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Echocardiographic phenotype of canine dilated cardiomyopathy differs based on diet type☆

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Abstract *Introduction:* Canine dilated cardiomyopathy (DCM) can result from numerous etiologies including genetic mutations, infections, toxins, and nutritional imbalances. This study sought to characterize differences in echocardiographic findings between dogs with DCM fed grain-free (GF) diets and grain-based (GB) diets.
Animals: Forty-eight dogs with DCM and known diet history.
Methods: This was a retrospective analysis of dogs with DCM from January 1, 2015 to May 1, 2018 with a known diet history. Dogs were grouped by diet (GF and GB), and the GF group was further divided into dogs eating the most common grain-free diet (GF-1) and other grain-free diets (GF-o). Demographics, diet history, echocardiographic parameters, taurine concentrations, and vertebral heart scale were compared between GB, all GF, GF-1, and GF-o groups at diagnosis and recheck.
Results: Dogs eating GF-1 weighed less than GB and GF-o dogs, but age and sex were not different between groups. Left ventricular size in diastole and systole

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was greater, and sphericity index was less for GF-1 compared with GB dogs. Diastolic left ventricular size was greater for all GF compared with that of GB dogs. Fractional shortening, left atrial size, and vertebral heart scale were not different between groups. Taurine deficiency was not identified in GF dogs, and presence of congestive heart failure was not different between groups. Seven dogs that were reevaluated after diet change (6 received taurine supplementation) had clinical and echocardiographic improvement.
Conclusions: Dietary-associated DCM occurs with some GF diets and can improve with nutritional management, including diet change. The role of taurine supplementation, even without deficiency, is uncertain.

Abbreviations

CHF	congestive heart failure
GF	grain-free
GB	grain-based
GF-1	most common grain-free diet
GF-o	other grain-free diets
IFA	immunofluorescence assay
LVIDdN	normalized left ventricular internal diastolic diameter
LVIDsN	normalized left ventricular internal systolic diameter
PCR	polymerase chain reaction

Introduction

Dilated cardiomyopathy (DCM) is a common cause of congestive heart failure (CHF) in dogs and is historically more common in male, large-breed dogs with certain breed predilections including Doberman Pinschers, Great Danes, and Irish Wolfhounds [1—5]. Ventricular dilation and impaired contractility in the absence of primary valvular or vascular diseases are the hallmark findings of DCM [1]. A number of disease processes can lead to the development of systolic dysfunction, which is the major pathophysiologic driver for clinical signs including dyspnea, tachypnea, syncope, and sudden death. Genetic mutations associated with DCM have been suspected or identified in some predisposed breeds [3—5]. Additionally, systemic disease, toxins, and a variety of infectious agents have been reported to cause myocardial failure resembling DCM and leading to CHF [6—9]. Nutritional causes of DCM, such as taurine and carnitine deficiency, are especially important to identify in clinical patients, as there appears to be a degree of reversibility associated with supplementation of these compounds [10—14]. Although taurine and L-carnitine deficiencies have been associated with DCM in some dogs, with suspected breed predispositions, little is known about other diet-related etiologies [13—15]. In people, macronutrient or micronutrient deficiencies (e.g. selenium, zinc, thiamine, copper, iron, taurine, and carnitine) as well as nutrient toxicities (e.g. iron and cobalt) have also been associated with DCM [16—19]. Nutrient imbalances may promote inefficient myocardial energy expenditure and could cause or exacerbate DCM.

This study was initiated because of increased recognition of DCM in dogs without a known breed or demographic predilection, and an observation that many of these dogs were eating specialty meat-based grain-free (GF) diets. We hypothesized that among dogs diagnosed with DCM, those eating meat-based GF diets would have more advanced left ventricular systolic dysfunction and remodeling than those eating meat and grain-based (GB) diets. We also hypothesized that dogs eating one particular brand of most common grain-free diet (GF-1) would have more advanced disease than dogs eating other brands of grain-free diet (GF-o).

Animal, materials, and methods

Medical records at the North Carolina State University, College of Veterinary Medicine, were retrospectively evaluated for the diagnosis of canine DCM from January 1, 2015 to May 1, 2018. Dogs were included if the food brand and variety were known and an echocardiographic diagnosis of DCM was made based on fractional shortening $\leq$25%, normalized left ventricular internal diastolic diameter (LVIDdN) $\geq$ 1.8, and normalized left ventricular internal systolic diameter (LVIDsN) $\geq$ 1.2 [1,20]. Exclusion criteria were congenital heart disease, primary valve disease,

unknown diet history, or vegan, vegetarian, or home-cooked diets.

