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Plasma and whole blood taurine in normal dogs of varying size fed commercially prepared food

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Summary

The objective of the present study was to examine the effect of signalment, body size and diet on plasma taurine and whole blood taurine concentrations. A total of 131 normal dogs consuming commercially prepared dog food had blood drawn 3–5 h post-prandially to be analysed for plasma amino acids and whole blood taurine. Body weight and morphometric measurements of each dog were taken. Plasma and whole blood taurine concentrations were 77 ± 2.1 nmol/ml (mean ± SEM) and 266 ± 5.1 nmol/ml (mean ± SEM), respectively. No effect of age, sex, body weight, body size, or diet was seen on plasma and whole blood taurine concentrations. Mean whole blood taurine concentrations were lower in dogs fed diets containing whole grain rice, rice bran or barley. The lowest whole blood concentrations were seen in dogs fed lamb or lamb meal and rice diets. Plasma methionine and cysteine concentrations were lower in dogs fed diets with animal meals or turkey, and whole grain rice, rice bran or barley. Fifteen of 131 dogs had plasma taurine concentrations lower than, or equal, to the previously reported lowest mean food-deprived plasma taurine concentration in normal dogs of 49 ± 5 nmol/ml (mean ± SEM) (Elliott et al., 2000). These findings support the theory that taurine deficiency in dogs may be related to the consumption of certain dietary ingredients. Scientific and clinical evidence supports the hypothesis that dilated cardiomyopathy is associated with low blood taurine concentration in dogs; therefore, further work is indicated to determine the mechanism by which diet can affect taurine status in dogs.

Introduction

Taurine deficiency, as a cause of disease, was first reported by Hayes et al. in cats (*Felis catus* L.) that developed retinal degeneration when fed a casein-based diet (Hayes et al., 1975). In 1987, Pion et al. reported that dietary taurine deficiency in cats also induced dilated cardiomyopathy (DCM) that was reversible with taurine supplementation. Reproductive failure and impaired foetal development have also been well established in cats with diet-induced taurine deficiency (Sturman et al., 1986).

Both dogs (*Canis familiaris* L.) and cats use only taurine to conjugate bile acids, but in dogs, a need for dietary taurine is not generally recognized. Dogs are known, like many species, to have the metabolic capacity to synthesize taurine from the dietary sulphur amino acids, cysteine and methionine (Malloy et al., 1981). It has been known for over two decades that commercial dog foods generally contain little or no added taurine (Aguirre, 1978) and that many of the ingredients in dog food have a low taurine content (Spitze et al., in press).

Recently reported experimental and clinical observations in dogs are supportive of the possibility that consumption of diets with inadequate and/or unavailable taurine, or taurine precursors, can result in taurine deficiency and low blood taurine concentrations. This can lead to the development of abnormal cardiac function and DCM (Freeman et al., 1996; Sanderson et al., 2001; R. C. Backus, unpublished data; A. J. Fascetti, unpublished data). Postulated causes for the taurine deficiency in these dogs were considered to be (i) insufficient synthesis of taurine; (ii) extraordinary loss of taurine or its precursors in urine; (iii) extraordinary gastrointestinal loss of taurine in bile acid conjugates, as found in cats; or (iv) a reduction in sulphur amino acid bioavailability (Morris et al., 1994).

One obstacle to investigating this problem is that reference intervals for blood taurine concentrations and complete amino acid profiles in dogs have not been determined. Currently, canine results are compared with values based on a modification of reference intervals for cats. It is suspected that the reference values for dogs will be significantly different from those established in cats, in part because of the effects of diet composition and metabolic body size. There are no references in the literature, to the authors' knowledge, on whole blood taurine concentrations in dogs. Only one study, conducted in two veterinary hospitals has reported reference intervals for plasma taurine concentrations in normal dogs and dogs with cardiac disease (Kramer et al., 1995). Each hospital used different anticoagulants, perhaps resulting in different taurine concentrations. Owing to the small number of dogs, differences based on metabolic body size were not determined. Furthermore, the effect of diet was not included in the analyses because taurine was not considered an essential amino acid for the dog.

