veterinary focus #31.3
The worldwide journal for the companion animal veterinarian
2021 – $10 / 10€

NUTRITION TOPICS IN SMALL ANIMAL PRACTICE

ROYAL CANIN®

ECVIM-CA

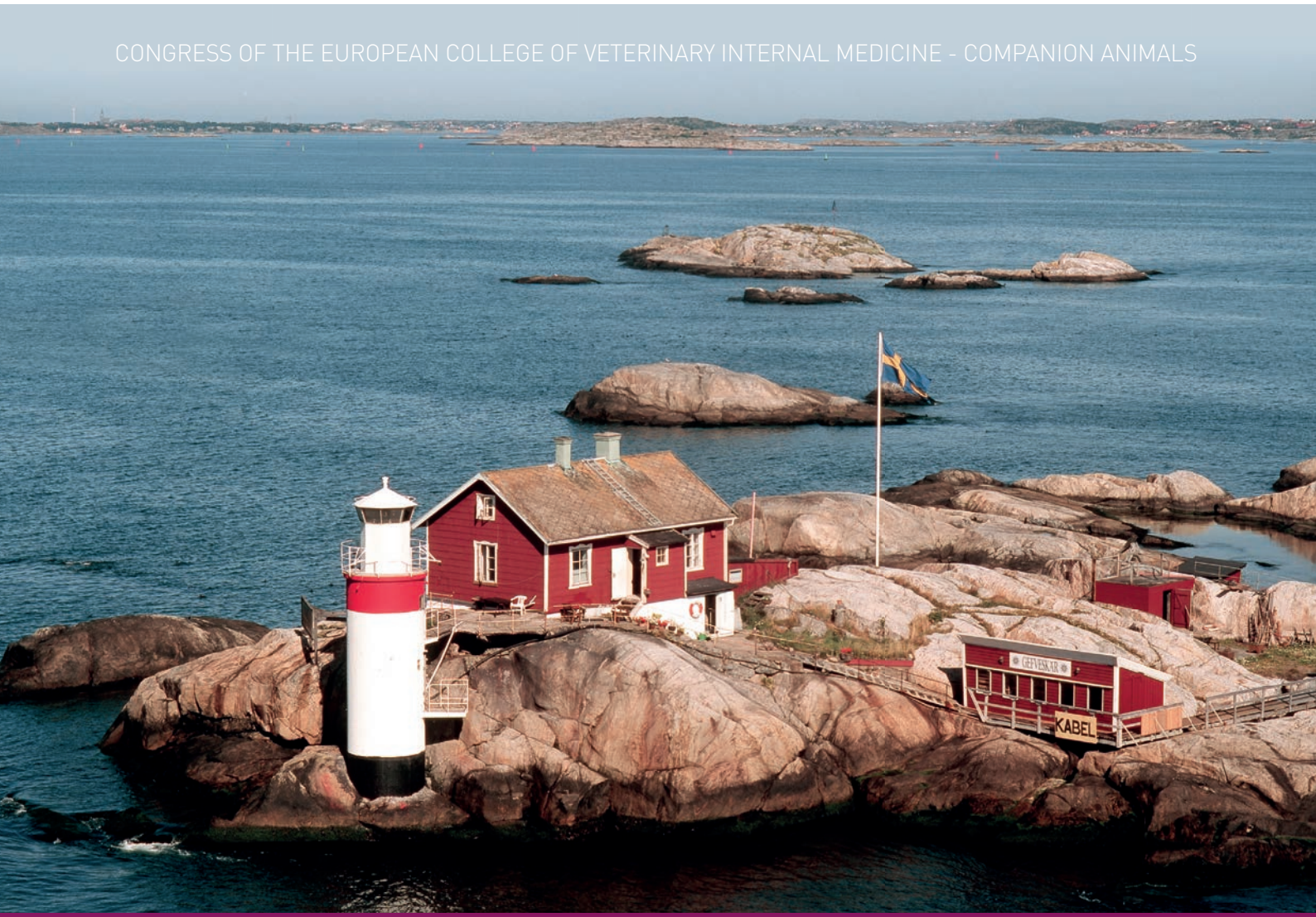
32ND ANNUAL CONGRESS

1-3 SEPTEMBER 2022

GOTEBORG – SWEDEN

SWEDISH EXHIBITION & CONGRESS CENTRE, GOTHIA TOWERS

CONGRESS OF THE EUROPEAN COLLEGE OF VETERINARY INTERNAL MEDICINE - COMPANION ANIMALS



SAVE THE DATE

WWW.ECVIMCONGRESS.ORG

FOOD FOR THOUGHT

***"Knowledge is the food of the soul"* – Plato**

Like most branches of science, the road that is the study of nutrition has been a long and winding one, and has involved many diversions and wrong turns into side paths or dead ends to get to where we are today in terms of knowing what is good - or bad - for both us and our animals. In fact, there is a good deal of evidence that the history of dietetics can be traced as far back to the beginnings of civilization, as confirmed by the writings of the Greek ancients such as Homer and Hippocrates. Conspicuous amongst these are the philosophies of Plato, who offered authority on almost every aspect of human knowledge, including what one should or should not eat. Indeed, his belief in the fundamental principle of moderation is evident in his references to Greek cuisine, declaring that the best diet should be based around cereals, fruits, honey and fish, with red meat and wine consumed only in modest quantities. Not that we would necessarily find everything listed on a Greek menu from 2,000 years ago to be appetizing – whilst we would no doubt enjoy the olives, figs and pomegranates, some of the other items would be less appealing. These included eels, locusts and small birds, whilst the universally popular garum, a fermented fish sauce that was liberally used as a condiment, would find little favor – as would the famous Spartan black soup, which was made with boiled pork meat and blood and flavored with salt and vinegar.

But Plato was ahead of his time in believing that excessive food intake could lead to ailments, as evidenced in his advocation for portion control. He also held that physicians should be responsible for regulating the human diet, seeing it as a science and not merely - as in the case of cookery - an art.

So while times change and we learn more about nutrition, this issue of *Veterinary Focus* opens up the path ahead to direct us towards better nutrition for our pets. Unlike the locusts, black soup or garum, it should leave the reader wanting more.

Ewan McNEILL
Editor-in-chief

In this issue of *Veterinary Focus*

Canine microbiome dysbiosis p.02
Jan S. Suchodolski

Myths in cat nutrition p.10
Karolina Hołda

Homemade diets – Good or bad? p.15
Marjorie L. Chandler

Diet and canine dilated cardiomyopathy p.22
Jennifer Larsen and Joshua A. Stern

FAQs about cat nutrition p.27
Ana Luisa Lourenço

Pet obesity: new challenges, new solutions p.33
Alexander J. German

Calcium and phosphorus – Getting the balance right p.42
Linda Böswald and Britta Dobenecker

CANINE MICROBIOME DYSBIOSIS

It is increasingly being recognized that a dysfunctional intestinal microbiome can be at the root of many gastrointestinal disorders; this paper discusses the diagnosis and therapeutic options for dysbiosis cases.

KEY POINTS

1. The intestinal microbiome is a metabolic organ that has a major impact on the health of the host.
2. Dysbiosis is an early marker of an abnormal gut environment, with treatment of the underlying disease being required for long-term resolution.
3. Dietary modulation should be the first line of treatment with dysbiosis associated with chronic enteropathies, as it is often clinically effective and has minimal side effects.
4. Fecal microbiota transplantation (FMT) is an emerging potential treatment for dysbiosis, but its use in dogs is still at the trial stage.

Introduction

The intestinal microbiome is the name given to the collective genome of all microbes (*i.e.*, bacteria, viruses, fungi, and protozoa) in the gastrointestinal (GI) tract, with bacteria being the most abundant constituent. The microbiome can be seen as both a component of the immune system and as a metabolic entity, as the bacteria produce metabolites that affect both the GI tract and other body organs. Dysbiosis is the name given to changes that occur in the microbiome during disease, and encompasses a reduction in microbiome diversity (*e.g.*, number of different bacteria), changes in quantities of bacteria, and functional changes (*e.g.*, altered production of bacteria-derived metabolites). Dysbiosis often occurs secondary to underlying pathologies within the intestine and will contribute to the clinical signs in some patients (1); because of this it is an additional marker for intestinal disease and should be assessed along with a patient's overall history and clinical presentation. Therapy for dysbiosis should aim to address the underlying pathology, with dietary manipulation as the first-line treatment.

Microbiome function

Bacteria either directly produce (vitamins) or convert dietary (fiber, protein, fat) or host (bile acids) molecules into bacteria-derived metabolites, and therefore the microbiota exerts many beneficial effects on the host. Important metabolites include short chain fatty acids (SCFA), indoles and secondary bile acids; these have various effects, including anti-inflammatory actions, modulation of intestinal motility, inhibition of enteropathogens, improvement of gut barrier function, and increased mucin production (2). Dysbiosis, which is often secondary to various luminal factors **(Box 1)**, leads to altered microbiota function which then contributes to clinical signs (1). Of particular interest to the regulation of microbiota are intestinal bile acids (BA). Briefly, primary BAs (cholic and chenodeoxycholic acids) are released into the small intestine after a meal to aid in fat digestion. Up to 95% of BA are reabsorbed in the

Box 1. Conditions and factors associated with intestinal dysbiosis.

- Exocrine pancreatic insufficiency (EPI), leading to undigested food in the GI lumen
- Chronic enteropathies, whereby intestinal inflammation fosters aerobic conditions and changes in the pH at the mucosal level
- Broad-spectrum antibiotics (*e.g.*, tylosin, metronidazole), which reduce normal levels of intestinal anaerobic bacteria
- Acid-suppressing drugs, which decrease gastric acid output
- Anatomic abnormalities
- Motility disorders

Jan S. Suchodolski

Dr. med. vet., PhD, AGAF, Dip. ACVM, College of Veterinary Medicine, Texas A&M University, USA

Dr. Suchodolski graduated in 1997 from Vienna's University of Veterinary Medicine and worked for several years in a small animal specialty clinic before returning to academia. He is board-certified in immunology by the American College of Veterinary Microbiologists (ACVM) and was awarded his Ph.D. from Texas A&M University for his work on molecular markers for the assessment of the intestinal microbiota. He currently serves as Professor and Associate Director for Research at the university's Gastrointestinal Laboratory.

ileum for enterohepatic circulation (3), with the remainder reaching the colon where they are converted by bacteria (mainly *Clostridium hiranonis* in dogs and cats) to secondary BAs (4). This conversion has important health consequences, as secondary BAs, in the correct quantity, have beneficial effects. They act as signaling agonists for various receptors across multiple organs, inducing anti-inflammatory and glucose-lowering effects and suppression of enteropathogens (5).

Assessment of the microbiome

There are various options for assessing a dog's microbiome, but some are more effective than others.

Bacterial culture

While still used by many veterinarians for the diagnosis of dysbiosis, bacterial culture of feces is not useful for microbiome assessment, as the majority of intestinal bacteria are strict anaerobes requiring special growth media **(Figure 1)**. Consequently, only a small percentage of bacterial species can be cultured by diagnostic laboratories. In a recent study, different fecal aliquots from healthy dogs and dogs with chronic diarrhea were submitted to three veterinary reference laboratories for the evaluation of dysbiosis (6). There was no agreement in the culture results between laboratories, and dysbiosis was actually more frequently reported amongst the healthy dog group. This study demonstrates that bacterial culture should not be used for microbiota assessment in dogs with chronic diarrhea, except for specific pathogens such as *Salmonella* spp.

