mean value at the time of initial examination of these 8 dogs. Mean fractional shortening was 22.5 ± 6.3% (*n* = 9; median, 20%;

Table 1—Proximate dry matter composition of diets fed to dogs with dilated cardiomyopathy and low blood or plasma taurine concentrations

Variable	Diet				
	A	B	C	D	E
Crude protein (%)	24	23	17	22	28
Crude fat (%)	14	13	7	11	13
NFE* (%)	48	57	63	62	46
Crude fiber (%)	2	1	7	5	4
Ash (%)	10	6	6	NR	9
ME (kcal/g)	3.8	3.9	3.4	3.5	3.8

All values were obtained from manufacturers' reported data.

*Calculated or reported nitrogen-free extract.

NR = Not reported. ME = Metabolizable energy.

range, 16.6 to 36%). This was significantly ($P = 0.002$) greater than the mean value at the time of initial examination of these 9 dogs. Blood and plasma taurine concentrations increased in the 3 dogs in which analyses were repeated. Mean plasma taurine concentration was 226 ± 54 nmol/mL ($n = 3$; median, 208 nmol/mL; range, 183 to 286 nmol/mL), and mean blood taurine concentration was 455 ± 88 nmol/mL ($n = 3$; median, 443 nmol/mL; range, 373 to 548 nmol/mL). Plasma taurine concentrations were significantly ($P = 0.02$) increased in these 3 dogs, compared with concentrations prior to institution of taurine supplementation. Blood taurine concentrations were not significantly increased, but the P value was close to the cutoff for significance ($P = 0.06$).

Seven of the 12 dogs were alive at the time of the study. Mean \pm SD survival time for all 12 dogs was 585 ± 499 days (median, 456 days; range, 1 to 1,460 days). One dog died within 1 day, 3 dogs died within 180 days, and 1 dog died within 365 days of initial examination. Survival times for the 7 dogs still alive at the time of the study ranged from 365 to 1,460 days (913 ± 380 days; median, 1,095 days). None of the 7 surviving dogs were receiving any cardiac medications at the time of the study other than taurine.

Discussion

With the exception of the single Newfoundland, none of the dogs in this study were of breeds predisposed to developing DCM. Breeds recognized to have a high prevalence of DCM include the Scottish Deerhound, Doberman Pinscher, Irish Wolfhound, Great Dane, Boxer, Saint Bernard, Afghan Hound, Newfoundland, and Old English Sheepdog.⁷ The dogs in the present study also generally had longer survival times than are typically reported in the literature for dogs with DCM. For instance, 2 recent multibreed retrospective studies^{8,9} reported overall probabilities of survival 1 year after diagnosis of DCM of 17.5 and 37.5% and median survival times of 27 and 65 days. In contrast, the median survival time for the 12 dogs in the present study was 456 days, and many of these dogs were still alive at the time of the study. Finally, several of the dogs in the present study regained substantial cardiac function and were weaned off all medications except taurine. This is unusual for most dogs with DCM, in which the disease is typically progressive and fatal. Information on follow-up cardiac evaluations and measurements of taurine concentrations was not available for all dogs in the present study. However, in those dogs in which this information was available, cardiac function improved and taurine concentrations increased to concentrations greater than those considered evidence of a deficiency. Furthermore, in all of the dogs that lived > 1 year, all cardiac medications other than taurine were discontinued. These characteristics suggest that the dogs in the present study did not have the typical form of DCM.

Consumption of commercial dry diets containing lamb meal or rice as a primary ingredient was a common finding among dogs in the present study. Three of the 5 diets that these dogs consumed contained both lamb meal and rice (diets A, C, and E); 1 contained

chicken meal and rice (diet B); and 1 contained ground whole wheat, lamb meal, and rice (diet D). Most of the dogs were changed to another diet at the time DCM was diagnosed. However, the owner of the Border Collie decided to keep the dog on the same commercial lamb meal and rice diet (diet D) but supplemented the dog with taurine. Increases in taurine concentration and cardiac function in this dog indicated a response to taurine supplementation and supported the hypothesis that diet played a contributing role in the development of DCM by causing taurine depletion.