Concurrent taurine, cysteine and methionine concentrations have never been reported in dogs fed commercial diets to the authors' knowledge. In addition, complete amino acid profiles have only been reported as part of experimental studies in small numbers of dogs fed the same diet in the past (Hansen et al., 1992; Elliott et al., 2000; Delaney et al., 2001). Therefore, the objectives of the current study were to determine the effect of breed, body size and diet on blood taurine concentrations, and to establish reference intervals for complete amino acid profiles and blood taurine concentrations in healthy, adult dogs eating a variety of commercial diets.

Materials and methods

Animals

A total of 131 dogs belonging to faculty, staff, students and clients of the School of Veterinary Medicine at the University of California, Davis, CA, attendees at a local pure-breed dog agility trial and staff of a private veterinary hospital for guide dogs participated in the study. To be included in the study all dogs had to be greater than 2 years of age, healthy (no current disease, with the exception of degenerative joint disease) and on the same commercially manufactured diet for at least 4 months. None of the dogs were on medications with the exception of flea (Ctenocephalides, *Felis felis* L.) and/or heartworm (*Dirofilaria immitis* L.) preventative and/or low-dose non-steroidal anti-inflammatory drugs. All owners of participating dogs signed a consent form permitting blood collection and analysis. As the purpose of this study was to determine taurine and complete amino acid concentrations in healthy animals, cardiac ultrasounds were not obtained for each dog. All dogs were fed their typical diet and amount, 3–5 h before blood collection.

Diet history and morphometric measurements

Detailed diet history forms were completed by participating dog owners. Owners were required to provide the following information on the form: age, sex, breed, diet(s), amount fed per day, number of times fed per day, length of time on current diet(s), food storage

method, treats, treat amounts, treat frequency, supplements, supplement amounts, supplement frequency, access to other animals' food, previous diet(s), medication(s), exercise frequency and questions regarding health status (presence of vomiting, diarrhoea, sneezing, coughing, appetite changes, weight changes and urination/drinking changes). Protein intake was subsequently calculated for each study-participant based on the information contained in the history form and diet nutrient analysis provided by the food manufacturer. The United States Department of Agriculture (USDA) Nutrient Database for Standard Reference, Release 14 (Nutrient Data Laboratory, Agricultural Research Service, Beltsville Human Nutrition Research Center, Beltsville, MD, USA) was used to determine the nutrient composition for all human foodstuffs fed as treats or supplements to the commercial diet(s). Morphometric measurements were obtained by determining each dog's cranial thoracic circumference, pelvic circumference, height (acromion to floor), and length (acromion to tailbase). Body condition scoring (Laflamme et al., 1994) was performed by only one investigator (SJD) to decrease variability.

Sample collection and taurine analysis

Three to 5 ml of blood was drawn from the jugular or cephalic vein into a heparinized syringe. The blood was then immediately placed into a glass tube containing lithium heparin. Half a millilitre of whole blood was then pipetted out of the glass tube and stored at −80 °C until analysis. The remaining blood was immediately centrifuged and the plasma was placed in 0.5-ml aliquots. Half a millilitre of 6% sulphosalicylic acid was added to 0.5 ml of plasma to precipitate the plasma proteins. All samples were stored at −80 °C until analysis. The median storage time until sample analysis was 18 days. Plasma amino acid and whole blood taurine concentrations were determined using Beckman 6300 and 121MB amino acid analyzers (Beckman Industries, Palo Alto, CA, USA), respectively. All amino acid results are reported as nmol/ml of plasma or whole blood.

Statistical analysis

Statistical analysis was performed using bmdp7d, version PC90 (BMDP Statistical Software Inc., Los Angeles, CA, USA). Pearson's product–moment correlation was estimated to evaluate the linear relationships of plasma and whole blood taurine with the following: plasma methionine, plasma cysteine, grams of daily crude protein intake per kilogram body weight, thoracic circumference, pelvic circumference, height, length, body condition score, body weight and age. Analysis of variance (anova) was performed to identify statistically significant differences in plasma taurine, plasma methionine, plasma cysteine and/or whole blood taurine with signalment, diet providing most of the daily calories of the dog (main diet), manufacturer of the main diet, first listed animal source ingredient in the main diet and first listed plant source ingredient in the main diet. When global anova results were significant, *post-hoc* comparisons were performed using Tukey's, Scheffe's and Bonferroni's method (the latter for pairwise *t*-tests) of adjustment for multiple comparisons. Results are reported as mean ± SEM. Probability levels, $p < 0.05$ were considered to be significant.

