Diet-associated DCM first came to light in cats in the late 1980s and in dogs in the mid-1990s. The association between diet and DCM in dogs has generally not been much in the news since the early 2000s, but over the past few years, an increasing number of DCM cases involving dogs appear to have been related to diet. The extent of this issue is not clear, not all cases have been confirmed to be linked to diet, and a true association has not been proven to exist. However, when one of the authors (RF) recently surveyed veterinary cardiologists about cases of possible diet-associated DCM in dogs examined in the past 2 years, information for > 240 cases was obtained, with responses received from the United States, United Kingdom, Canada, Israel, and Austria (unpublished data). Dogs for which breed was specified consisted of mixed-breed dogs (n = 134), Golden Retrievers (23), Labrador Retrievers (9), German Shepherd Dogs (8), Cocker Spaniels (7), and between 1 and 5 dogs each of 25 other breeds. Further, possible diet-associated DCM represented 16% of all cases of DCM diagnosed by the respondents during this period.

The recent announcement from the US FDA alerting pet owners and veterinarians about reports of DCM in dogs eating pet foods containing peas, lentils, other legume seeds, or potatoes as main ingredients has raised concerns among the pet-owning public. Therefore, we wanted to increase awareness of this issue among veterinarians, review what is currently known about the possible association between certain diets and DCM in dogs, and discuss what veterinarians can do to help identify underlying causes.

Diet and Diet in Dogs and Cats

Dilated cardiomyopathy used to be one of the most common cardiac diseases in cats. In 1987, however, Pion et al published a landmark paper reporting that DCM in cats was associated with taurine deficiency and could be reversed by providing supplemental taurine. On the basis of that report and substantial subsequent research, the requirement for taurine in cat foods was increased, and taurine deficiency-related DCM is now uncommon in cats. However, it can still be seen in cats eating home-prepared diets or commercial diets prepared with inadequate nutritional expertise or quality control.

In 1995, veterinary cardiologists investigating the role of taurine deficiency in dogs with DCM suggested that certain breeds (eg, Golden Retrievers and American Cocker Spaniels) may be predisposed to taurine deficiency, and a study in Cocker Spaniels subsequently showed that supplementation with taurine and l-carnitine could partially or completely reverse the disease. Additional dog breeds potentially predisposed to taurine deficiency–associated DCM were identified, including Newfoundlands, English Setters, Saint Bernards, and Irish Wolfhounds. Later, certain types of diets, including lamb and rice, low-protein, and high-fiber diets were associated with taurine deficiency in some dogs. Research suggested that other ingredients (eg, beet pulp) may also increase the risk of taurine deficiency, although the exact role of these ingredients was still unclear. In addition, the apparent breed predispositions suggested that genetic factors, breed-specific metabolic abnormalities, or low metabolic rates may also have been playing a role.

Current Concerns About Diet and DCM in Dogs

Beginning in the early 2000s, the number of dogs with taurine deficiency and DCM subjectively appeared to decrease. Recently, however, we have heard from veterinary cardiologists who had an impression that they were diagnosing DCM in Golden Retrievers at higher rates than expected and in dogs of breeds
Diet-associated DCM in dogs with taurine deficiency

Golden Retrievers have been reported, as a breed, to be susceptible to development of taurine deficiency–associated DCM, leading some to suggest a breed-wide genetic propensity for diet-associated DCM. One of the authors (JAS) recently concluded a study evaluating 24 Golden Retrievers with eco-cardiographically confirmed DCM and low plasma or whole blood taurine concentrations that were followed up for 12 to 24 months after a diet change and the addition of supplemental taurine to their diet (unpublished data). Although the results are still preliminary, all but 1 dog for which follow-up data were available had substantial echocardiographic improvement. In addition, in all 9 dogs that initially had CHF, the heart failure resolved, and diuretic administration was substantially reduced or safely discontinued. All 24 of these Golden Retrievers were eating BEG diets at the time DCM was diagnosed.

Although taurine deficiency appears to be more common in Golden Retrievers than in dogs of other breeds, plasma and whole blood taurine concentrations should be measured in every dog with DCM because some dogs of other breeds with DCM have been found to have taurine deficiency. Even dogs of breeds that have previously been found to be genetically predisposed to developing DCM, such as Doberman Pinschers and Boxers, should be tested because taurine concentrations have been found to be low in some of these dogs also. In addition, taurine deficiency should be considered as a possibility not just in dogs eating BEG, very-low-protein, or high-fiber diets, but also in dogs eating vegetarian, vegan, or home-prepared diets.

The reasons for taurine deficiency in dogs are not completely understood but could be related to reduced synthesis of taurine resulting from an absolute dietary deficiency of the taurine precursors methionine and cystine; reduced bioavailability of taurine, methionine, or cystine in the diet; abnormal enterohepatic recycling of bile acids because of fiber content of the diet; increased urinary loss of taurine; or altered metabolism of taurine in the intestine as a result of interactions between certain dietary components and intestinal microbes. In addition to the possibility of breed-related metabolic differences, there may be genetic factors that play a role in susceptibility to taurine deficiency, as appeared to be the case in cats with taurine deficiency.