Taurine is the most abundant of the free amino acids in tissues. High concentrations of taurine are found in animal tissues, especially muscle, viscera, and brain. In cats, 3 manifestations of taurine deficiency have been identified: central retinal degeneration, reproductive failure and impaired fetal development, and DCM.¹⁰ Taurine deficiency can also cause hearing loss, platelet hyperaggregation, and impaired immune function, although specific clinical disorders have not been recognized.¹⁰ The mechanism of heart failure in taurine-deficient cats and dogs is poorly understood, but in the myocardium, taurine appears to participate in many functions, including cellular osmoregulation, free-radical scavenging, and modulation of contraction strength through regulation of calcium concentration.³

In cats, inadequate provision of dietary taurine results in DCM.³ Cats have a limited ability to synthesize taurine because of low concentrations of the enzymes cysteine sulfenic acid decarboxylase and cysteine dioxygenase.³ In contrast, dogs have not been generally recognized to have a need for dietary taurine, because they have the metabolic capacity to synthesize taurine from cysteine and methionine.⁴ The concentration of taurine necessary to prevent clinical signs of a taurine deficiency in cats varies with diet composition and processing, but ranges from 400 mg of taurine/kg of diet to $> 2,000$ mg of taurine/kg of diet.^{11,12}

Possible causes of the taurine deficiency in the dogs in the present report include insufficient synthesis of taurine, extraordinary loss of taurine or its precursors in urine, extraordinary gastrointestinal tract loss of taurine in bile acid conjugates as found in cats, and a reduction in protein digestibility.¹³ On the basis of our clinical findings, in conjunction with the blood and plasma taurine concentrations and common diet histories in these dogs, we hypothesize that the consumption of diets with inadequate or unavailable sulfur amino acid precursors of taurine resulted in taurine deficiency and low blood taurine concentrations that, in turn, led to the development of abnormal cardiac function and DCM.

Recent experimental and clinical observations in dogs are supportive of the possibility that insufficient synthesis of taurine from sulfur amino acid precursors results in the development of DCM. Sanderson et al¹⁴ found low plasma taurine concentrations in Beagles fed an energy-dense, protein-restricted (10% protein on a dry-matter basis) diet for 48 months. One dog developed DCM, and taurine supplementation (500 mg, PO, q 12 h) reversed the cardiac changes in this dog. Prolonged feeding of a commercial prescription diet with a protein-to-calorie ratio similar to one used by

Sanderson et al¹⁴ may have induced development of DCM in Dalmatians,¹⁵ but whether taurine deficiency caused DCM in these dogs is unclear. Blood and plasma taurine concentrations in the Dalmatians were within reference limits at the time of clinical evaluation. However, taurine concentrations in blood and tissues at the time DCM developed were not known. It is possible that a period of taurine deficiency could produce myocardial damage in dogs that cannot be reversed. Following a change in diet, blood taurine concentrations may indicate normal body taurine stores and not reveal that a period of deficiency had occurred.

Alternatively, the rice bran and whole rice products in the commercial diets consumed by most of the dogs in the present study may have been a factor in the development of low blood and plasma taurine concentrations. Rice bran and whole-rice products are sources of moderately soluble fiber and contain relatively high amounts of fat. The fiber, fat, or protein content of the rice bran may accelerate excretion of bile acids, predisposing dogs to taurine deficiency. Stratton-Phelps et al¹⁶ recently demonstrated that cats fed a purified diet with 26% (dry-matter basis) full-fat rice bran had critically low plasma and blood taurine concentrations after 6 and 12 weeks, respectively. Extraordinary intestinal loss of taurine secondary to increases in bacterial populations appears to be contributing to taurine deficiency in these cats.¹⁶ Preliminary results from a dose-response study conducted by the authors of the present study indicate that cats can develop critically low blood taurine concentrations when consuming full-fat rice bran at concentrations as low as 4% (dry-matter basis).

Dogs in the present study were all fed a commercial dry diet containing high quantities of lamb meal or rice products, but why this should be associated with taurine deficiency was not readily apparent, as the diets appeared sufficient in protein and sulfur amino acid contents and had passed testing in accordance with the Association of American Feed Control Officials' feeding trials for all life stages. However, because lamb meals may be of low quality, limited bioavailability of sulfur amino acids required for taurine synthesis in the diet was suspected. Relative to other meat meal sources, lamb meal has been shown to have poor ileal nitrogen and cystine digestibility in dogs.¹⁷ Results of a study involving feeding a lamb meal and rice diet to young Beagles for 8 months indicate that plasma taurine concentrations decreased substantially during the first month after dogs were switched to the diet but did not change thereafter.¹⁸ Thus, the lamb meal and rice diet appeared to have an effect on taurine status, but not to the point of a depletion sufficient to cause DCM.

Recently, low blood taurine concentrations have been identified in Newfoundlanders.¹⁹ Reduced reproductive performance, small litter sizes, poor litter growth, and small puppies were reported, and similar findings have been reported for cats with taurine deficiency.²⁰ Diet appeared to be the cause of the taurine deficiency in these dogs, in that 7 of the 12 dogs with plasma taurine concentrations < 40 nmol/mL were

consuming diets containing lamb meal and rice. Methionine supplementation in dogs consuming lamb meal and rice diets resulted in substantial improvement in taurine concentrations, and plasma taurine concentrations increased when a dietary change was instituted but were unchanged when the diet was not changed.