Results

The dogs ranged in age from 2 to 14 years (median, 5.3 years). Thirty-one dogs were intact females, 47 were spayed females, 27 were intact males and 26 were castrated males. Breeds included mixed breeds ($n = 30$), Labrador Retriever ($n = 18$), Australian Shepherd ($n = 12$), Border Collie ($n = 11$), Doberman Pinscher ($n = 7$), Golden Retriever ($n = 6$), Shetland Sheepdog ($n = 5$), Foxhound ($n = 4$), German Shepherd Dog ($n = 4$), Rottweiler ($n = 4$), Boxer ($n = 3$), Pointer ($n = 3$), Newfoundland ($n = 3$), Whippet

($n = 2$) and 1 each Siberian Husky, Belgian Sheepdog, Belgian Tervuren, Border Terrier, Briard, Chihuahua, Corgi, Dalmatian, Flat-coated Retriever, Great Pyrenees, Irish Wolfhound, Jack Russel Terrier, Mastiff, Miniature Australian Shepherd, Miniature Dachshund, Miniature Long-haired Whippet, Portugese Water Dog, Smooth-coated Collie, Tibetian Terrier and Vizsla. The body weights ranged from 5.3 to 79 kg (median 25.6 kg). Body condition scores using a nine-point scale (with a score of 5 as ideal) ranged from 3 to 7 (median 5).

The mean plasma and whole blood taurine concentrations for all the dogs in the study were 77 ± 2.1 and 266 ± 5.1 nmol/ml, respectively. The values for the first, second, third and fourth quartiles for plasma taurine concentrations were 60, 78, 90, and 145 nmol/ml, respectively. The values for the first, second, third and fourth quartiles for whole blood taurine concentrations were 224, 260, 304, and 476 nmol/ml, respectively. The mean ± SEM and the first, second, third and fourth quartiles for all the amino acids are reported in Table 1.

No statistically significant correlation of plasma and whole blood taurine to grams of daily crude protein intake per kilogram body weight, thoracic circumference, pelvic circumference, height, length, body condition score, body weight and age were identified. The correlation coefficients for cysteine ($r = 0.54$) and methionine ($r = 0.51$) on plasma taurine were statistically significant ($p < 0.001$). ANOVA did not identify statistically significant differences between plasma taurine with breed, sex, main diet, manufacturer of the main diet, first listed animal source ingredient in the main diet and first listed plant source ingredient in the main diet.

Results were evaluated to determine if there were any differences based on breed or diet. There was a significant difference between the mean whole blood taurine

Table 1. **Plasma amino acid and whole blood taurine values**

Amino acid	Mean (nmol/ml)	SEM (nmol/ml)	First quartile (nmol/ml)	Second quartile (nmol/ml)	Third quartile (nmol/ml)	Fourth quartile (nmol/ml)
Alanine	388	9.6	320	380	455	721
Arginine	102	2.6	85	100	123	190
Asparagine	40	1.1	30	40	49	70
Aspartate	7	0.2	6	7	8	16
Citrulline	41	1.9	27	37	50	144
Half-cystine	46	1.3	36	46	53	96
Glutamate	23	1.2	15	21	26	71
Glutamine	495	9.4	417	494	569	739
Glycine	268	8.4	207	251	310	791
Histidine	71	1.6	60	69	80	130
Hydroxyproline	67	4.1	44	58	78	401
Isoleucine	51	1.3	40	49	57	115
Leucine	120	3.2	95	116	134	257
Lysine	132	5	94	124	159	426
Methionine	57	1.6	45	55	65	136
Ornithine	35	1.5	23	32	43	121
Phenylalanine	45	0.9	39	44	52	78
Proline	246	8.2	174	234	304	676
Serine	107	2.6	87	105	126	191
Taurine	77	2.1	60	78	90	145
Whole blood taurine	266	5.1	224	260	304	476
Threonine	178	5.0	138	169	211	335
Tryptophan	60	1.7	45	58	68	124
Tyrosine	39	1.1	30	37	47	70
Valine	157	4.1	130	157	179	326

concentration in Australian Shepherds (222 ± 10.2 nmol/ml) and Labrador Retrievers (294.6 ± 14.1 nmol/ml, $p = 0.0003$). Significant differences were found when whole blood taurine was evaluated to see if there were effects based on food manufacturer. Dogs consuming diets from one manufacturer that supplemented with taurine had greater mean whole blood taurine concentrations (334 ± 28.3 nmol/ml) compared with dogs consuming foods produced by two other manufacturers who did not add taurine to their diets (229 ± 10.4 nmol/ml, $p \leq 0.05$) and (241 ± 6.0 nmol/ml, $p = 0.03$), respectively.