Molecular sequencing of 16S rRNA genes

Molecular techniques based on sequencing of 16S rRNA genes provides comprehensive information on the microbial composition of a fecal sample, and are used in research settings. Various companies offer sequencing for microbiome assessment on a commercial basis for individual animals, but there are currently no standardized methods (*e.g.*, DNA extraction, PCR primers used)

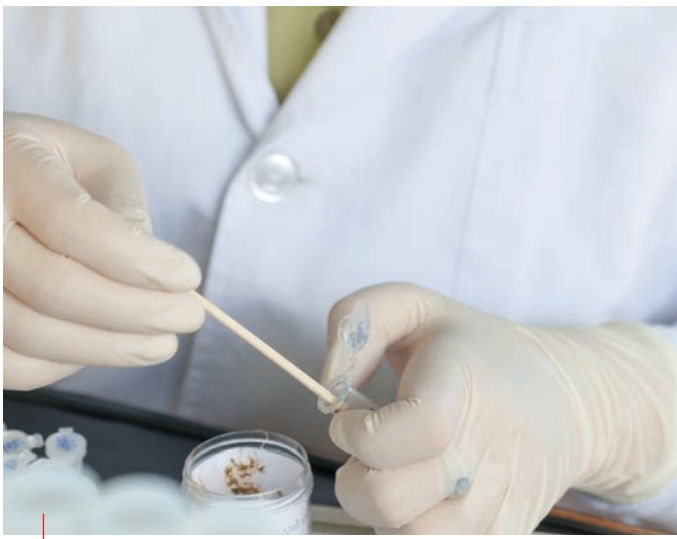


Figure 1. Many veterinarians will routinely submit a fecal sample for culture and sensitivity analysis when investigating a dog with diarrhea, but this is not useful for microbiome assessment, as the majority of intestinal bacteria are strict anaerobes requiring special growth media, and the test will provide only limited (and sometimes misleading) information.

between these laboratories. Because no reference intervals are defined for animals, and because each company has a different report, result interpretation is difficult. Furthermore, inter-assay variation is common, and no analytical validation data have been reported for these assays, therefore sequencing-based microbiome assessment is not currently recommended for individual patients.

Canine microbiota dysbiosis index (DI)

The dysbiosis index (DI) is a quantitative PCR based test that is currently commercially available in North America and Europe and is now used in many clinical studies (4,7) as it is the only validated assay to assess canine microbiome dysbiosis[1]. The DI measures the levels of seven intestinal bacteria **(Box 2)** which are commonly altered in dogs with chronic enteropathies (CE) or after broad-spectrum antibiotic use (*e.g.*, tylosin, metronidazole) (8,9).

[1] https://tx.ag/DysbiosisGI

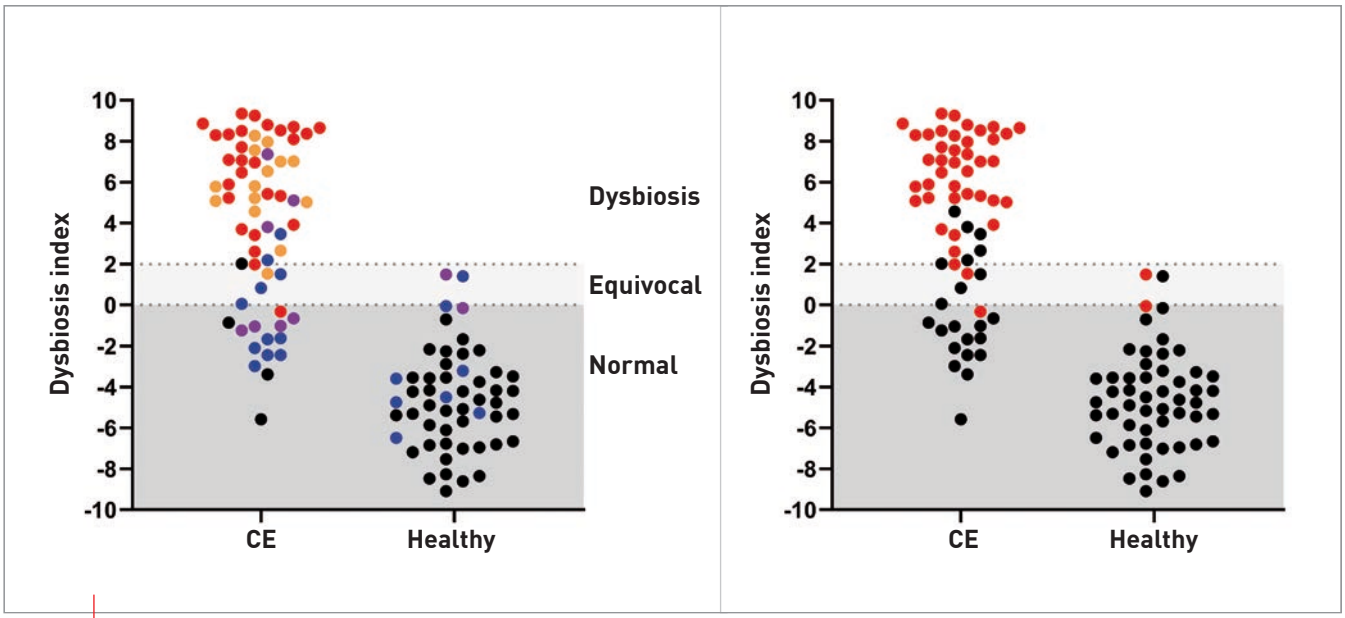


Figure 2. The graph shows the difference in Dysbiosis Index (DI) in a cohort of dogs with CE when compared to normal dogs (7). The higher the DI, the more abnormal the microbiome is; a DI > 2 has high specificity for an abnormal microbiome, whilst values between 0 and 2 are equivocal. A higher DI is generally characterized by less diversity and more bacterial taxa outside the reference intervals; here the different colors indicate the number of bacterial taxa outside the reference interval (black 0, blue 1, purple 2, orange 3, red > 3). However, some dogs have all taxa within the reference intervals, but the DI is increased due to abnormal shifts within the reference intervals (black dots) **(left)**.

Data from the same two cohorts illustrate how the levels of *C. hiranonis* have a major effect on the microbiome. Samples in red denote decreased levels of the bacterium and therefore reduced conversion of primary to secondary intestinal bile acids, leading to an abnormal shift in the microbiome **(right)**.

Box 2. The seven bacterial groups included in the canine Dysbiosis Index and how their levels alter in dysbiosis.

Bacterial group	Change in dysbiosis
Faecalibacterium spp.	↓
Turicibacter spp.	↓
Blautia spp.	↓
Fusobacterium spp.	↓
C. hiranonis	↓
Streptococcus spp.	↑
E. coli	↑

Box 3. The sensitivity and specificity of the Dysbiosis Index (DI) for chronic enteropathies; a DI between 0-2 represents a moderate shift in the microbiota, whilst above 2 indicates a major shift.

Dysbiosis index	Sensitivity	CI (95%)	Specificity	CI (95%)
-1	0.82	0.73-0.88	0.91	0.84-0.96
0	0.74	0.65-0.82	0.95	0.89-0.98
2	0.63	0.53-0.72	1	0.96-1.00

The assay provides reference intervals for these bacterial groups and combines data into a single number that expresses the extent of the dysbiosis **(Figure 2)**; a DI between 0 and 2 represents a moderate shift in the microbiota, whilst a DI > 2 indicates a major shift. The sensitivity and specificity of the method is shown in **Box 3**.

The DI also predicts, by assessing the concentration of *C. hiranonis*, the ability of the intestinal microbiota to convert primary BAs to secondary BAs (4). Normal amounts of secondary bile acids are antimicrobial and suppress potential enteropathogens such as *C. difficile*, *C. perfringens*, and *E. coli* (10), so reduced levels of *C. hiranonis* and decreased conversion of bile acids is strongly associated with intestinal dysbiosis and overgrowth of enteropathogens in dogs **(Figure 2)** (4,7,8,11). Identification of some or all of these enteropathogens in a dog with diarrhea will suggest overgrowth due to an underlying dysbiosis secondary to chronic enteropathy, rather than a primary infection. Up to 60% of dogs with a chronic enteropathy (CE) have decreased levels of *C. hiranonis*, and therefore decreased secondary BA (12).

The microbiome in disease

Table 1 summarizes the various ways in which intestinal bacteria can contribute to disease, although the underlying pathologies will vary between individual patients depending on the location and severity of intestinal damage.

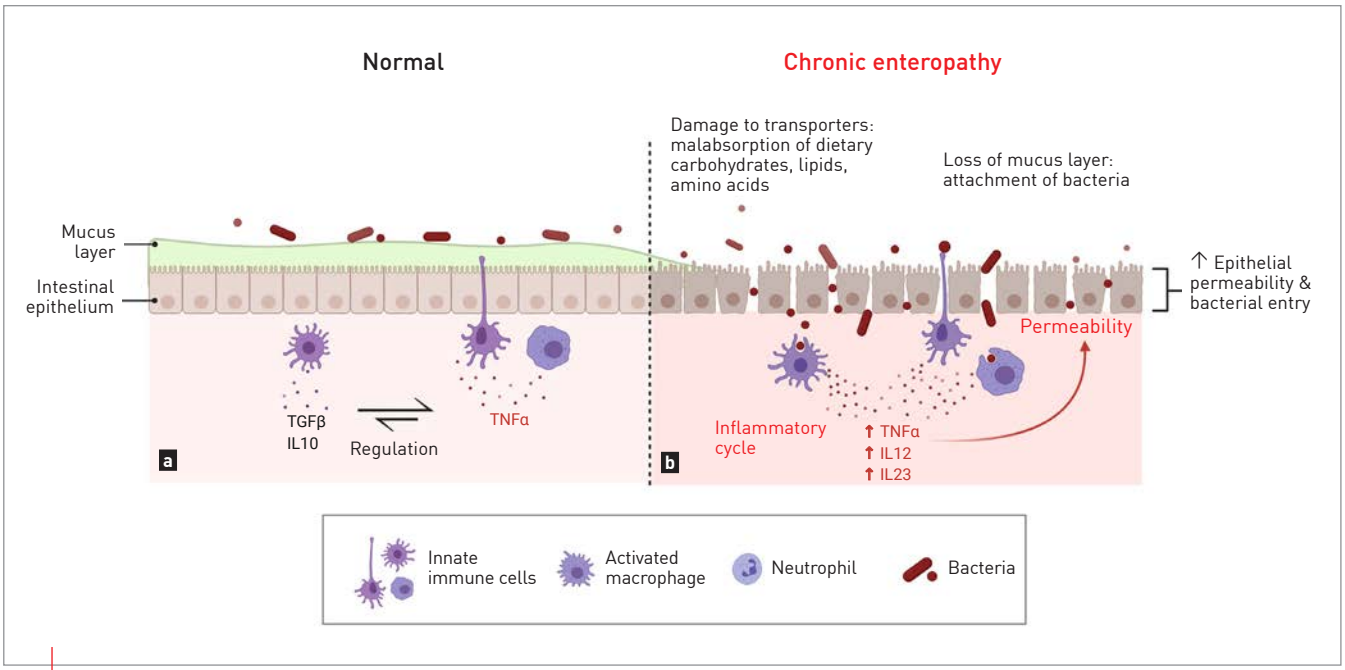


Figure 3. The intestinal tract in health and disease. A healthy intestine **(a)** is characterized by a balanced microbiome, with a mucus layer separating luminal bacteria from the epithelial cells, a tight epithelial cell barrier, and a balanced immune system. In a chronic enteropathy **(b)** various changes may occur, all of which may potentially contribute to clinical signs, therapy should therefore be multi-modal. The changes include:

- the microbiome becoming dysbiotic;
- loss of mucus, allowing luminal bacteria to attach to epithelial cells, stimulating pro-inflammatory cytokines;
- a broken epithelial barrier, leading to translocation of food and bacterial antigens, which also activates the immune system;
- loss of transporters in the brush border, leading to malabsorption of dietary compounds, which can allow bacterial overgrowth.