The authors have also examined 2 dogs with taurine deficiency that were being fed a home-prepared, low-protein, tofu-based diet that meet the National Research Council's requirements for adult maintenance.²¹ Taurine deficiency in these dogs was attributed to inadequate synthesis, and it was assumed that the low concentrations of protein in general and of sulfur amino acids in particular provided an inadequate supply of precursors for taurine synthesis. An additional contributing factor may have been an increase in taurine loss, as soybean protein augments bile acid loss through microbial degradation and accelerates cholecystokinin-mediated bile acid turnover.^{5,22} Similarly, we are aware of 3 Golden Retrievers with taurine deficiency that lived in the same household and were consuming a vegetarian diet formulated by the owner.

Taken together, these findings suggest that taurine deficiency may result in DCM in dogs other than American Cocker Spaniels fed a diet that contains insufficient amino acid precursors for adequate taurine synthesis or that accelerates taurine loss. We recommend that a complete diet history be obtained for every dog each time it is examined by a veterinarian, including the name and amount of food fed, the name and amount of any snacks and treats, a description of the manner in which the dog is fed, whether the dog has access to other food sources, and whether any dietary supplements are given. We also recommend that all home-cooked diets be evaluated by a veterinary nutritionist.

Blood and plasma taurine concentrations should be measured in all dogs with DCM, just as measurement of blood and plasma taurine concentrations is recommended for all cats with DCM.²³ Although blood taurine concentration is only a fraction of the concentration in the tissues, blood and plasma taurine concentrations do change in proportion with tissue concentrations.^{4,24} Blood taurine concentrations may be used to substantiate a diagnosis of taurine deficiency when plasma concentrations are equivocal. In addition, blood taurine concentrations are only slightly altered after eating, whereas plasma taurine concentration may change substantially in taurine-depleted animals.²³ A substantial increase in plasma or serum taurine concentration can occur secondary to taurine leakage from granulocytes and platelets, as occurs with clotting or hemolysis, but analysis of blood taurine concentration is not confounded by these effects. Serum taurine concentrations are of questionable clinical value because of the variations in time of clotting and the method of serum separation, and in our experience, the variability in serum taurine concentrations is greater than the variability in plasma taurine concentrations.

Finally, we recommend that taurine be administered to all dogs with DCM, pending results of taurine

analysis. Taurine is inexpensive and readily available and has no reported adverse effects when administered orally. Follow-up measurement of blood and plasma taurine concentrations should be performed after 1 to 2 months of taurine supplementation to confirm that taurine status has improved and verify owner compliance with regard to administration.

One manufacturer produced 3 of the diets (diets A, B, and C) associated with taurine deficiency in the present study. Since identification of these dogs, the manufacturer has added taurine to 1 of the 3 diets (diet A). Diets D and E, each produced by other manufacturers at the time the dogs consuming them developed low taurine concentrations and DCM, have recently been acquired by other companies. The formulation of diet E has been changed, but neither diet D nor diet E includes additional taurine. Nevertheless, we suggest that veterinarians not focus on particular diets but on the issue of taurine deficiency as a whole. Further research is needed to identify dietary factors inducing taurine deficiency and determine the mechanisms of their effects.

^aSystem 7300 and Model 121-M automated amino acid analyzers, Beckman Instruments Inc, Palo Alto, Calif.

^bSYSTAT, version 9.0, SPSS Inc, Chicago, Ill.

^cNutro Natural Choice lamb meal and rice formula dog food, Nutro Products Inc, City of Industry, Calif.

^dNutro Natural Choice senior dog food, Nutro Products Inc, City of Industry, Calif.

^eNutro Natural Choice lite rice and lamb meal formula for overweight dogs, Nutro Products Inc, City of Industry, Calif.

^fNature's Recipe adult maintenance lamb meal and rice, Nature's Recipe Pet Foods, Heinz Pet Products, Pittsburgh, Pa.

^gSensible Choice adult lamb meal and rice formula, Pet Products Plus Inc, St Charles, Mo.

^hStratton-Phelps M, Rogers QR, Backus RC, et al. Oral antibiotics do not replete taurine in cats consuming 26% dietary rice bran (abstr), in *Proceedings*. Fed Am Soc Exp Biol 2002;5055.