Dogs were grouped by diet type into GF and GB based on label ingredients as reported by the manufacturer. Foods were considered GB if wheat, rice, oats, cornmeal, barley, bulgur, millet, rye, or spelt were listed as ingredients and GF if none of these grain products were listed. The GF group was subdivided into dogs fed GF-1 and GF-o. Demographic information (age, breed, weight in kilograms [kg], and sex) was recorded. The recorded diet represented the main calorie source as reported by the owner. Echocardiographic parameters that were independent of or normalized for body weight (fractional shortening, LVIDdN by M-mode, LVIDsN by M-mode, ratio of the left atrial dimension to the aortic annulus dimension by 2-D right parasternal short-axis view [21], and diastolic left ventricular sphericity index [SI] as previously described [22,23]) were recorded from each included case. In addition, records were evaluated for the presence or absence of CHF at the time of diagnosis, and radiographic vertebral heart scale [24] was evaluated and recorded. Echocardiograms were reviewed by one investigator (DA). Results of ancillary testing such as whole blood or plasma taurine concentrations, plasma L-carnitine concentrations, blood selenium concentrations, infectious disease testing (Bartonella immunofluorescence assay [IFA] and polymerase chain reaction [PCR], Rickettsia IFA and PCR, Anaplasma IFA and PCR, Babesia PCR, Ehrlichia IFA and PCR, Mycoplasma PCR, Lyme IFA, Trypanosoma cruzi PCR, and Leishmania IFA), and necropsies were recorded where available. The established reference range for laboratories[a,b,c] and published studies were utilized for interpretation of tests [14,25,26].

Dogs that were diagnosed with DCM and underwent a diet change had serial echocardiographic information collected at each recheck during the study period.

Statistical analysis was performed using commercially available software[d]. Data collected from each group were tested for normality using the Kolmogorov-Smirnov test and presented as median and 95% confidence intervals. Demographic data, echocardiographic parameters, whole blood taurine concentrations, and radiographic vertebral heart scale at the time of diagnoses were compared between groups using one-way analysis of variance if data were normally distributed or Kruskal-Wallis test if data were not normally distributed. Post hoc testing was performed where indicated (Tukey's or Dunn's respectively). The presence or absence of CHF and sex were compared between groups using Fisher's exact test. Data from dogs with follow-up evaluations after diet change were assessed for echocardiographic changes using a 2-tailed, paired *t*-test.

Results

Dogs

A total of 91 dogs were diagnosed with DCM at the North Carolina State University, College of Veterinary Medicine, from January 1, 2015 to May 1, 2018. Forty-three dogs were excluded from group comparisons because of the lack of a diet history (41) or vegan or vegetarian diet (2). All dogs were treated with standard medications at recommended doses for CHF or for preclinical heart disease using a combination of pimobendan, enalapril, and furosemide if indicated.

Diets

Twelve of the included dogs were eating GB at the time of DCM diagnosis, and 36 were eating GF. Of the GF dogs, 14 were eating GF-1, and 22 were eating GF-o. There were two pairs of unrelated housemates included (both eating GF-1) and one pair of related housemate dogs included in the GF group. Seven brands of GB diets were represented, and 13 brands of GF were represented. The number of dogs eating each GB brand ranged from 1 to 3. The number of dogs eating each GF brand ranged from 1 to 5 for GF-o with 14 dogs eating GF-1.

A diet change from GF to GB manufactured by a major brand pet food company with veterinary nutritionists on staff was consistently recommended for all dogs in the GF group after June 2017 but was not recommended for the GB group and inconsistently recommended for the GF group before this time. Two dogs in the GF group were switched to a major brand food that was GF. All but one dog in the GF group received supplementation with taurine (30 mg/kg twice daily) after diagnosis and diet change, even if whole blood taurine concentrations were within or above the reference range.

[a] Metabolic Analysis Labs, Inc. Madison, WI.
[b] Amino Acid Laboratory, Davis, CA.
[c] Michigan State University, Veterinary Diagnostic Laboratory, Lansing, MI.
[d] GraphPad Prism 6, La Jolla, CA, USA.