The microbiota is in contact with the mucus layer of the gut, the immune system, and luminal substrates, and changes in one or more of these will affect microbiota composition, so dysbiosis is often an early marker of an abnormal gut environment in disease **(Figure 3)**.

A dysbiosis restricted mainly to the gut lumen is often present in patients with exocrine pancreatic insufficiency (EPI) (13), after broad-spectrum antibiotic treatment (8,9), or in younger animals due to an immature immune system. Chronic enteropathies are accompanied by inflammation and destruction of the mucus layer and mucosal structure, leading to more oxygen at the mucosal surface, increased numbers of aerobic bacteria (*E. coli)*, and a decrease in normal anaerobic flora. The loss of mucosal architecture that develops with CE leads to a lack of transporters for carbohydrates, amino acids, fatty acids and bile acids, resulting in malabsorption of these compounds (14). Increased amounts of these substrates in the GI lumen can directly lead to osmotic or secretory diarrhea, as well as bacterial overgrowth.

Table 1. Mechanisms by which bacteria contribute to GI disease.

Major types of dysbiosis	Possible consequences
Abnormal substrate (*e.g.*, undigested nutrients, medications) in gut lumen	Increase in bacterial metabolites causing diarrhea
Poor microbiota function due to loss of commensal bacteria (*e.g., C. hiranonis*)	Reduced conversion of primary to secondary bile acids leads to overgrowth of enteropathogens Lack of anti-inflammatory metabolites
Increase in total bacterial load in small intestine	Increased microbial metabolites, causing diarrhea Increased inflammatory immune response
Increased mucosa-adherent bacteria	Increased inflammatory immune response

Due to disruption of the mucus layer covering the epithelium, dogs with CE have often increased number of mucosa-adherent bacteria (15). This is linked to a reduction in *C. hiranonis* and therefore abnormal bile acid conversion, allowing a secondary overgrowth of *C. difficile* and *C. perfringens* which can lead to increased pro-inflammatory host responses.

A diagnostic approach to dysbiosis

Since dysbiosis commonly develops secondary to a changed gut environment with intestinal disease and/or altered environmental factors, it should be assessed along with a patient's medication history and the clinical presentation. Interpretation of the DI result should be done alongside the levels of the individual bacterial taxa, and especially *C. hiranonis*, as a decrease in the latter is a major contributor

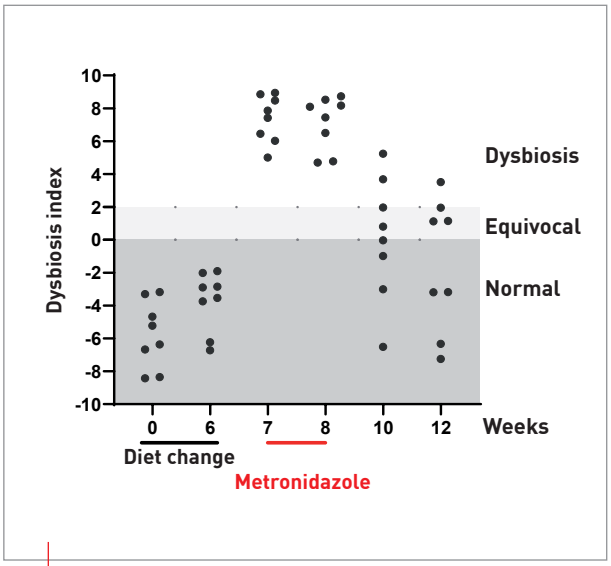


Figure 4. The effect of dietary transition and metronidazole on the intestinal microbiome in healthy dogs (from (8)). A hydrolyzed protein diet (fed between weeks 0-6) has only minor effects on the intestinal microbiota, whereas metronidazole (given on weeks 7 and 8) induces significant dysbiosis, with some dogs retaining an abnormal microbiome composition after the drug is withdrawn (weeks 10-12). Dietary modulation is therefore the preferred first-line treatment in intestinal disease, as it has no negative impact on the gut microbiota, especially when compared to metronidazole.

to an abnormal microbiome. A DI above 2 indicates dysbiosis with high specificity, while a DI in the equivocal range indicates a minor shift in the fecal microbiome. Some dogs with CE can have a DI < 0 but with some bacterial taxa outside the reference intervals, and this represents a minor form of dysbiosis. In general, an abnormal DI suggests underlying intestinal disease, and a workup for CE is therefore indicated.

Note that some drugs can influence the DI. For example, omeprazole can lead to a transient increase, but with normal levels of *C. hiranonis*, and the DI normalizes 1-2 weeks after therapy finishes. Broad-spectrum antibiotics (*e.g.*, metronidazole and tylosin) can induce severe fecal dysbiosis **(Figure 4)**, but again the microbiota typically normalizes within 2-4 weeks after administration ends in most dogs, although some individuals may have a persistent dysbiosis with lack of *C. hiranonis* for several months (8,11).

Compositional microbiota changes in the small intestine will often lead to detectable changes in the fecal microbiome as assessed by the DI. However, in some patients, an increased number of bacteria in the small intestine may cause disease. Small intestinal dysbiosis is suggestive if serum concentrations of folate are increased and serum cobalamin are decreased on a GI panel, but note that both markers have low sensitivity and specificity.

Therapy for dysbiosis

Dysbiosis is often just one component of intestinal disease, and multi-modal therapy addressing the underlying cause is usually required. In some cases, such as in animals with EPI, treatment with pancreatic enzyme supplementation leads to improvement in clinical signs, and often the intestinal microbiome will normalize after several weeks (13), but in dogs with CE there are no markers that predict which treatment is best for an individual patient, so stepwise treatment trials are often necessary (16). Therapies for dysbiosis include dietary modulation, pre- and probiotics,

Table 2. Treatment options for dysbiosis.

Treatment	Likely mechanism	Potential side effects
• Dietary changes	• high digestibility leads to less residual substrate available for bacterial overgrowth • elimination diets (with either novel or hydrolyzed ingredients) remove dietary antigens if underlying disease is immune-mediated	• none (if no food sensitivity present)
• Prebiotics/fibers	• growth of beneficial bacteria • prebiotic converted to SCFA • fibers bind harmful bacterial metabolites	• soluble/fermentable fibers can initially cause flatulence and diarrhea
• Probiotics	• can improve barrier function immunomodulatory	• side effects are rare, but often not clear which patient would benefit best from which strain
• Antibiotics	• reduction in total bacterial load and/or mucosa-adherent bacteria	• long-term changes in microbiota • regrowth of bacterial load when drug withdrawn • increased antimicrobial resistance
• Fecal microbiota transplantation (FMT)	• alters luminal microbiota	• efficacy depends on underlying disease, but side effects are rare • minor effect on mucosa-adherent bacteria • recurrence of dysbiosis when concurrent inflammation still present

antimicrobials, and fecal microbiota transplantation (FMT), with each approach addressing a different mechanism **(Table 2)**; a combination of treatments will often offer the best success.

Dietary changes should always be the first treatment option in stable patients. Various studies have shown that between 50-70% of dogs with CE are food-responsive (16), and highly digestible diets containing hydrolyzed or novel proteins are most commonly used. Most of these diets are hypoallergenic and reduce undigested nutrients in the GI lumen, decreasing the potential for bacterial overgrowth. In most cases of food-responsive enteropathy, the dietary change alone is sufficient to achieve clinical remission, leading to gradual improvement of intestinal inflammation and dysbiosis over several months (10,17).

Probiotics can be administered alone in mild cases, or together with dietary modulation. Because the number of bacteria administered in any probiotic is small when compared to the existing gut microbiota, they have a minor direct impact on microbiota composition. However, they attach to the mucosa and can exert beneficial effects, including shortening the duration of acute diarrhea and reducing antibiotic-associated GI side effects such as vomiting or diarrhea (18). High-potency multi-strain probiotics have been shown to reduce *C. perfringens* in dogs with acute hemorrhagic diarrhea (19) and strengthen the intestinal barrier in dogs with CE (20). However, because many commercial products lack proper quality controls, it is important to choose a preparation that has shown efficacy in a published clinical study.

Prebiotics are indigestible carbohydrates that promote growth of beneficial micro-organisms, and can be divided into soluble/insoluble and fermentable/non-fermentable fibers. Fermentable prebiotics are converted by colonic bacteria to SCFA. Most commercial GI diets contain prebiotics, but for some diseases (*e.g.*, colitis) high-fiber diets can be beneficial. Addition of psyllium husk, a soluble fiber, to the diet at 0.5-1 g/ kg bodyweight daily can improve stool quality in animals with large bowel disease. The product should be introduced at lower doses and titrated up to achieve the desired stool consistency.

Antibiotics such as tylosin or metronidazole have been traditionally recommended for treatment of CE, but their first-line use is now debated (16). Although they can lead to an improvement in clinical signs, presumably due to a reduction in bacterial load, patients will often relapse after treatment as the bacteria regrow, as antibiotics rarely resolve the underlying disease process (15,21,22). Commonly

"Dietary modulation is the preferred first-line treatment in intestinal disease, as it has no negative impact on the gut microbiota."

Jan S. Suchodolski

© Ewan McNeill

Figure 5. Preparation of a sample for fecal microbiota transplantation, with feces from a donor dog being blended with saline.

© Ewan McNeill

Figure 6. The FMT is achieved by administering the blended fecal material to the recipient dog as an enema, using a catheter and syringe.

used options include metronidazole (10-15 mg/kg q12h) and tylosin (25 mg/kg q12h) for 4-6 weeks, but as noted above, both drugs have been shown to induce large intestinal dysbiosis that can sometimes last for months (8,9,11). Studies report that metronidazole has promoted lasting dysbiosis in dogs with acute diarrhea (11), while amoxicillin-clavulanic acid can encourage an increase in resistant *E. coli* (23). Antibiotics are generally not recommended as first-line treatment in CE for a variety of reasons – only 10-16% of CE dogs are antibiotic-responsive, most cases relapse after treatment is withdrawn, and the drugs have negative effects on the microbiome. Antibiosis should, however, be considered after failed dietary and anti-inflammatory trials, or for patients with signs of systemic inflammation (16) and invasion and persistence of bacteria in the intestinal mucosa (*e. g.*, *E. Coli* associated granulomatous colitis). A small subset of dogs with CE may respond to no other treatment, in which case long-term administration may be necessary, with the dosage tapered to the lowest effective point.