Several dietary plant protein sources had an effect on whole blood taurine concentrations. When whole grain brown rice was the first plant ingredient in the main diet there was a significantly lower mean whole blood taurine concentration (221 ± 10.9 nmol/ml) compared with dogs fed a group of other assorted plant protein sources (306 ± 20.8 nmol/ml, $p = 0.002$). Dogs consuming diets with ground corn as the first plant ingredient had a higher whole blood taurine concentration (307 ± 15.8 nmol/ml) compared with dogs fed a diet in which the first plant ingredient was whole grain brown rice (221 ± 10.9 nmol/ml, $p = 0.001$).

Multiple combinations of dietary animal and plant protein sources were evaluated to determine if there was an effect on amino acid concentrations. The mean plasma cysteine concentration in dogs fed a combination of lamb meal and rice (62 ± 7.7 nmol/ml) was higher than in dogs fed lamb meal and ground rice (34 ± 3.5 nmol/ml, $p = 0.009$). The mean cysteine concentration in dogs fed turkey and ground barley (39 ± 3.4 nmol/ml) was significantly lesser than in dogs fed lamb meal and rice (62 ± 7.7 nmol/ml, $p = 0.02$). Dogs fed chicken and brown rice had a higher mean plasma methionine concentration (84.0 ± 21.3 nmol/ml) compared with dogs fed chicken meal and rice (42 ± 2.7 nmol/ml, $p = 0.0006$), lamb meal and ground whole grain brown rice (45 ± 3.3 nmol/ml, $p = 0.0002$), and turkey and ground barley (49 ± 3.2 nmol/ml, $p = 0.0004$). Dogs consuming lamb meal and rice had a lower mean whole blood taurine concentration (246 ± 7.3 nmol/ml) compared with dogs consuming foods in the other category (294 ± 14.8 nmol/ml, $p = 0.001$).

Discussion

The mean plasma taurine concentration determined in this study (77 ± 2.1 nmol/ml) was very similar to the mean concentration reported by Kramer et al. (1995) (80 ± 2.3 nmol/ml). However, the lowest concentration reported in the normal dogs by the Kramer et al. (1995) study was 50 nmol/ml while in this study it was 21 nmol/ml. Based on their findings, Kramer et al. (1995) concluded that plasma taurine concentrations below 50 nmol/ml were low. Using that standard, 15 of the 131 dogs, or 11.5% of the normal dogs in this study had low plasma taurine concentrations. Currently, the authors' laboratory considers plasma taurine concentrations less than 40 nmol/ml as critically low. Based on this minimum concentration, five of 131 dogs, or 4% had critically low plasma taurine concentrations. Overall, the plasma amino acid concentrations found in this study are consistent with the values previously reported in smaller numbers of healthy adult dogs (Strombeck and Rogers, 1978; Hansen et al., 1992; Elliott et al., 2000).

Kramer et al. (1995) found that 17% of dogs with DCM had plasma taurine concentrations less than 25 nmol/ml. In this study only one dog had a plasma taurine concentration below 25 nmol/ml. At this time the dog has not been evaluated for cardiac changes; but based on communication with the owner and physical examination, the dog does not appear to have clinical signs of cardiac disease. Low plasma taurine concentrations do exist without the presence of DCM. In cats, the onset of clinical signs was variable once taurine concentrations declined markedly below the normal range (Pion et al., 1987). It is reasonable to predict that similar findings may exist in dogs. The authors recommend that taurine concentrations that fall below the critical range in a seemingly otherwise healthy

animal be repeated, and a dietary change or taurine supplementation be instituted pending results of further analyses.