Diet-associated DCM in dogs without taurine deficiency

Preliminary results of a study performed by one of the authors (DBA) found that dogs with DCM that had been eating grain-free diets had more advanced cardiomyopathic changes than did dogs with DCM that had been eating grain-based diets. Unreported results of the study indicated that a subset of dogs without diet, compared with a chicken-based diet, and can be affected by the amount and types of fiber in the diet.
clinically and echocardiographically improved after a diet change. Notably, however, some dogs improved after a diet change from one grain-free diet to another, and this finding, along with the differences identified between dogs fed various Beg diets, suggested that DCM was not necessarily tied to the grain-free status of the diet. Taurine supplementation was prescribed for many of these dogs despite the lack of apparent deficiency, and it is unclear what role taurine may have played in their recovery.

Although DCM in some dogs without any apparent taurine deficiency appears to be reversible with a change in diet, with or without taurine supplementation, no cause has thus far been identified for non-taurine deficiency-associated DCM. Possible causes that are being investigated include absolute deficiencies of other nutrients, altered bioavailability of certain nutrients because of nutrient-nutrient interactions, and the inadvertent inclusion of toxic ingredients.

For example, Beg diets could possibly be more likely to have deficiencies of nutrients other than taurine, such as choline, copper, L-carnitine, magnesium, thiamine, or vitamin E and selenium, that have been associated with cardiomyopathies.19 Although pet foods are required to be nutritionally complete and balanced (unless they have a label statement that they are for intermittent or supplemental use only), that does not always provide a guarantee, and deficiencies could occur if diets do not contain appropriate amounts of all dietary nutrients. Further, a deficiency may occur even if a diet contains the required minimum amount of a nutrient because of reduced bioavailability or interaction with other ingredients in the diet. This may be a concern for diets based on exotic ingredients, whose nutritional properties may not be as well studied.

Researchers are also exploring whether diet-associated DCM in dogs without taurine deficiency may be related to inclusion of a cardiotoxic ingredient in the diet. This could be an adulterated ingredient, as with ingredients containing melamine–cyanuric acid that affected pet foods in 2007, resulting in extensive recalls; a heavy metal; a chemical sprayed on 1 of the ingredients; or even a natural chemical compound in 1 of the ingredients that has toxic effects when fed in large amounts.

Of course, the cause may be even more complicated, such as an interaction between gut microbiota and a dietary factor (eg, trimethylamine N-oxide).22 It is encouraging that some recovery of cardiac function has been observed in some dogs following a change in diet, with or without taurine supplementation. However, research is needed to identify the underlying cause.

**Diet History**

For many years, veterinary nutritionists have emphasized the importance of nutritional assessment.23,24 Nutritional assessment includes 4 key components: body weight, body condition score, muscle condition score, and diet history. Body weight and body condition score are likely already a part of most clinicians' standard physical examination, and muscle condition scoring would be a valuable addition. Cardiac cachexia (muscle loss) occurs early in patients with CHF and should be detected at its mildest stages, when interventions are more likely to be successful.25 Muscle condition scoring charts and training videos are available.26,27

The fourth component of nutritional assessment—diet history—may not be routinely collected but is equally important. A diet history, for example, can help identify issues that could be contributing to an underlying disease. For patients with recent-onset CHF, for example, the diet history may reveal that the owner changed to a new diet with a higher sodium content. Other diet-associated issues that can be identified from the diet history include anemia or thiamine deficiency caused by a nutritionally unbalanced home-prepared diet or diarrhea due to a contaminated raw meat diet. Veterinary cardiologists examining dogs with DCM were able to make an association with Beg diets because they were obtaining a diet history, and obtaining a diet history may help researchers identify patterns (eg, products made by the same manufacturer or by manufacturers using ingredients from the same supplier) that could eventually lead to determining the underlying cause.

A diet history can also identify an individual patient's food preferences, such as whether canned or dry food is preferred or whether specific flavors are preferred, that can be helpful for feeding when the patient is hospitalized. And, a diet history is useful in determining whether the patient's usual diet is appropriate after discharge or needs to be changed. For example, dietary modification will be required for dogs with cardiac disease that are eating high-sodium dog food or treats.

The diet history should include the main foods being fed. However, this is more than just “dry dog food” or “brand X dog food.” It is critical to solicit information on brand, the exact product, and even the flavor, as these factors can make a big difference in the ingredients and nutrient profile. We recommend telling owners that their description of a product should be detailed enough that we could go to the store and buy the exact product they are feeding. If owners are feeding a home-prepared diet, the exact recipe should be provided.

Of course, pet food is often just the tip of the iceberg. The diet history should also include all treats; table food; rawhides, bully sticks, and other chews; dietary supplements; and foods used to administer medications. These other components of the diet can contribute large amounts of sodium and other nutrients to a patient’s overall intake or unbalance the overall diet. In addition, these other components may contribute to adverse effects. For example, a Fanconi-like syndrome associated with jerky treats has been reported but may not have been identified if complete diet histories had not been obtained for affected dogs. In addition, although diet-associated DCM is most likely related to pet food, it may possibly be a result of another dietary
component (eg, treats, chews, or supplements) commonly fed to dogs eating these diets.