Fecal microbiota transplantation (FMT) can help restore the normal microbiota and improve clinical signs (11) in some cases of dysbiosis. The technique involves the transfer of stool from a healthy donor into the gut of a recipient via oral capsules, endoscopy or enema **(Figures 5 and 6)**. In humans, FMT has a high success rate (> 90%) with infectious and recurrent *C. difficile* infection, but has more limited success for inflammatory bowel disease due to the chronic underlying intestinal inflammation.

FMT in animals is still an emerging therapy. A simple protocol is shown in **Box 4**, although to date only a few case series have been reported, with success apparently dependent on the

Box 4. FMT protocol via enema (based on 24).

The donor should be healthy, with no history of gastrointestinal disease or recent antibiotic exposure, and should have no signs of systemic disease. The donor feces should be screened for parasites and enteropathogens, and be pre-evaluated using the DI (because some clinically healthy dogs lack *C. hiranonis*, which is necessary for proper BA conversion).

Storage – stool can be fresh or stored at 4° C for up to one week in plastic bags. When feces need to be frozen for longer, mixing the stool with glycerol before freezing preserves the bacteria (10 grams of stool with 35 mLs of saline and 5 mLs of glycerol, frozen in 50 mL aliquots.

Materials needed: 0.9% NaCl, 12 or 14 FG red rubber catheter, 60 mL catheter tip syringes, blender, donor stool, non-bacteriostatic lubricant.

1. Calculate amount of stool needed, approximately 5 grams per kg body weight.
2. Add approximately 60 mL of 0.9% NaCl to a blender, then add fresh or frozen stool and blend on high speed until the stool is liquefied and no large pieces are seen. For very large dogs a larger volume of saline may be needed to obtain sufficiently liquefied stool.
3. Draw up the blended material into the syringe and attach the rubber catheter. Depress the syringe plunger until the fecal material appears at the catheter tip – this ensures no air will be introduced into the recipient's colon.
4. Feed the catheter into the colon fully, then administer the enema. The recipient dog does not need to be sedated.
5. After the transplant, if possible the recipient dog should be fasted for 4-6 hours and its activity restricted, to lessen the chances of a premature bowel movement.

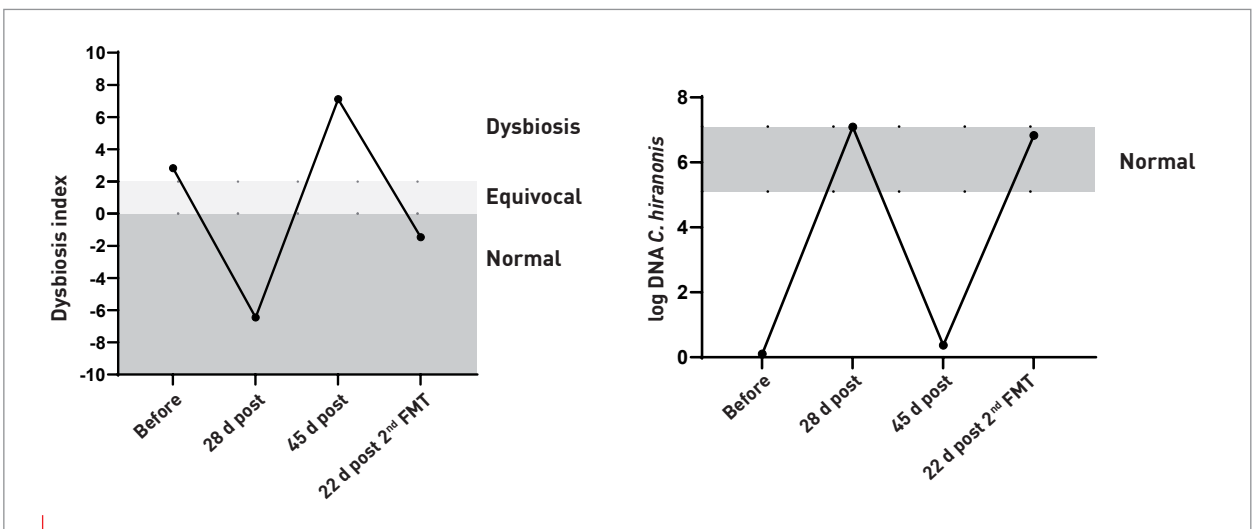


Figure 7. The effect of fecal microbiota transplantation (FMT) on the intestinal microbiome can be seen in these two graphs which show the dysbiosis index **(a)** and *C. hiranonis* levels **(b)** in a dog with CE which was unresponsive to other standard treatments. After FMT, the DI and the abundance of the bile acid-converting bacterium *C. hiranonis* normalized, and stool quality improved within 2 days. Approximately 45 days after the FMT, stool quality worsened again and the DI was increased, so a second FMT was performed, resulting in better stool quality. Because the underlying structural damage remains in many dogs with CE, dysbiosis often returns and requires repeated procedures.

underlying disease (24). The technique helps to restore bile acid metabolism by promoting levels of *C. hiranonis* **(Figure 7)**, so it may be useful in dogs with abnormal BA conversion with associated overgrowth of enteropathogens such as *C. difficile* or *C. perfringens* and/or animals with antibiotic-induced dysbiosis and minor underlying damage of the intestinal mucosa. It has also been shown to improve fecal scores in cases of acute diarrhea and when used as an adjunct to standard antimicrobial therapy in puppies with parvovirus infection, and for young dogs with chronic diarrhea due to confirmed *C. difficile* infection (25).

In dogs with CE, dysbiosis is often a secondary effect of the intestinal inflammation and structural damage, and recurrence of dysbiosis and clinical signs will occur if the underlying pathology is not eradicated. FMT therefore has a very variable success rate in CE, and anecdotal reports suggest many dogs with CE will have improved fecal scores within 2-3 days of treatment, but will relapse and develop recurrent diarrhea a few weeks later. Therefore in these patients appropriate dietary and anti-inflammatory treatment of the underlying disease process is required (see above), and FMT can be considered as adjunctive treatment for patients that show a suboptimal response (*e.g.*, continuing soft stools) despite standard therapies.

Disclosure: The author is an employee of the Texas A&M Gastrointestinal Laboratory that offers microbiome testing on a commercial basis.

CONCLUSION

The intestinal microbiome plays a crucial role in host health and many animals with gastrointestinal disease will develop dysbiosis, resulting in abnormal microbial function which can contribute to clinical signs. The dysbiosis index is a useful diagnostic tool for many cases, but as there can be various underlying causes, a multi-modal and often long-term therapeutic approach is necessary to improve microbiota composition.

REFERENCES

1. Ziese AL, Suchodolski JS. Impact of changes in gastrointestinal microbiota in canine and feline digestive diseases. *Vet. Clin. North. Am. Small. Anim. Pract.* 2021;51(1):155-169.
2. Whitfield-Cargile CM, Cohen ND, Chapkin RS, *et al*. The microbiota-derived metabolite indole decreases mucosal inflammation and injury in a murine model of NSAID enteropathy. *Gut. Microbes.* 2016;7(3):246-261.
3. Giaretta PR, Suchodolski JS, Blick AK, *et al*. Distribution of bile acid receptor TGR5 in the gastrointestinal tract of dogs. *Histol. Histopathol.* 2019;34(1):69-79.
4. Li Q, Larouche-Lebel E, Loughran KA, *et al*. Gut dysbiosis and its associations with gut microbiota-derived metabolites in dogs with myxomatous mitral valve disease. *MSystems.* 2021;in press.
5. Pavlidis P, Powell N, Vincent RP, *et al*. Systematic review: bile acids and intestinal inflammation-luminal aggressors or regulators of mucosal defence? *Aliment. Pharmacol. Ther.* 2015;42(7):802-817.
6. Werner M, Suchodolski JS, Lidbury JA, *et al*. Diagnostic value of fecal cultures in dogs with chronic diarrhea. *J. Vet. Intern. Med.* 2021;35(1):199-208.
7. Al Shawaqfeh MK, Wajid B, Minamoto Y, *et al*. A dysbiosis index to assess microbial changes in fecal samples of dogs with chronic inflammatory enteropathy. *FEMS Microbiol. Ecol.* 2017;93(11): DOI: 10.1093/femsec/fix136
8. Pilla R, Gaschen FP, Barr JW, *et al*. Effects of metronidazole on the fecal microbiome and metabolome in healthy dogs. *J. Vet. Intern. Med.* 2020;34(5):1853-1866.
9. Manchester AC, Webb CB, Blake AB, *et al*. Long-term impact of tylosin on fecal microbiota and fecal bile acids of healthy dogs. *J. Vet. Intern. Med.* 2019;33(6):2605-2617.
10. Wang S, Martins R, Sullivan MC, *et al*. Diet-induced remission in chronic enteropathy is associated with altered microbial community structure and synthesis of secondary bile acids. *Microbiome* 2019;7(1):126.
11. Chaitman J, Ziese AL, Pilla R, *et al*. Fecal microbial and metabolic profiles in dogs with acute diarrhea receiving either fecal microbiota transplantation or oral metronidazole. *Front. Vet. Sci.* 2020;7:192.
12. Blake AB, Guard BC, Honneffer JB, *et al*. Altered microbiota, fecal lactate, and fecal bile acids in dogs with gastrointestinal disease. *PLOS One* 2019;14(10):e0224454.
13. Isaiah A, Parambeth JC, Steiner JM, *et al*. The fecal microbiome of dogs with exocrine pancreatic insufficiency. *Anaerobe* 2017;45:50-58.
14. Giaretta PR, Rech RR, Guard BC, *et al*. Comparison of intestinal expression of the apical sodium-dependent bile acid transporter between dogs with and without chronic inflammatory enteropathy. *J. Vet. Intern. Med.* 2018;32(6):1918-1926.
15. Giaretta PR, Suchodolski JS, Jergens AE, *et al*. Bacterial biogeography of the colon in dogs with chronic inflammatory enteropathy. *Vet. Pathol.* 2020;57(2):258-265.
16. Procoli F. Inflammatory bowel disease, food-responsive, antibiotic-responsive diarrhoea, protein losing enteropathy. *Advance Small Anim. Care* 2020;1:127-141.
17. Bresciani F, Minamoto Y, Suchodolski JS, *et al*. Effect of an extruded animal protein-free diet on fecal microbiota of dogs with food-responsive enteropathy. *J. Vet. Intern. Med*. 2018;32(6):1903-1910.
18. Torres-Henderson C, Suchodolski J, Lappin MR. Effect of *Enterococcus faecium* strain SF68 on gastrointestinal signs and fecal microbiome in cats administered amoxicillin-clavulanate. *Top. Companion Anim. Med.* 2017;32(3):104-108.
19. Ziese AL, Suchodolski JS, Hartmann K, *et al*. Effect of probiotic treatment on the clinical course, intestinal microbiome, and toxigenic *Clostridium perfringens* in dogs with acute hemorrhagic diarrhea. *PLOS One* 2018;13(9):e0204691.
20. White R, Atherly T, Guard B, *et al*. Randomized, controlled trial evaluating the effect of multi-strain probiotic on the mucosal microbiota in canine idiopathic inflammatory bowel disease. *Gut Microbe*s 2017;8(5):451-466.
21. Westermarck E, Skrzypczak T, Harmoinen J, *et al*. Tylosin-responsive chronic diarrhea in dogs. *J. Vet. Intern. Med*. 2005;19(2):177-186.
22. Westermarck E, Myllys V, Aho M. Effect of treatment on the jejunal and colonic bacterial flora of dogs with exocrine pancreatic insufficiency. *Pancreas* 1993;8:559-562.
23. Werner M, Suchodolski JS, Straubinger RK, *et al*. Effect of amoxicillin-clavulanic acid on clinical scores, intestinal microbiome, and amoxicillin-resistant *Escherichia coli* in dogs with uncomplicated acute diarrhea. *J. Vet. Intern. Med*. 2020;34(3):1166-1176.
24. Chaitman J, Gaschen F. Fecal microbiota transplantation in dogs. *Vet. Clin. North. Am. Small Anim. Pract*. 2021;51(1):219-233.
25. Pereira GQ, Gomes LA, Santos IS, *et al*. Fecal microbiota transplantation in puppies with canine parvovirus infection. *J. Vet. Intern. Med*. 2018;32(2):707-711.