Table 1 Median and 95% confidence intervals for weight, age, percentage male, percentage of dogs with congestive heart failure (CHF) at diagnosis, and vertebral heart scale.

	GB (n = 12)	All GF (n = 36)	GF-1 (n = 14)	GF-o (n = 22)	*P* value
Weight (kg)	36.5 (25.3–41.2)	30 (23.3–33.7)	21.0 (13.0–26.5)[a,b]	31.7 (27.1–39.9)	0.01
Age (yrs)	7.5 (4.7–9.7)	5.0 (4.9–6.9)	5.0 (4.6–7.6)	5.0 (4.5–801)	0.6
Male (%)	75	56	57	55	0.65
CHF (%)	67	78	93	68	0.32
VHS	11.4 (11.1–12.3)	12.4 (12.0–12.8)	12.5 (12.3–13.5)	11.9 (11.5–12.5)	NS

GB, grain-based diets; All GF, all grain-free diets, GF-1, most common grain-free diet, GF-o, other grain-free diets; VHS, vertebral heart scale.
[a] Significantly different from GB.
[b] significantly different from GF-o. VHS was significantly different between groups (p=0.04); however, this significance was lost after multiple comparison testing.

Demographic and radiographic data

Breeds of dogs eating GB diets included Doberman Pinscher (4), Labrador Retriever (2), Chihuahua (1), Weimaraner (1), Australian Shepherd (1), Cavalier King Charles Spaniel (1), Bouvier des Flandres (1), and German shepherd (1). Breeds of dogs eating GF diets included Doberman Pinscher (6), Golden Retriever (5), Great Dane (4), Labrador Retriever (4), mixed breed (4), Miniature Schnauzer (2), Yorkshire Terrier (1), Miniature Poodle (1), Standard Poodle (1), Malti-poo (1), Shetland Sheepdog (1), Old English Sheepdog (1), Chesapeake Bay Retriever (1), German Shorthaired Pointer (1), German Shepherd (1), Miniature Pinscher (1), and Boxer (1).

Table 1 summarizes demographic information (age, sex, and weight) and presence or absence of CHF at the time of diagnosis. There was no difference in age or sex between groups, but GF-1 dogs weighed less than GF-o dogs and less than GB dogs (p=0.01). No differences were identified between groups with regard to the presence of CHF at the time of diagnosis (p=0.3) or radiographic heart size as assessed by vertebral heart scale (p=0.04 between groups but not significant after multiple comparison testing).

Echocardiographic data

Table 2 shows the echocardiographic variables for each diet group. There was no difference in fractional shortening (p=0.9) between groups, but GF-1 dogs had higher LVIDdN (p=0.003) and LVIDsN (p=0.01) than GB dogs. Additionally, all GF dogs, regardless of brand, had higher LVIDdN compared with GB (p=0.003). Left ventricular diastolic SI was lower for GF-1 compared with GB diets indicating a more spherical left ventricular shape (p=0.02). Left atrial size, as assessed by the ratio of the left atrial dimension to the aortic annulus dimension, was not different between groups (p=0.5).

Ancillary testing

Figure 1 shows whole blood taurine and L-carnitine concentrations from dogs that were tested (21 [61%] of GF dogs and 5 [42%] of GB dogs had taurine testing, 4 [11%] of GF dogs had L-carnitine testing). No taurine (as assessed by whole blood or plasma concentrations) or plasma L-carnitine deficiencies were identified among the GF dogs tested (21 taurine, 4 L-carnitine) when utilizing the reference ranges supplied by the testing laboratory, and there was no difference in whole blood taurine

Table 2 Median and 95% confidence intervals for echocardiographic variables at diagnosis.

	GB (n = 12)	All GF (n = 36)	GF-1 (n = 14)	GF-o (n = 22)	*P* value
FS (%)	13.0 (11.1–17.8)	15.5 (13.8–16.9)	14.5 (11.9–18.2)	16.5 (13.6–17.3)	0.9
LVIDdN	2.13 (2.01–2.20)	2.36 (2.28–2.48)[a]	2.49 (2.36–2.64)[a]	2.22 (2.15–2.42)	0.003
LVIDsN	1.71 (1.57–1.79)	1.85 (1.80–1.99)	1.91 (1.86–2.16)[a]	1.77 (1.68–1.93)	0.01
SI	1.34 (1.30–1.58)	1.27 (1.26–1.37)	1.23 (1.19–1.30)[a]	1.32 (1.30–1.44)	0.02
LA:Ao	1.98 (1.74–2.16)	2.02 (1.88–2.15)	2.11 (1.89–2.39)	1.94 (1.78–2.10)	0.5