Use of a standard form, such as the generic form recommended by the World Small Animal Veterinary Association,26 or a cardiology-specific form (Supplementary Appendix S1), available at avmajournals.avma.org/doi/suppl/10.2460/javma.253.11.1390) will facilitate obtaining a complete diet history. We recommend all clinicians collect a diet history for every dog and cat patient at every appointment. Because many owners are unable to recall specific diet details at the time of their appointment, we recommend having owners complete the diet history form at home prior to the appointment so that they can provide exact details on all components of the diet.

Recommendations

If DCM is diagnosed in a dog that is eating a BEG, vegetarian, vegan, or home-prepared diet, we recommend measuring plasma and whole blood taurine concentrations.9 It is still unclear whether plasma or whole blood taurine concentration more accurately reflects myocardial concentration in dogs, so measurement of both plasma and whole blood taurine concentrations is recommended. However, if cost is an issue, measurement of whole blood taurine concentration should be prioritized because it is thought to be a better indicator of long-term taurine status. Importantly, reference ranges for taurine concentrations in dogs should be interpreted cautiously. Dilated cardiomyopathy has been diagnosed in some dogs, particularly Golden Retrievers, with whole blood taurine concentrations between 200 and 250 nmol/L, which would generally be considered within reference limits, although at the low end of the reference range. At least some of these patients, however, have responded well to a diet change and taurine supplementation. Therefore, reference ranges for plasma and whole blood taurine concentrations may need to be breed specific. Research in Golden Retrievers with taurine deficiency–associated DCM is ongoing, but a whole blood taurine concentration of at least 250 nmol/L is recommended for this breed.

We also recommend that all other dogs in the household that are eating the same diet be screened for DCM. Further, we recommend that owners of dogs with possible diet-associated DCM be instructed to save samples of all dietary components they are currently feeding, including not only the main food itself but also all treats, chews, and supplements. Ideally, this would include not just samples of the dietary components but also product bags or labels. With complete diet information in hand, the veterinarian or owner should report the case to the FDA, which can be done either online or by telephone20 because this will help the agency identify possible underlying causes as quickly as possible. A recently published article30 provides an excellent summary of information for veterinarians on reporting suspected animal food issues. If the dog is a Golden Retriever, the veterinarian or owner may also consider reporting the case to the Josh Stern Cardiac Genetics Laboratory,21 which is currently evaluating possible genetic factors that may increase susceptibility to taurine deficiency.

For dogs in which possible diet-associated DCM is diagnosed, we recommend the owner change the diet to one made by a well-established manufacturer that contains standard ingredients (eg, chicken, beef, rice, corn, and wheat). In the authors’ (LMF and JER) hospital, we recommend several specific products with a low sodium content that only contain standard ingredients.32 We also emphasize that changing to a raw or home-prepared diet may not be sufficient to improve cardiac abnormalities and may increase the risk for other nutritional deficiencies or infectious diseases. For dogs that require a home-prepared diet or that have other medical conditions that require special dietary considerations, consultation with a board-certified veterinary nutritionist is recommended.

We also provide supplemental taurine for all dogs with possible diet-associated DCM. In dogs with a taurine deficiency, taurine supplementation is critical. In dogs with taurine concentrations within reference limits, it is unclear whether taurine supplementation is needed, and some patients have recovered with only a diet change. However, taurine supplementation may still have some benefits owing to other effects of taurine (eg, antioxidant and positive inotropic effects). Taurine supplements from manufacturers with a history of good quality control should be used. A 2009 study33 identified certain brands with good quality control. In addition, ConsumerLab is expected to release a report in late 2018 on independent quality control testing of taurine supplements.

Although the optimal taurine dosage for dogs with taurine deficiency is not fully understood, we recommended 250 mg, PO, every 12 hours for dogs weighing < 10 kg (22 lb); 500 mg, PO, every 12 hours for dogs weighing 10 to 25 kg (55 lb); and 1,000 mg, PO, every 12 hours for dogs weighing ≥ 25 kg.

Follow-up echocardiography should be performed in 3 to 6 months. In our experience, some improvements are typically evident in this time span. However, in certain dogs, it may take even longer for improvements to be apparent echocardiographically.

Finally, although an association between BEG diets and DCM in cats has not been recognized, we recommend collecting diet histories on all cats as well and especially in cats with DCM. If cats with DCM are eating a BEG, vegetarian, vegan, or home-prepared diet, we recommend following the same protocol as described for dogs.

Summary

Pet food marketing has outpaced the science, and owners are not always making healthy, science-based decisions even though they want to do the best for their pets. The recent cases of possible diet-associated DCM are obviously concerning and warrant vigilance within the veterinary and research communities. Importantly, although there appears to be an association between DCM and feeding BEG, vegetarian, vegan, or home-prepared diets in dogs, a cause-and-effect rela-
tionship has not been proven, and other factors may be equally or more important. Assessing diet history in all patients can help to identify diet-related cardiac diseases as early as possible and can help identify the cause and, potentially, best treatment for diet-associated DCM in dogs.

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Footnotes


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