MYTHS IN CAT NUTRITION

A plethora of myths exist as to what a cat should or should not be fed to ensure a healthy lifestyle; this paper aims to put some of the inaccuracies to rest!

KEY POINTS

1. The best way to support dental hygiene in cats is to combine active and passive methods of oral healthcare; a typical dry maintenance petfood in itself offers few or no benefits.
2. Pork is an appropriate source of both protein and fat for the feline diet, and – provided it is cooked – there are no contraindications for including pork in a cat's food.
3. Avoid giving cats an excessive number of snacks, and especially table scraps; treats should not constitute more than 10% of an animal's daily ration.
4. *Ad lib* feeding of cats is not recommended; instead, an owner should measure out portions and offer several small meals at regular intervals throughout the day.

Introduction

Cats are one of the most common creatures to be found in legends and folklore, often being portrayed as mysterious and contrary in nature. It is perhaps therefore not surprising that there are many confusing tales, half-truths and downright fallacies concerning their dietary requirements in real life. This short paper separates some of the myths from reality when it comes to feeding one of our favorite pets.

MYTH – dry food cleans the teeth

It is commonly believed that feeding dry food will reduce the amount of plaque and tartar on a cat's teeth, and that it offers significant benefits in terms of oral hygiene when compared to feeding wet food. Certainly, chewing on hard kibble or croquettes seems to cleanse the teeth; as the animal chews on dry food, the pellets break and crumble, causing a little mechanical cleaning. In addition, dry kibble leaves less food residue in the mouth for oral bacteria to feed on, and plaque builds up more slowly. Nevertheless, many animals given dry food will still have an abundant build-up of plaque and tartar and suffer from periodontal disease (1,2), so the evidence as to the benefits of feeding dry food can be confusing.

One study has shown that companion animals fed soft foods had more gingivitis and plaque when compared to those that were offered harder foodstuffs (3). Another study that contrasted homemade diets to commercial (wet and/or dry) foods found that feeding cats a home-cooked preparation increased the likelihood of oral health problems, and reported that offering commercial diets (as compared to homemade recipes) was of significant benefit when dry food constituted at least part of the ration (4). A further study also demonstrated a lessening in the levels of periodontal disease and tartar, along with a reduction in lymphadenopathy, in cats fed a dry diet compared to those given soft meals (either home-prepared or commercial) (5). Nevertheless, other case studies have found that moist foods have a similar effect on the build-up of plaque and tartar as an average dry diet (1,6). Importantly, it should be noted that dry commercial foods typically break down at the edges of the incisor teeth, which is of little or no use for improving oral hygiene, because the plaque and tartar deposits at the gingival borders and in the subgingival areas (*i.e.*, the most important regions for dental health) remain unaffected (7,8).

Given that two thirds of cats over the age of two show signs of periodontal disease (5), it is not surprising that some commercial dry foods for adult cats have been developed which demonstrate an improved oral cleaning capacity when compared to typical dry maintenance foods. These "dental diets" have a consistency that maximizes contact with the teeth, with kibbles that are shaped, sized and textured to help control plaque and tartar (9). Many dental diets also contain ingredients that help prevent plaque and tartar accumulation, and certainly VOHC®[1]-approved products do have a proven benefit for oral health.

[1] Veterinary Oral Health Council

Karolina Hołda

PhD, Warsaw, Poland

Dr. Hołda graduated from the Warsaw University of Life Sciences, Poland and went on to undertake further study at the Faculty of Veterinary Medicine in Ghent. A specialist in feline and canine nutrition and the owner of a nutritional advisory service (karolinaholda.com), she also runs online courses on the proper nutrition of dogs and cats, and has written over 70 scientific and popular science articles for both veterinary and zoological journals.

Dental diets, as well as supplements and snacks for tooth cleaning, are a form of passive oral hygiene, as opposed to methods that deliver active oral hygiene, whereby the owner removes plaque by brushing the teeth and/or applying gels to the oral cavity. Passive methods alone will not keep the gingiva clinically healthy, as animals do not use all their teeth when chewing a dental product – and even for the teeth that do come into contact with the product, the entire surface of each tooth is not involved with the chewing action, so many areas within the mouth are neglected. In fact, the passive action of dental chews is most effective on the premolars, because the animal mainly uses these teeth to bite **(Figure 1)**, whereas active methods are more effective on the incisors and canines, because the owner finds it easier to access the front teeth (10).

The best method of maintaining oral hygiene is still to brush the teeth daily, which reduces the amount of bacterial plaque, although it can be a major challenge for many owners **(Figure 2)**. While the notion that dry food helps cleans teeth is appealing, it appears that most dry maintenance foods by themselves do not significantly reduce the risk of periodontal disease. Certain dental preparations may have some benefits, but they must be products that also clean the subgingival areas, and when recommending nutritional dental products to clients it is best to choose those that meet the high requirements of the VOHC® (11). Ultimately, the best way to keep a cat's mouth healthy is to combine active and passive methods, with regular veterinary dental care, which starts with excellent communication between the clinician and the cat's owner!

© Shutterstock

Figure 1. A cat with a dental chew will mainly use its premolar teeth when chewing.

© Philippe Hennet

Figure 2. Daily tooth brushing is an active method of maintaining good oral hygiene.

MYTH – cats should not eat pork

Pork is a good source of protein and essential amino acids, yet there is a common myth that it should not be fed to cats. Certainly, pork has a high-fat content, so this must be taken into account if using it as a basis for a diet. It is a popular constituent in both dry and wet commercial foods – as meat, skin, fat, offal or meal – as it serves as a concentrated protein source. The poor reputation of pork is mainly linked to a virus of the *Herpesviridae* family which causes Aujeszky's disease (also known as pseudorabies). This is an infectious disease found in both farmed and wild animals, mainly pigs, but cats (and dogs) can contract the disease by eating raw infected pork (12). Affected animals develop signs that are mainly linked to the nervous system: paresis, paralysis, and severe itching that leads to self-harm. Whilst humans are not susceptible to infection with this virus, the disease is unfortunately fatal in cats.

Control of Aujeszky's disease is generally led by a country's national veterinary service, with ongoing disease-monitoring in pig herds performed by taking random blood samples for testing. The number of reported outbreaks is currently steadily declining, and many European regions have been

© Shutterstock

Figure 3. Wild boar are common in certain parts of Western Europe and can act as a reservoir for Aujeszky's virus.

officially declared free from the disease. However, according to the European Advisory Board on Cat Diseases (13), the incidence of Aujeszky's disease infection in wild boar may be high in Western Europe **(Figure 3)**, and the virus is still sporadically found in hunting dogs, although this in itself does not represent an increased risk of the virus contaminating cat food.

Importantly, it is advisable to cook all pork before giving it to pets, as this destroys the virus and renders the meat safe. However, the author advises against feeding cats with pork products intended for humans; this is because all kinds of ham, sausages and cold cuts, although manufactured from cooked pork, usually have a high-fat content, and they also contain preservatives such as sodium nitrite and phosphates, which are potentially harmful to animals. In short, highly processed animal products from the human table do not constitute a healthy snack for a pet cat.

MYTH – serving human food is not harmful to cats

Most owners know that some foods from the human table can be toxic to dogs and cats – for example, onions and garlic (which are often used in cooking sauces), or raisins and chocolate which – although commonly eaten by humans as a sweet snack – can be toxic and even fatal for pets. Feeding table scraps is also not recommended for other reasons. Human food is often highly seasoned (*e.g.*, with salt, pepper, or spices), and ready-to-eat, processed foods (as noted above) also contain constituents that may adversely affect animal health. In addition, human food is usually fat-rich, and therefore high in calories, and unbalanced in terms of a cat's nutritional needs. Food intended for humans certainly cannot and should not form the basis of an animal's diet. If an owner really wants to give their cat something "from their plate", the clinician should advise that the food does not contain any toxic ingredients and that all snacks are served in moderation. It is important to recommend that the number of treats between meals does not exceed 10% of the daily caloric dose, as otherwise this will quickly lead to obesity; in addition, an excess of snacks will result in an imbalance in the basic diet, which may cause deficiencies or excesses of individual nutrients.

The owner needs to be aware that human food is also not recommended for behavioral reasons. When cats learn that they can get something tasty from an owner, they will beg and/or refuse to eat their staple food in the hope of getting something "better". It is also worth counselling that any member of the household may inadvertently slip into bad behavior that encourages this trait – for example, a child or grandparent may secretly feed the cat every time they open the refrigerator door to get milk for coffee or tea **(Figure 4)**.

MYTH – "It's only one extra kilogram"

Obesity is an incredibly common phenomenon in pets for several reasons, but not least because owners compare a cat's excess weight to their own situation. If a person puts on a single extra kilogram, the physiological difference is minimal; for a cat that usually weighs 5 kg, if it gains "only" 1 kg, this is a 20% increase in body weight, which puts it in the obese category. In this situation it is worth explaining what the equivalent of excessive kilograms would be in a human, in order to make the owner aware of their distorted perception – for example, if a woman weighing around 55 kg was to gain an extra 20% (11 kg), she would definitely notice the difference in her bodyweight. A good solution that helps owner education is to hang a poster in the consult room of the veterinary clinic which shows the caloric content of snacks commonly fed to cats, converted to the equivalent caloric content for humans **(Figure 5)**. This can get

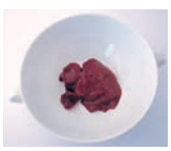
© Shutterstock

Figure 4. Household members may get into the regrettable habit of feeding the family cat every time they go to the refrigerator; this can lead to the cat becoming accustomed to being fed every time they hear the door being opened.

Figure 5.

Contribution of treats to daily energy intake

When an owner gives treats on top of the daily food ration, this drastically increases the overall daily energy intake*. This has the potential to cause weight gain in cats fed at maintenance levels, and slow/stop/reverse weight loss in cats on a weight management program. Below are few examples illustrating the potential effect of treats on food intake.