FS (%), percent fractional shortening; LVIDdN, normalized left ventricular internal diameter in diastole; LVIDsN, normalized left ventricular diameter in systole; SI, diastolic left ventricular sphericity index; LA:Ao, left atrial to aortic ratio; GB, grain-based diets; All GF, all grain-free diets, GF-1, most common grain-free diet, GF-o, other grain-free diets.
[a] Significantly different from GB.

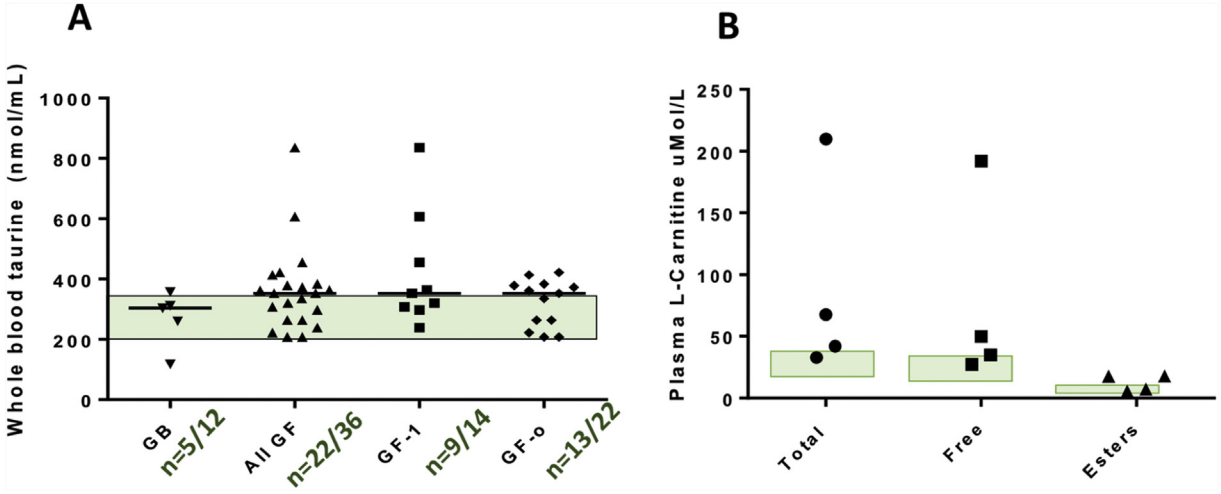


Fig 1 A) Dot plot for whole blood taurine concentrations for each group. The shaded area represents the reference range. The central line is the median. The number of dogs in each group with blood taurine concentrations evaluated is listed on the x-axis next to the group. $P = 0.4$ between groups. (B) Dot plot for plasma L-carnitine concentrations (total, free and esters) for 4 dogs in the GF group (2 GF-1 and 2 GF-o). The shaded area represents the reference range. GB, grain-based diets; All GF, all grain-free diets; GF-1, most common grain-free diet; GF-o, other grain-free diets.