Unfortunately, it is unknown whether the plasma taurine concentrations reported by the Kramer et al. (1995) study were obtained from food-deprived or fed dogs. In humans, there is no effect of fasting on plasma taurine concentrations (Trautwein and Hayes, 1990), while in cats food deprivation causes a small but significant reduction in plasma taurine concentrations (Pion et al., 1989, 1991). In dogs, plasma amino acid concentrations following a large meal can either somewhat increase or decrease depending on whether the amino acid of interest is limiting or non-limiting (Longenecker and Hause, 1959; Delaney et al., 2001). Therefore, as the samples collected in this study were post-prandial samples, the results could be higher than results obtained in food-deprived dogs. This raises the possibility that an even larger proportion of the dogs in this study have low plasma taurine concentrations based on the intervals set by Kramer et al. (1995).

Whole blood taurine is insensitive to the post-prandial period in both humans (Trautwein and Hayes, 1990) and cats (Pion et al., 1991), and presumably in dogs. In cats, whole blood taurine is known to more closely reflect skeletal muscle and presumably cardiac muscle taurine concentrations than plasma during dietary depletion (Pacioretty et al., 2001). Therefore, whole blood taurine concentrations in dogs are most likely a better indicator of overall taurine status.

The mean whole blood taurine concentration determined in this study in healthy dogs is 266 ± 5.1 nmol/ml. This is slightly lower than whole blood taurine concentrations in cats, reported to be between 300 and 400 nmol/ml (Trautwein and Hayes, 1991). Currently, the authors' laboratory considers values less than 150 nmol/ml to be critical. Using this standard, only the dog with the lowest plasma taurine concentration would also have been considered to be below the critical range with respect to whole blood taurine. This dog was being fed a diet supplemented with taurine. The manufacturer had added taurine to this diet because of previous reports linking it to low blood taurine concentrations and DCM in dogs (A. J. Fascetti, unpublished data). It should also be noted that dogs fed diets from this manufacturer had the overall third lowest mean plasma taurine concentration of the 17 manufacturers represented in the study. It is possible that the food this dog was consuming at the time of blood collection was produced prior to taurine supplementation and was still available for purchase because of delays in distribution. Of more concern is the possibility that the degree of supplementation was not enough to overcome the problem. Supplementation with taurine can improve taurine status (A. J. Fascetti, unpublished data), but care must be taken that the added taurine is in sufficient quantities to overcome the increased microbial degradation associated with heat processing and the formation of Maillard reaction products (Kim et al., 1996; Backus et al., 1998). Unfortunately, none of the diets fed were available for taurine analysis following the results of the study.

As methionine and cysteine are precursors of taurine, it would be expected that plasma taurine would correlate with plasma cysteine and methionine concentrations. These findings are consistent with work in cats where cysteine supplementation increased taurine synthesis (Laidlaw et al., 1987). Unfortunately, plasma cystine and methionine concentrations were not reported in that study. Similarly, in a second study, low taurine concentrations in dogs were corrected with methionine supplementation (R. C. Backus, unpublished data). However, plasma taurine concentrations were not significantly correlated with plasma concentrations of cysteine or methionine.

Although body size did not appear to affect taurine status, it cannot be dismissed that this could play a contributing role in the development of low taurine concentrations and DCM. The study could not, by design, control for dietary differences among participating dogs. Therefore, the lack of correlation between body size and taurine status may have been a result more of the variation in diets than the lack of a true correlation. However, it should be noted that numerous small and large dogs in the same household, fed the same diets failed to show differences in taurine status.

Breed differences were noted, but whether the differences noted are significant is unknown. However, this finding does support the theory that there may be a genetic contribution to taurine homeostasis that may predispose certain breeds to be prone to taurine deficiency. Breeds previously identified as having low taurine status were either absent from this study, such as the American Cocker Spaniel, (Kittleson et al., 1997) or were too small in number to show a statistical significance, such as the Newfoundland ($n = 3$) (R. C. Backus, unpublished data).