Quantity	Energy intake (in kcal)	Excess daily energy intake* *For a 4 kg cat/based on 200 kcal per day
 2 x 15 mL spoons of **low-fat cheese**	54	21%
 2 x 15 mL spoons of **yoghurt**	41	16%
 100 mL of **full fat milk**	58	23%
 25 g of **cream**	96	38%
 25 g of **tuna** (in brine)	28	11%
 43 g of **liver pâté**	154	61%
 25 g of **liver**	30	12%
 30 g of **ham rind**	255	101%
 60 g of **high-fat cream cheese**	62	25%

© Shutterstock

Figure 6. Daily use of a puzzle feeding toy will give mental stimulation to a cat as well as helping avoid excess calorie intake.

the message across that a small cube of cheese for a cat is comparable to a double hamburger for a human, and it cannot be over-emphasized that research indicates overfeeding (*i.e.*, *ad libitum* and snacking) and low physical activity are the main causes of obesity in cats (14).

MYTH – a cat should have constant access to food

Many cats will have dry food left in a bowl for the whole day (often without being properly measured out), with wet food being served in addition at certain times. This is usually due to the logistical impossibility of owners being able to serve their cat multiple small meals a day. Unfortunately, this method of feeding means that the owner is unable to properly control how much food the cat eats. Dry food is a product with a high energy concentration, so a small volume of kibble provides a lot of calories. Many owners hope that their pet will know "how much they need to eat" but in most cases cats eat more than they need, and excess energy leads to obesity (15). In addition, many cats will eat to excess because of boredom, as their owners do not provide them with sufficient environmental enrichment or active play.

Dry food does not resemble the consistency or calorific value of the type of food that cats eat in the wild, in that it provides the necessary energy and nutrients in a relatively small portion. This means that some cats will feel hungry even after eating an amount of food that matches their nutritional needs. The situation is different with wet food, which is typically four times less calorific, as it has a water content around 80%. This makes it much more difficult to overfeed a cat. In addition, there are also economic and practical considerations – wet food is more expensive per calorie, so owners are unlikely to offer more than their cat needs, and because wet food deteriorates quickly, owners are more likely to serve measured portions and at fixed times throughout the day.

In particular, owners of cats with a tendency towards excess weight and obesity should refrain from feeding at will, and the daily amount of food should be precisely measured on kitchen scales and then divided into several meals. Meals are best served regularly (*i.e.*, at fixed times during the day), but not only in a bowl – interactive feeding toys should be used on a daily basis as well, as these can contribute to a slower eating pattern and a reduced calorie intake, as well as offering the cat useful mental stimulation **(Figure 6)**.

CONCLUSION

It can be surprisingly common for the first opinion veterinarian to encounter clients who hold unexpected views on what their cats should or should not be fed – yet these owners will follow such myths in the belief that they are doing the best for their pet. Clear, unhurried communication between veterinarian and owner, coupled with good educational materials, can help disabuse these myths in many cases and ensure that we can promote the best possible nutrition for pet cats.

REFERENCES

1. Harvey CE, Shofer FS, Laster L. Correlation of diet, other chewing activities and periodontal disease in North American client-owned dogs. *J. Vet. Dent.* 1996;13:101-105.
2. Logan EI. Dietary influences on periodontal health in dogs and cats. *Vet. Clin. North. Am. Small. Anim. Pract.* 2006;36:1385-1401.
3. Watson AO. Diet and periodontal disease in dogs and cats. *Aust. Vet. J.* 1994;71(10):313-318.
4. Buckley C, Colyer A, Skrzywanek M, *et al*. The impact of home-prepared diets and home oral hygiene on oral health in cats and dogs. *Brit. J. Nutr*. 2011;106(0):S124-S127.
5. Gawor JP, Reiter AM, Jodkowska K, *et al*. Influence of diet on oral health in cats and dogs. *J. Nutr*. 2006;136:2021S-2023S.
6. Boyce EN, Logan EI. Oral health assessment in dogs: study design and results. *J. Vet. Dent*. 1994;11:64-74.
7. Westfelt E, Rylander H, Dahlen G, *et al*. The effect of supragingival plaque control on the progression of advanced periodontal disease. *J. Clin. Periodontol*. 1998;25:536-541.
8. Niemiec BA. Periodontal therapy. *Top. Companion. Anim. Med.* 2008;23:81-90.
9. Logan EI, Finney O, Herrerren JJ. Effects of a dental food on plaque accumulation and gingival health in dogs. *J. Vet. Dent*. 2002;19(1):15-18.
10. Niemiec B, Gawor J, Nemec A, *et al*. World Small Animal Veterinary Association Global Dental Guidelines. *J. Small Anim. Pract.* 2020;61(7):E120-E125.
11. Nemec A. The VOHC® seal – what does it mean? *Vet. Focus website (https://vetfocus.royalcanin.com/en/scientific/the-vohc-seal-what-does-it-mean)* 2021.
12. Thiry E, Addie D, Belák S, *et al*. Aujeszky's Disease / Pseudorabies in Cats: ABCD guidelines on prevention and management. *J. Feline Med. Surg.* 2013;15(7):555-556.
13. European Advisory Board on Cat Diseases Web site. Aujeszky's Disease – Pseudorabies in cats. Available at: http://www.abcd-catsvets.org/aujeszkys-disease-pseudorabies/ Accessed April 30, 2021
14. Kienzle E, Bergler R. Human-animal relationship of owners of normal and overweight cats. *J. Nutr.* 2006;136(7):1947S-1950S.
15. Rowe E, Browne W, Casey R, *et al*. Risk factors identified for owner-reported feline obesity at around one year of age: dry diet and indoor lifestyle. *Prev. Vet. Med*. 2015;121(3-4):273-281.

HOMEMADE DIETS – GOOD OR BAD?

Marjorie L. Chandler

DVM, MS, MANZCVS, Dip. ACVN, Dip. ACVIM, MRCVS, VetsNow, Glasgow, UK

Dr. Chandler gained her BS from California State University and her MS and DVM from Colorado State University (CSU). After several years in practice, she did residencies in small animal medicine and clinical nutrition at CSU and Massey University in New Zealand. As well as being a Diplomate of both ACVIM and ACVN, she is a member of ANZCVS, Chair of the FEDIAF Scientific Advisory Committee, a founding member of the European Veterinary Nutrition Educators Group, and Co-Chair of the WSAVA Global Nutrition Committee. She currently works as a clinical nutritionist and internal medicine specialist in UK referral hospitals.

Clinicians will often be faced with an owner who wants to feed their pet a homemade diet; this article looks at the potential problems and benefits such an approach can bring.

KEY POINTS

1. While less commonly fed than commercial diets, owners may feed homemade foods with good intentions and out of mistrust and misunderstanding of prepared foods.
2. The majority of recipes for homemade diets available on websites and textbooks do not meet a pet's nutritional requirements, and could lead to disease.
3. If raw diets are fed there is a potential risk of pathogenic bacterial contamination and fecal shedding of pathogens, putting both owners and members of the public at risk.
4. Understanding an owner's dietary choices and using clear, compassionate, communication and information are needed to ensure pets receive a complete and balanced diet.

Introduction

The term "homemade" when applied to petfood encompasses any non-commercial diet, and covers the entire spectrum from meat-only recipes to vegetarian or vegan diets, and includes both cooked and raw ingredients. Most owners who decide to prepare their pet's food wish to provide what they perceive as excellent nutrition for their cat or dog. Whilst they may seek the help of a board-certified veterinary nutritionist®, they may also – and are perhaps more likely to – source recipes from books, the internet, friends, and others who lack adequate training in small animal nutrition. This article offers an overview of such diets and discusses the risks and potential benefits that they may bring.

The prevalence of homemade diets

It can be difficult to determine an exact figure on how many pets receive a homemade diet. For example, a 2008 survey in the USA and Australia revealed that over 93.2% of dog owners and 98.9% of cat owners reported at least part of their pet's diet included commercial foodstuffs (1). However, 30.6% of dogs and 13.1% of cats received table scraps, leftovers or homemade foods, and bones or raw food was included in the pet's main meal in 16.2% of dogs and 9.6% of cats. Over 80% of people who included bones or raw foods in their pet's diet were residing in Australia. Less than 3% of owners fed exclusively home-prepared diets, but approximately 7% of dogs received at least half their diet as home-prepared foods.

Another study from the same year found that 95.5% of owners fed their cat a commercial diet, with only 2.7% meeting the criterion for offering a non-commercial diet, whilst 86.8% of dog owners were classified as "commercial" feeders and 10.0% as "non-commercial" feeders, with the remaining 3.2% not meeting the criterion for either category (2). A more recent international study reported that 79% of dogs and 90% of cats were fed conventional commercial foods, although only 13% of dogs and 32% of cats were fed such diets exclusively (3). Homemade food was offered to 64% of the dogs and 46% of the cats, with raw food being given to 66% of dogs and 53% of cats. As noted in the study above, feeding homemade and/or raw foods was again more prevalent in Australia, and there are obviously considerable geographical differences when it comes to pet feeding practices. In contrast to the above studies, 42% of dogs in Sri Lanka are reported to be fed home-cooked food, whilst only 18% are fed commercial diets, with the remaining 40% getting a mixture of both options. In addition, the same study showed that 49% of dogs receive milk as a separate meal as well as their normal diet, and 57% receive dietary supplements (4).

Overall, these and other surveys report the prevalence of owners feeding homemade diets to dogs appears to be about 7-10%, and to cats less than 4%; however, such results may not reflect the overall pet-owning population, due to selection bias. For example, in one of the above studies (3) the survey was completed by owners chosen via self-selection from canine and feline interest groups on social media. This type of sampling can skew results due to feedback from potentially biased segments of the population – for instance, owners who feed homemade diets may be more interested in doing a survey on feeding practices, or conversely could be less likely to report their feeding practices, so it is difficult to ascertain the real percentage of owners feeding homemade diets.

Why choose a homemade diet?

Pets are often considered to be family members, and the decision on what diet to offer may reflect an owner's cultural beliefs, ideologies and identity. So individuals may wish to feed according to their own eating philosophy, such as using only vegan, organic or natural foods. With the humanization of pets, offering a meal which looks more like the owner's dinner may have some appeal. Other reasons for feeding homemade foods include palatability (*i.e.*, choosing foods the pet prefers), mistrust and/or misunderstanding of petfood processing, a wish to exclude certain ingredients (such as grains, meat by-products or meat derivatives), or a desire to have "better" control over the pet's diet **(Figure 1)**. Owners have also reported motivations for feeding home-prepared or raw foods to include a desire to pamper their pet, concerns that commercial foods may be less wholesome or nutritious than desired, or to achieve a real or perceived medical benefit (2) **(Table 1)**.

© Shutterstock

Figure 1. Owners may choose a diet they perceive to be more palatable and enjoyable for their pet.

At least one study has suggested that there is an association between concerns of pet owners about commercial pet foods and the practice of feeding home-prepared foods. Owners who fed their pets a diet that consisted of at least 50% non-commercial foodstuffs had more concerns and misgivings about commercial petfood, food processing, and the petfood industry than owners who gave their pet at least 75% commercial products (2). These non-commercial feeders also had more positive attitudes toward raw and home-prepared diets than commercial feeders. Owners feeding home-prepared foods were more likely to believe that processed foods for pets are unhealthy, that cooking destroys vital nutrients, and that organic foods are safer and healthier than other foods. Some individuals also enjoy preparing their pet's food, which could potentially influence their responses (2).

Table 1. Some reasons stated by owners for feeding homemade diets.