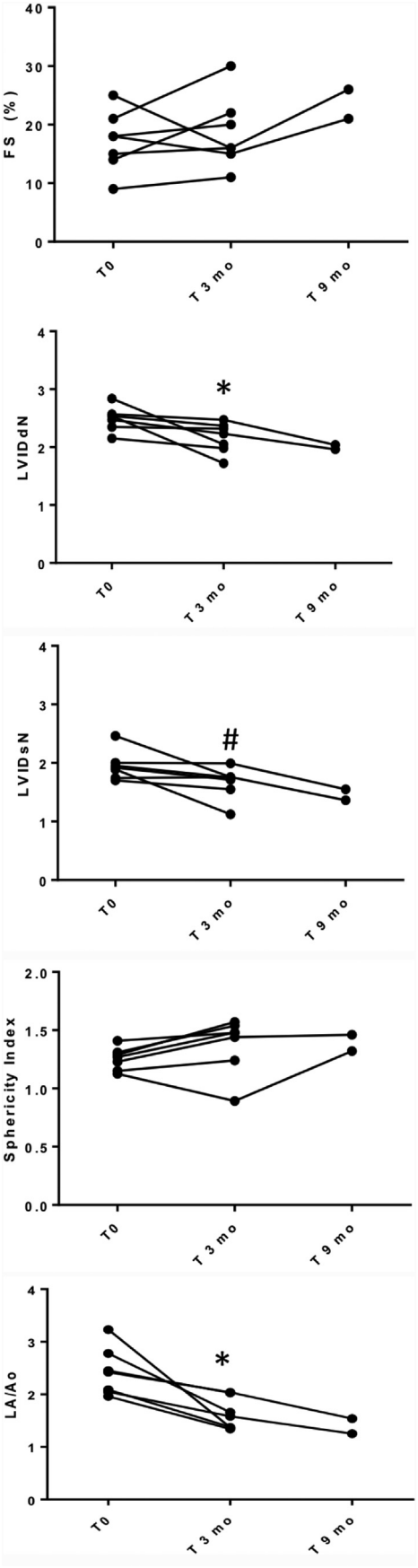
concentrations between groups (p=0.4). Three dogs in the GF group had whole blood taurine concentrations that were within the reference range (200—350 nmol/mL) but in the 25th percentile reported for normal dogs (whole blood 208, 208 and 222 nmol/mL) [25]. Twelve GF dogs and one GB dog had whole blood taurine concentrations above the reference range. Taurine deficiency was identified in two dogs being fed GB (whole blood 118 nmol/ml and plasma 38 nmol/ml). One of these GB died in-hospital (German shepherd), and the other dog clinically, radiographically, and echocardiographically improved with a diet change and taurine supplementation (Bouvier des Flandres), similar to previous reports of taurine-responsive DCM [10,12].

Four dogs in the GB group and eight dogs in the GF group had cardiac troponin I evaluated, and values were not different between groups (p=0.2, GB median = 0.37 ng/mL, range <0.20—0.83 ng/mL; GF median = 0.88 ng/mL, range = 0.26—14.30 ng/ml; reference range <0.2 ng/mL). Two dogs in the GF group were negative for selenium deficiency (serum = 331 ng/mL for one dog, reference range = 200—300 ng/mL, and whole blood = 405 ng/ml for another dog, reference range = 200—400 ng/mL). Five GF dogs were tested for infectious diseases with all negative results.

Three dogs in the GF group had myocardial histopathology performed as part of necropsy; findings were unremarkable in one dog, disclosed mild myocardial fibrosis with myofiber degeneration in a second dog, and myofiber vacuolar degeneration and atrophy with moderate subendocardial fibroelastosis and interstitial edema in a third dog. No specific etiology for DCM was evident on necropsy in any case.

Serial echocardiographic evaluations

Seven GF dogs had follow-up echocardiograms 3 months after DCM diagnosis and a diet change, and 2/7 dogs had another follow-up echocardiogram 9 months after diagnosis and a diet change. Six of these seven dogs were eating GF-1 at the time of diagnosis, and one was eating GF-o. The diet was changed from GF to GB in 5/7 dogs and to a major brand GF diet in 2/7 dogs. Taurine was supplemented in 6/7 dogs despite whole blood taurine concentrations that were within or above the reference range (5 dogs), or because taurine was not measured at presentation (1 dog). One of the seven dogs did not receive taurine supplementation because whole blood taurine concentration was above the reference range. Figure 2 shows the change in echocardiographic parameters between diagnosis and 3 months after diagnosis for seven dogs and 9 months after diagnosis for two of these dogs. There was a decrease in LVIDdN (p=0.02), LVIDsN (p=0.05), and ratio of the left atrial dimension to the aortic annulus dimension (p=0.02) at the 3-month recheck. Fractional shortening (p=0.9) and SI (p=0.2) did not change



at the 3-month recheck. Two dogs were rechecked a second time 9 months after diet change. One of these dogs received taurine supplementation and showed progressive improvement at both 3 and 9 months. The other dog did not receive taurine supplementation because whole blood taurine concentrations were high, and this dog showed improvement at 9 months that was not evident at 3 months. One of the seven dogs had a normal echocardiogram 2 years prior to presentation with CHF due to DCM, and this dog also showed echocardiographic improvement 3 months after DCM diagnosis, diet change, and taurine supplementation.