References

- Buchanan JW. Causes and prevalence of cardiovascular disease. In: Kirk RW, Bonagura JD, eds. *Current veterinary therapy XI*. Philadelphia: WB Saunders Co, 1992;647–655.
- Pion PD, Kittleson MD, Rogers QR, et al. Myocardial failure in cats is associated with low plasma taurine: a reversible cardiomyopathy. *Science* 1987;237:764–768.
- Pion PD, Sanderson SL, Kittleson MD. The effectiveness of taurine and levocarnitine in dogs with heart disease. *Vet Clin North Am Small Anim Pract* 1998;28:1495–1514.
- Hayes KC. Taurine nutrition. *Nutr Res Rev* 1988;1:99–113.
- Kim SW, Morris JG, Rogers QR. Dietary soybean protein decreases plasma taurine in cats. *J Nutr* 1995;125:2831–2837.
- Kittleson MD, Keene B, Pion PD, et al. Results of the Multicenter Spaniel Trial (MUST): taurine- and carnitine-responsive dilated cardiomyopathy in American Cocker Spaniels with decreased plasma taurine concentration. *J Vet Intern Med* 1997;11:204–211.
- Sisson DD, Thomas WP, Keene BW. Primary myocardial disease in the dog. In: Ettinger SJ, Feldman EC, eds. *Textbook of veterinary internal medicine*. 5th ed. Philadelphia: WB Saunders Co, 2000; 874–895.
- Tidholm A, Svensson H, Sylven C. Survival and prognostic factors in 189 dogs with dilated cardiomyopathy. *J Am Anim Hosp Assoc* 1997;33:364–368.
- Monnet E, Orton EC, Salam M, et al. Idiopathic dilated cardiomyopathy in dogs: survival and prognostic indicators. *J Vet Intern Med* 1995;9:12–17.
- Kirk CA, Debraekeleer J, Armstrong PJ. Normal cats. In: Hand MS, Thatcher CD, Remillard RL, et al, eds. *Small animal clinical nutrition*. 4th ed. Marline, Mo: Walsworth Publishing Co, 2000; 291–347.
- National Research Council. *Nutrient requirements of cats*. Washington, DC: National Academy Press, 1986;13–15.
- 2002 Official publication. Oxford, England. In: Association of American Feed Control Officials Inc, 2002;140–141.
- Morris JG, Rogers QR, Kim SW, et al. Dietary taurine requirement of cats is determined by microbial degradation of taurine in the gut. In: Huxtable RJ, Michalk D, eds. *Taurine in health and disease*. New York: Plenum Press, 1994;59–70.
- Sanderson SL, Gross KL, Ogburn PN, et al. Effects of dietary fat and L-carnitine on plasma and whole blood taurine concentrations and cardiac function in healthy dogs fed protein-restricted diets. *Am J Vet Res* 2001;62:1616–1623.
- Freeman LM, Michel KE, Brown DJ, et al. Idiopathic dilated cardiomyopathy in Dalmatians: nine cases (1990–1995). *J Am Vet Med Assoc* 1996;209:1592–1596.
- Stratton-Phelps M, Backus RC, Rogers QR, et al. Dietary rice bran decreases plasma and whole-blood taurine in cats. *J Nutr* 2002;132(6 suppl 2):1745S–1747S.
- Johnson ML, Parsons CM, Fahey GC, et al. Effects of species raw material source, ash content and processing temperature on amino acid digestibility of animal by-product meals by cecectomized and ileally cannulated dogs. *J Anim Sci* 1998;76:1112–1122.
- Torres CL, Backus RC, Fascetti AJ, et al. Taurine status in normal dogs fed a commercial diet associated with taurine deficiency and dilated cardiomyopathy. *J Anim Physiol Anim Nutr* 2003;in press.
- Backus RC, Cohen GL, Pion PD, et al. Taurine deficiency in Newfoundlands fed commercially available complete and balanced diets. *J Am Vet Med Assoc* 2003;223:1130–1136.
- Sturman JA, Gargano AD, Messing JM, et al. Feline maternal taurine deficiency: effect on mother and offspring. *J Nutr* 1985; 116:655–667.
- National Research Council. *Nutrient requirements of dogs*. Washington, DC: National Academy Press, 1985;11–12.
- Backus RC, Rogers QR, Rosenquist GL, et al. Diets causing taurine depletion in cats substantially elevate postprandial plasma cholecystokinin (CCK) concentration. *J Nutr* 1995;125:2650–2657.
- Pion PD, Lewis J, Greene K, et al. Effect of meal-feeding and food deprivation on plasma and whole blood taurine concentrations in cats. *J Nutr* 1991;121(suppl 11):S177–S178.
- Pacioretty L, Hickman MA, Morris JG, et al. Kinetics of taurine depletion and repletion in plasma, serum, whole blood and skeletal muscle in cats. *Amino Acids* 2001;21:417–427.