- Palatability – owner can choose foods the pet likes
- Wish to pamper pet
- Fits with owner's eating philosophy, *e.g.*, vegetarian, organic foods
- Mistrust or misunderstanding of petfood processing or belief that processed (cooked) foods are unhealthy
- Mistrust of petfood companies
- Desire to exclude ingredients, *e.g.*, grains, by-products
- Wish to control diet
- Wish to feed a high-protein or "carnivorous" diet to dog
- Use for specific nutritional purpose where a commercial diet isn't available, *e.g.*, co-morbidities or adverse reactions to multiple ingredients

Proponents of raw food diets reportedly feel that raw foods and a high-protein diet provide more natural nutrition, comparable to that consumed by wild canids and felids (1). In a study of 218 owners who fed raw foods to their dogs, 26% indicated the main reason was to respect the dog's carnivorous nature, 24% to improve the pet's health, 21% because commercial diets had caused problems in the past, 19% because they did not trust commercial pet food, 6% because their dog did not eat commercial pet food, and 4% stated another reason (5). The main advantage of feeding a raw diet was thought by 57% of these owners to enable total control over the diet and awareness of its composition, while 23% cited their preference for animal proteins as the principal dietary component; 11% of owners cited the perceived benefit that it took a longer time for the meal to be eaten, along with the animal's apparent consequent greater satisfaction, to be the prime advantage. Only 3% of respondents considered good palatability, 1% the absence of carbohydrates, and 1% the rawness of the ingredients to be the main advantage.

Homemade diets for nutritional therapy

Although there are a wide variety of commercial pet foods available for healthy pets and for those requiring therapeutic diets, homemade diets can be useful where there is no preparation available that is suitable for an individual animal with specific problems. For example, for a dog with chronic pancreatitis and kidney disease, a suitable homemade low-fat, low-phosphorus diet can be formulated by a veterinary nutritionist, and a diet with a fat content lower than that found in commercial foods can be useful for some intestinal disorders, such as lymphangiectasia. Some animals will also have adverse reactions to multiple food ingredients and can benefit from a bespoke diet. Homemade diets may also be more palatable in some situations, as the owners (and pets) can select their preferred foods; this can be especially useful in cases such as chronic kidney disease, where the pet's appetite may be poor, although also potentially deleterious with overweight pets.

It has been proposed that homemade diets can be more digestible than both dry and wet commercially available options, resulting in better quality or smaller feces. However, there are various factors that affect a food's digestibility, including the ingredients, the amount and types of fiber, and the different heat processing techniques. One study in cats fed a dry diet, a raw diet and the same "raw" diet but cooked showed higher apparent fecal digestibility of the raw and homemade vs. the dry food (6). Another study comparing fecal digestibility of two raw diets and a dry cooked diet in kittens showed the digestibility of organic matter, protein and energy was higher for the raw products, resulting in smaller feces, but no difference in fecal score (7).

These studies compared diets with both a variety of ingredients and processing, so the effects of processing alone are difficult to determine.

As long as digestibility is high enough to provide adequate nutrition, increased digestibility of a petfood is not necessarily beneficial for all pets. Overweight pets needing lower calorie density, and animals that require a lot of fiber in their diet for colonic health may benefit from increased dietary fiber that has lower digestibility. Conversely, high digestibility can benefit some patients with some small intestinal diseases or those requiring higher caloric density.

Potential problems with homemade diets

Nutrient imbalances

There are numerous case reports and case series of problems resulting from feeding nutritionally unbalanced and/or incomplete homemade diets **(Table 2)**. Many of these involve growing puppies or kittens, where nutrient imbalances are more critical, but they are also reported in adult dogs and cats (8,9). Metabolic bone disease and nutritional secondary hyperparathyroidism occur when dietary calcium is deficient or the calcium: phosphorus ratio is incorrect **(Figure 2)**, and can occur simultaneously with rickets due to vitamin D deficiency. Abnormalities reported in dogs fed homemade diets include hypovitaminosis D, hypocalcemia, vitamin A deficiency (10), hyponatremia, hypochloremia, hyperphosphatemia, and taurine deficiency (11). There are reports of skeletal disease in kittens associated with unbalanced homemade diets due to calcium and/or vitamin D deficiencies (12). Pansteatitis from consumption of high-fat diets with insufficient vitamin E has been reported in cats fed unbalanced homemade diet (13). A diet comprised mostly of liver fed to cats can result in hypervitaminosis A, causing irreversible extensive bony osteophytes and exostoses resulting in pain and lameness.
Of course, many nutrition-related cases in practice go unreported, so the actual prevalence of these disorders is not known.

Table 2. Common nutrient deficiencies in homemade maintenance pet diets.

- Calcium
- Vitamin D
- Zinc
- Essential fatty acids (linoleic, omega-3 fatty acids)
- Vitamin E
- Choline
- Copper
- Iron
- Thiamine
- Manganese
- Selenium

© Dr Pauline Jamieson, VetsNow Referrals

Figure 2. A D-V skull radiograph of a dog with chronic kidney disease which was fed a nutrient-deficient homemade diet, resulting in nutritional secondary hyperparathyroidism and likely also renal secondary hyperparathyroidism. There is generalized osteopenia present, with thinning of some of the cortices.

Nutrient analyses of homemade recipes

Several studies have analyzed recipes for homemade diets, all of which reported deficiencies in most of the published recipes (14-16) **(Figure 3)**. A study of 200 homemade maintenance diets for dogs (64.5% written by veterinarians and 35.5% by non-veterinarians) from 34 sources determined that most were not nutritionally complete (14). Of these recipes, 92% had vague or incomplete instructions (*e.g.*, regarding ingredients, method of preparation, and supplements) and 29% omitted supplements. Calories per recipe varied from 380 to 16,348 kcal, and whilst there was at least one essential nutrient below NRC[1] or AAFCO[2] guideline in 95% of the recipes, 83.5% had multiple deficiencies. The most common deficiencies were a lack of vitamin D, vitamin E, zinc, choline, copper, omega-3 fatty acids and calcium. A study of 114 homemade diets for cats found similar vague, inadequate instructions and nutritional deficiencies, notably in choline, iron, thiamine, zinc, manganese, vitamin E, and copper. None of the recipes met the recommended NRC nutrient allowances (16).

With regard to therapeutic diets, another study reported that none of 67 homemade renal diet recipes for dogs and cats (taken from veterinary texts, pet-owner books and websites) met all of the NRC nutrient recommendation. There were frequent amino acid deficiencies, and many recipes were low in choline, selenium, zinc, and calcium (17). However, one report cites 18 dogs with CKD and hyperkalemia which had been fed commercial therapeutic renal diets; when switched to a potassium-reduced homemade renal diet devised with the supervision of a board-certified veterinary nutritionist®, serum potassium concentrations had reverted to normal in all but one dog within one to two weeks (18).

Many published recipes will include the use of a non-specific vitamin and mineral supplement. These products vary in their constituents, and most of the ones sold for pets are not formulated for use with homemade diets. In addition, some supplements designed for human use may contain vitamin D levels which can be excessive for dogs and cats. Human supplements are also unlikely to contain taurine, which is essential for cats and may or may not be sufficient in a homemade diet. For example, one study showed that whole rabbit samples did not meet any nutrient recommendation for taurine dietary concentrations, ranging from 20 to 90% of the minimum values (19)

It is often suggested that rotation of different dietary formulations provides a variety of nutrients which will compensate for any deficiencies in any one of the diets, but a study that analyzed the effect of rotating seven separate recipes showed

"Pet owners will often modify the recipes they are given, a process known as recipe drift. Modifications may include changing the amounts of ingredients, substituting ingredients, or omitting supplements, and any variation can alter the nutritional composition of the diet and potentially make it unsuitable"

Marjorie L. Chandler

[1] NRC = National Research Council
[2] AAFCO = Association of American Feed Control Officials

Figure 3. The results of a computer analysis using BalanceIt®.com software to assess an internet homemade diet recipe for an adult dog based on turkey, rice, and mixed vegetables. The gray bars denote sufficient levels for a given nutrient, while the red and white bars represent nutrients which are deficient.

Nutrient	% of requirement	Amount (per Mcal)	Range
Protein	170.4%	76.702 g	45 to (no max) g
Arginine	434.1%	5.556 g	1.28 to (no max) g
Histidine	458.9%	2.203 g	0.48 to (no max) g
Isoleucine	362.9%	3.448 g	0.95 to (no max) g
Leucine	364.6%	6.199 g	1.7 to (no max) g
Lysine	391.3%	6.182 g	1.58 to (no max) g
Methionine	246.8%	2.049 g	0.83 to (no max) g
Methionine – cystine	176.0%	2.869 g	1.63 to (no max) g
Phenylalanine	276.2%	3.121 g	1.13 to (no max) g
Phenylalanine – tyrosine	312.2%	5.776 g	1.85 to (no max) g
Threonine	283.2%	3.398 g	1.2 to (no max) g
Tryptophan	218.8%	0.875 g	0.4 to (no max) g
Valine	301.4%	3.707 g	1.23 to (no max) g
Total lipid	189.7%	26.181 g	13.8 to (no max) g
Carbohydrate	100.0%	114.014 g	0 to (no max) g
Choline	81.4%	273.063 mg	335.429 to (no max) mg
Folate	162.3%	87.653 mcg	54 to no max mcg
Niacin	780.7%	26.543 mg	3.4 to (no max) mg
Pantothenic acid	132.6%	3.978 mg	3 to (no max) mg
Riboflavin	69.6%	0.905 mg	1.3 to (no max) mg
Thiamine	124.2%	0.696 mg	0.56 to (no max) mg
Vitamin A	185.5%	695.680 mcg	375 to 18750 mcg
Vitamin B12	41.7%	0.003 mg	0.007 to (no max) mg
Vitamin B6	549.0%	2.086 mg	0.38 to (no max) mg
Vitamin E	11.8%	1.477 IU	12.5 to (no max) IU
Calcium	14.3%	0.179 g	1.25 to 6.25 g
Chloride	219.0%	0.657 g	0.3 to (no max)
Copper	49.5%	0.906 mg	1.83 to (no max) mg
Iodine	0.0%	0.000 mg	0.25 to 2.75 mg
Iron	73.7%	7.368 mg	10 to (no max) mg
Magnesium	181.5%	0.272 g	0.15 to (no max) g
Manganese	328.7%	4.102 mg	1.25 to (no max) mg
Phosphorus	97.6%	0.976 g	1 to 4 g
Potassium	86.3%	1.295 g	1.5 to (no max) g
Selenium	126.2%	0.101 mg	0.08 to 0.5 mg
Sodium	146.0%	0.292 g	0.2 to (no max) g
Zinc	52.9%	10.580 mg	20 to no max mg
Ca:P ratio	100.0%	0.183	0 to 2 n/a
EPA + DHA	100.0%	0.042 g	0 to 10.53 g
Vitamin D	14.0%	17.642 IU	125 to 750 IU

that the deficiencies were not eliminated (14). As many homemade recipes have similar deficiencies (*e.g.*, zinc), alternating recipes will not provide a complete and balanced diet.

Diet formulations

Even with a well-formulated diet, nutrient imbalances may occur, as the diet fed will only correspond with the computer formulation if the actual ingredients are consistent with those in the database. One study has shown good consistency when diets were chemically analyzed and compared to the computer analysis (14), but owners may not choose the exact ingredient recommended, and (for example) the amount of fat in ground meats can vary considerably. More pertinently, pet owners will often modify the recipes they are given, a process known as recipe or diet drift. Modifications may include changing amounts of ingredients, adding, omitting, or substituting ingredients, or omitting or changing supplements. Any of these variations can alter the nutritional composition of a diet and could potentially make it unsuitable.