Serial clinical evaluations

No GF dogs experienced new onset or recurrent CHF after diet change, but one dog died suddenly 4 months after diagnosis (1 month after the 3-month recheck which showed echocardiographic improvement). No GF dogs required an increase in diuretic dosage, and furosemide was discontinued in three dogs 6–9 months after diagnosis and diet change.

Discussion

This retrospective study showed that body weight and some echocardiographic variables differ between dogs with DCM depending on the diet-type being fed. The lower median weight for GF-1 dogs compared with GB dogs supports causes other than genetic because small breed dogs are not considered typically predisposed to the development of DCM [2]. For their size, dogs eating all GF diets had larger left ventricular diastolic diameters than dogs eating GB diets. Additionally, dogs eating the GF-1 in this study not only had larger left

Fig 2 Echocardiographic parameters are shown for seven dogs eating GF diets at the time of diagnosis and at subsequent recheck evaluations after diet change and taurine supplementation in 6/7 dogs. Seven dogs were reevaluated 3 months after they were switched to a GB diet (5 dogs) or major brand GF diet (2 dogs). Two of these dogs were evaluated again 9 months after diagnosis. *$p<0.05$ compared to baseline. $^{\#}p=0.05$ compared to baseline. FS (%), percent fractional shortening; LVIDdN, normalized left ventricular internal diameter in diastole; LVIDsN, normalized left ventricular diameter in systole; SI, diastolic left ventricular sphericity index; LA:Ao, left atrial to aortic ratio; T 0, time 0 months (baseline); T 3 mo, time 3 months; T 9 mo, time 9 months.

ventricular end diastolic diameters but also had larger left ventricular end systolic diameters and lower sphericity indices compared with dogs eating GB, indicating more advanced myocardial dysfunction and remodeling in these dogs. These findings do not appear to be explained by differences in disease stage because the number of dogs with CHF in each group was not statistically different. Significant differences in echocardiographic variables were not found between GF-o and GB. It is possible that the differences between all GF and GB are mainly due to the dramatic effect of GF-1 values, but small numbers and inadequate statistical power may have also influenced these results.

The clinical and echocardiographic improvement observed in seven dogs that were eating GF diets following a diet change deserves emphasis. The improvement in GF dogs after diet change supports potential causality, and the finding of two affected pairs of unrelated housemates eating GF-1 also causally supports diet as a common environmental factor for the development of DCM in these dogs.

The differences in echocardiographic variables suggest a unique pathologic remodeling process occurring in dogs eating GF diets and especially GF-1, but the cause of this presumed diet-related DCM is not known. The predominant legumes in these diets are peas or lentils, and it is possible that the processing, sourcing, or interactions of these or other ingredients could have resulted in deficiency or toxicity of important nutrients in a manner that did not occur with GB diets. The predominance of one particular GF diet (GF-1) in these dogs with DCM suggests that there are differences within the category of GF diets, and that there may be a separate, unidentified mechanism other than GF composition that is responsible for disease. Additionally, the clinical and echocardiographic improvement seen in two GF-1 dogs that were switched to a major brand GF diet suggests that factors besides GF composition or legume type are important, again raising the possibility that sourcing, processing, or interaction of ingredients may be problematic for some diets. Although most of the group differences in echocardiographic parameters appear attributable to GF-1 diet, one of the dogs that responded to a diet change was eating GF-o, supporting a similar pathophysiologic process and reversibility for GF-o diets.

Most dogs eating GF diets in this study received taurine supplementation in addition to undergoing a diet change, even though whole blood taurine concentrations were normal or elevated in the dogs that were evaluated. The role of taurine supplementation in this condition is, therefore, uncertain. Taurine may have contributed to improvement in these dogs as a result of its antioxidant effects, calcium handling effects, or positive inotropic effects [27—29]. Oxidative damage and negative energy balance in the failing myocardium may be improved with taurine supplementation, and it is possible that whole blood concentrations within the reference range provide insufficient support for the failing heart, essentially rendering it a conditionally required amino acid [27,28]. While it is difficult to draw conclusions from low case numbers, the single dog that did not receive taurine supplementation showed minimal echocardiographic improvement at 3 months but did improve 9 months after diagnosis, potentially suggesting slower improvement compared with dogs that were supplemented with taurine. Variability between dogs may also account for this slower improvement. We noted that several dogs eating GF diets had very high whole blood taurine concentrations. These dogs were not supplemented before the diagnosis of DCM; therefore, these values are unexpected in light of studies of taurine concentrations of normal dogs and dogs with DCM [25,26]. Elevated whole blood taurine has been reported in people after myocardial infarction because taurine is rapidly taken up by platelets after myocardial release into the bloodstream with cardiomyocyte injury and death [30,31]. The high values noted in some GF dogs may have been related to myocardial injury. Although some dogs also had cardiac troponin I evaluated, there were too few dogs with both tests performed to meaningfully explore a correlation. The possibility of masked taurine deficiency could be investigated in the future by assessing paired plasma and whole blood taurine concentrations in affected dogs.