The lack of correlation between grams of daily protein intake per kilogram bodyweight and taurine status needs to be interpreted carefully. First, owner information about the quantity of food fed daily may have been incorrect in some cases, as the amount of food fed prior to blood collection was not measured. Secondly, it is known in cats that dietary taurine requirements vary with dietary protein quality and quantity (Backus et al., 1998). Therefore, without knowing the digestibility of each dietary protein source, the significance of the total amount of crude protein may be limited. It has been shown that there is a large variation in amino acid ileal digestibilities among common animal meals when fed to dogs (Johnson et al., 1998). In addition, it has been hypothesized that the requirement for essential amino acids in dogs increases with dietary protein intake (Delaney et al., 2001). Therefore, a high intake of protein might actually lower the taurine status of a dog if the requirement for sulphur amino acids has increased in response to an increase in dietary nitrogen intake.

A consistent overall factor affecting taurine status was the consumption of diets containing rice bran or whole grain rice. Dogs consuming diets in which the first plant source ingredient was whole grain brown rice had lower whole blood taurine concentrations than dogs fed an assortment of plant protein sources. Although the mean value was not below previously reported critical ranges, it was well below the study mean. It appears that the presence of dietary rice bran may be a possible explanation for the differences between plasma cysteine concentrations in dogs fed the lamb meal and rice diets, and lamb meal and ground rice diets. The lamb meal diets that used ground rice as the principle plant source ingredient all had rice bran as the second plant source ingredient, whereas the lamb meal and rice diets did not contain any source of rice bran. Differences in plasma methionine concentrations between dogs consuming chicken meal diets with brown rice or rice may be the result of the presence of rice bran as well. Rice bran was present in all the diets with rice as the first plant source ingredient, usually as the diet's second plant source ingredient, whereas there was no rice bran in the diets containing brown rice. The lower plasma methionine concentrations determined in dogs eating lamb meal and ground whole grain brown rice may be the result of the rice bran that is included in the whole grain brown rice. It has been reported that dietary rice bran decreases plasma and whole blood taurine concentrations in cats (Stratton-Phelps et al., 2002). The exact mechanism is currently under investigation. One proposed mechanism is that the fibre, fat and/or protein content of the rice bran may alter the excretion of bile acids thereby predisposing animals to taurine deficiency (Stratton-Phelps et al., 2002). The turkey and barley diet did not contain any rice bran, but it is possible that a similar mechanism may also occur with barley.

Dogs in this study fed commercial lamb meal and rice diets had lower blood taurine concentrations when compared with other animal and plant source ingredient combinations. This is consistent with two recent reports; one in Newfoundland dogs with low taurine concentrations but without concurrent cardiac disease, the second in a variety of breeds with low taurine concentrations and concurrent DCM (R. C. Backus, unpublished data; A. J. Fascetti, unpublished data). The mechanism for this may lie in the effects of rice bran (Stratton-Phelps et al., 2002) coupled with the decreased amino acid digestibility of lamb meal (Johnson et al., 1998). In fact, eight of the 15 dogs with the lowest plasma taurine concentrations were fed lamb, or lamb meal as the main animal source ingredient in their diet. Alternatively, three of the 15 dogs with the highest

plasma taurine concentrations were also fed lamb, or lamb meal as the main animal source ingredient in their diet. However, all three of those dogs were consuming diets with supplemental taurine. It was of special interest to discover that two of the eight dogs with the lowest plasma taurine concentrations were consuming lamb, or lamb meal diets that were also supplemented with taurine. This raises the concern that the level of supplementation may have to be increased to prevent taurine depletion in dogs fed lamb, or lamb meal and rice diets. In fact, the only dog with both plasma and whole blood taurine concentrations below the arbitrarily set critical levels was fed a lamb meal and rice diet supplemented with taurine. In addition, 10 of the 15 lowest plasma taurine concentrations were from dogs fed rice bran or whole grain rice as the first or second ingredient in their diet, whereas none of the 15 dogs with the highest plasma taurine concentrations received a majority of their daily calories from a diet that contained any rice bran or whole grain rice.

These findings support the theory that there is an effect of diet on blood taurine concentrations in dogs. Of specific concern are diets containing rice bran or whole grain rice coupled with lamb meal. Signalment and body size do not appear to play a critical role in taurine status, and may only play a synergistic role for which this study did not find any evidence. The findings of this paper further support the recommendation that dogs diagnosed with DCM have blood taurine concentrations analysed and receive taurine supplementation (1000–3000 mg/day, per os), pending results of plasma amino acid analysis (A. J. Fascetti, unpublished data). Further research to identify dietary factors that affect taurine concentrations in dogs and the mechanism of their effects is needed.

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