This study had several limitations inherent in its retrospective design. The presence of a detailed diet history was inconsistent in our medical records during this study period, which reduced the number of included dogs and could have introduced bias. Dietary intake beyond the commercial dog food that was reported by the owner as the major source of calories, such as treats and supplements, was not recorded and could play an important role in the development of nutritionally based DCM. Additionally, the length of time that dogs had been eating their commercial food before presentation was not consistently recorded. Ancillary testing was not always performed. Although a relatively large number of GF dogs underwent taurine testing which was normal or

elevated, not all dogs had taurine assessed, and we may have missed some deficient dogs. Although our data are most supportive of a causation with one particular diet (GF-1), other GF diets were fed to dogs in the GF group, some to as many as four or five dogs with DCM and some only to a single dog. Because these numbers are small, it is difficult to interpret a role for causation for some less represented diets. Additionally, the improvement noted in two dogs that were switched from GF-1 to a major brand of dog food that was also GF raises the possibility that GF composition may not be the problem. Thus, our group divisions into GF and GB may be irrelevant and subject to refinement in the future, pending ongoing investigations. Additionally, group divisions could be incorrect if the underlying cause of this nutritional cardiomyopathy is related to unusual protein sources or specific manufacturing practices. Some of the dogs in this study were breeds that have a known predisposition to DCM, and follow-up was not obtained for most of the dogs, so it is possible that diet was independent of their disease process. While we cannot discount this possibility for many dogs, one dog that showed echocardiographic improvement was a Great Dane eating GF-o, indicating a probable role for diet in his disease process.

Grain-free diets are rapidly growing in popularity, and it is impossible to determine the prevalence of DCM in dogs eating GF diets without broadly screening populations of dogs by echocardiography. Although we identified 36 dogs with DCM eating GF diets during a 30-month period, this is no doubt a vanishingly small percentage of dogs eating GF diets. Factors such as age, exercise, genetic predisposition, dietary or microbiota modification of gene expression, or other unknown factors may clearly also influence the manifestation of disease in some dogs. A specific and possibly even variable milieu, to which some GF diets may contribute, may be responsible for the clinical development of DCM. Additionally, the length of time necessary for disease development on a specific diet is unknown.

Despite these limitations, this study reveals morphologic differences between the echocardiograms of dogs eating GF and GB diets that point to a potential association between some GF diets and DCM in dogs. Additionally, the echocardiographic differences between GF-1 and GF-o suggest brand differences that could be related to ingredient bioavailability, processing, sourcing, or interactions. This descriptive study should serve as a springboard for future studies to mechanistically explore this relationship and also to encourage clinicians to incorporate dietary history as part of every examination. Food analysis is underway to investigate possible nutrient deficiency or toxicity that could promote the development of DCM possibly related to legume sourcing or processing or interactions with absorption of other nutrients. Dietary trials to determine the risk of DCM associated with certain GF diets would also be useful.

Conclusions

This study shows compelling evidence that a nutritionally based, partially reversible cardiomyopathy occurs in some dogs fed non-major brand GF diets and may be associated with factors other than the omission of grains. The echocardiographic changes seen in these dogs indicate more advanced disease or a diet-enhanced pathophysiology compared with dogs eating GB diets. Importantly, clinical and echocardiographic improvement was noted in a subset of dogs that were reevaluated after a diet change. The role of taurine supplementation despite adequate blood concentrations is uncertain, but it may be therapeutically additive. Dietary-induced DCM associated with some GF diets remains unproven but should be considered a potential underlying cause or contributor in dogs with DCM, regardless of signalment.

Conflicts of interest statement

Dr. Adin has received/acknowledges research support from Nestlé Purina PetCare. None of the other authors have a conflict of interest to disclose that relate to this study.

Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jvc.2018.11.002.

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