Nutritional problems have been identified in commercial pet foods as well; for example, thiamine (vitamin B1) concentrations below the AAFCO minimum were identified in 12 of 90 US canned cat foods, especially in pâtés and products made by smaller companies (20). There have been recalls of commercial pet products associated with vitamin D excess, for example resulting from a mistake in a premix used in dog foods. These errors should be found at quality testing and result in recalls being made to remove the affected batches. The potential for ration imbalance therefore underlines the importance of quality control and regular nutritional analysis of pet foods. This is an inherent disadvantage of homemade diets, as there is no quality control involved in their preparation and, unlike commercial diets, such recipes are usually not tested for nutrient balance or safety. Essentially, the "feeding trial" is made on that individual pet. Even if an owner selects the food correctly and does not change the recipe, an exact match with the database cannot be ensured, especially over a prolonged period of time, as the food suppliers may change what is available. This is especially important in pets fed homemade therapeutic diets, as it may affect their disease management.

Cost

Owners may wish to feed homemade diets because they believe it will save money; however, one study showed complete homemade diets for dogs are usually more expensive than commercial dry food, although they can be cheaper than some canned diets (21).

Raw food risks

Homemade diets may include raw meat-based products and bones. Chewing on large bones does not provide sufficient dietary calcium, does not prevent dental plaque or periodontitis, and can cause tooth fractures. Raw meat-based diets, whether homemade or commercial, can be a health risk for dogs, cats, and their owners due to potential pathogen contamination. While this has occasionally also been reported in dry commercial cooked petfoods, it is uncommon, as such products are processed at high temperatures which kill bacteria. Contamination is even more unlikely in unopened canned foods, as they are sterilized in the can. Microbiological contamination is much more likely with raw foods, and has been reported numerous times; for example, various studies have noted commercial raw frozen or freeze-dried pet foods to be contaminated with a variety of zoonotic bacterial and parasitic pathogens (22,23).

It is not possible to know what percentage of raw homemade diets are contaminated, as they are not monitored, but the prevalence of contamination of meat and poultry products for human consumption is known. A meta-analysis of 78 studies conducted in 21 European countries showed that *Staphylococcus aureus* was the main pathogen, being detected in 38.5% of poultry meat (with a range of 25.4-53.4%), followed by *Campylobacter* species at 33.3% (22.3-46.4%). *Listeria monocytogenes* and *Salmonella* spp. were present at a lower prevalence at 19.3% (14.4-25.3%) and 7.1% (4.60-10.8%), respectively (24).

© Shutterstock

Figure 4. A nutritional assessment, including a discussion of the pet's current diet, should be a feature of every consultation.

Importantly, whilst owners may not see overt clinical signs of bacterial infection in animals fed contaminated raw foods, the pets can still be shedding pathogens in their feces and saliva. The fecal shedding of pathogens constitutes both a public health hazard and a risk to household members, especially those who are immunocompromised, young, elderly or pregnant. Raw feeding may also contribute to bacterial antibiotic resistance; such foods have been identified as a risk factor for shedding of extended-spectrum beta-lactamase-producing *Enterobacteriaceae* in household cats (25).

Discussing diet choice with owners

Owners may have strong feelings about their choice of diet for their pet, so nutrition can be a challenging discussion. It is important to ask about feeding as part of the nutritional assessment, and to have a non-judgmental discussion about the reasons for the choice of diet **(Figure 4)**. Owners may have misperceptions about ingredients or processing of commercial pet foods, and may have obtained their "data" from biased and misinformed online or book sources, so it can be appropriate to ask the owner if they would like more advice. In particular, when the diet being fed may not be complete or balanced, as is the case for most homemade recipes, providing information about the pet's nutritional requirements may help, and where there is a risk for, or the presence of, a diet-related disorder (such as nutritional

secondary hyperparathyroidism in a young animal), there is some urgency in correcting the diet. Providing highly visual and written information is more effective than only verbal advice, which may not be recalled correctly or may be misinterpreted.

The healthcare team should recognize that the owner's feeding choices were likely made in the hope that they were in the best interest of the pet. Positive aspects of the owner's management and care for their pet should be noted; if an owner feels that they are being judged for poor management of their pet, they are more likely to become defensive and less likely to implement the necessary dietary changes. Once an owner is willing to contemplate a change in feeding, a definitive plan should be outlined for transition to a complete and balanced diet, and can include the options of switching to a commercial diet, utilizing a program and supplement such as that provided by a reputable website (*e.g.*, Balanceit.com), or being referred to a board-certified veterinary nutritionist®.

CONCLUSION

While owners may choose a homemade food for their pet because they believe it to be the healthiest – or possibly only – choice available, they should be aware of the potential risks involved, as well as any perceived benefits. Ultimately, any diet should be as safe from pathogens as possible, and must deliver complete, balanced nutrition including appropriate supplements. The clinician should strive to include dietary advice whenever appropriate during a consultation, as omission could lead to a nutritionally related disorder in either the short or longer term for the pet.

REFERENCES

1. Laflamme DP, Abood SK, Fascetti AJ, *et al*. Pet feeding practices of dog and cat owners in the United States and Australia. *J. Am. Vet. Med. Assoc.* 2008;5:687-694.
2. Michel JE, Willoughby KN, Abood SK, *et al*. Attitudes of pet owners toward pet foods and feeding management of cats and dogs. *J. Am. Vet. Med. Assoc.* 2008;233:1699-1703.
3. Dodd S, Cave N, Abood S, *et al*. An observational study of pet feeding practices and how these have changed between 2008 and 2018. *Vet. Rec.* 2020;186:(19):643.
4. Seneviratne M, Subasinghe DWD, Watson PJ. A survey of pet feeding practices of dog owners visiting a veterinary practice in Colombo, Sri Lanka. *Vet. Med. Sci.* 2016;2(2):106-116. DOI: 10.1002/vms3.16
5. Morelli G, Bastianello S, Catellani P, *et al*. Raw meat-based diets for dogs: survey of owners' motivations, attitudes and practices. *BMC Vet. Res.* 2019;15:74.
6. Kerr KR, Vester Boler BM, Morris CL, *et al*. Apparent total tract energy and macronutrient digestibility and fecal fermentative end-product concentrations of domestic cats fed extruded, raw beef-based, and cooked beef-based diets. *J. Anim. Sci.* 2012;90(2):515-522.
7. Hamper BA, Kirk CA, Bartges JW. Apparent nutrient digestibility of two raw diets in domestic kittens. *J. Feline Med. Surg.* 2016;18(12):991-996.
8. de Fornel-Thibaud P, Blanchard G, Escoffier-Chateau L, *et al*. Unusual case of osteopenia associated with nutritional calcium and vitamin D deficiency in an adult dog. *J. Am. Anim. Hosp. Assoc.* 2007;43(1):52-60.
9. Chastain CB, Panciera D, Waters C. Nutritional secondary hyperparathyroidism in six cats. *Small Anim. Clin. Endocrinol.* 2000;10(2):5.
10. Hall G, Breheny C, Khan Z, *et al*. Severe nutritional deficiencies and osteopenia in a dog fed a homemade raw diet. *Vet. Rec. Case Rep.* 2020;8(1):e001038.
11. Hutchinson D, Freeman LM, McCarthy R, *et al*. Seizures and severe nutrient deficiencies in a puppy fed a homemade diet. *J. Am. Vet. Med. Assoc.* 2012;241:477-483.
12. Lenox C, Becvarova I, Archipow W. Metabolic bone disease and central retinal degeneration in a kitten due to nutritional inadequacy of an all-meat raw diet. *JFMS Open Reports* 2015;1(1):2055116915579682.
13. Niza MMRE, Vilela CL, Ferreira LMA. Feline pansteatitis revisited: hazards of unbalanced home-made diets. *J. Feline Med. Surg.* 2003;5:271-277.
14. Stockman J, Fascetti AJ, Kass PH, *et al*. Evaluation of recipes of home-prepared maintenance diets for dogs. *J. Am. Vet. Med. Assoc.* 2013;242:1500-1505.
15. Dillitzer N, Becker N, Kienzle E. Intake of minerals, trace elements and vitamins in bone and raw food rations in adult dogs. *Br. J. Nutr.* 2011;106 Suppl 1:S53-56.
16. Wilson SA, Villaverde C, Fascetti AJ, *et al*. Evaluation of the nutritional adequacy of recipes for home-prepared maintenance diets for cats. *J. Am. Vet. Med. Assoc.* 2019;15:254(10):1172-1179.
17. Larsen JA, Parks EM, Heinze CR, *et al*. Evaluation of recipes for home-prepared diets for dogs and cats with chronic kidney disease. *J. Am. Vet. Med. Assoc.* 2012;240(5):532-538.
18. Segev G, Fascetti AJ, Weeth LP, *et al*. Correction of hyperkalemia in dogs with chronic kidney disease consuming commercial renal therapeutic diets by a potassium-reduced home-prepared diet. *J. Vet. Intern. Med*. 2010;24(3):546-550.
19. Owens TJ, Fascetti AJ, Calvert CC, *et al*. Rabbit carcasses for use in feline diets: amino acid concentrations in fresh and frozen carcasses with and without gastrointestinal tracts. *Front. Vet. Sci*. 2021 https://doi.org/10.3389/fvets.2020.592753
20. Markovich JE, Freeman LM, Heinze CR. Analysis of thiamine concentrations in commercial canned foods formulated for cats. *J. Am. Vet. Med. Assoc*. 2014;244(2):175-179. DOI: 10.2460/javma.244.2.175
21. Vendramini THA, Pedreinelli V, Macedo HT, *et al*. Homemade versus extruded and wet commercial diets for dogs: cost comparison. *PLOS One* 2020;15(7):e0236672.
22. van Bree FPJ, Bokken GCAM, Mineur R, *et al*. Zoonotic bacteria and parasites found in raw meat-based diets for cats and dogs. *Vet. Rec.* 2018;182:50.
23. Jones JL, Wang L, Ceric O, *et al*. Whole genome sequencing confirms source of pathogens associated with bacterial foodborne illness in pets fed raw pet food. *J. Vet. Diagn. Invest.* 2019;31(2):235-240. DOI: 10.1177/1040638718823046
24. Gonçalves-Tenório A, Silva B, Rodrigues V, *et al*. Prevalence of pathogens in poultry meat: a meta-analysis of European published surveys. *Foods* 2018;7(5):69-85. DOI: 10.3390/foods7050069
25. Baede VO, Broens EM, Spaninks MP, *et al*. Raw pet food as a risk factor for shedding of extended-spectrum beta-lactamase-producing *Enterobacteriaceae* in household cats. *PLOS One* 2017;12:e0187239.

DIET AND CANINE DILATED CARDIOMYOPATHY

KEY POINTS

- Recent reports that grain-free diets can contribute to dilated cardiomyopathy (DCM) in dogs are currently the subject of much research.
- Additional research is necessary to investigate the possible role of taurine in canine DCM, as well as identifying other potential factors that may influence this disease.

What is the link between certain diets and canine heart disease? This paper gives an overview of the current situation and offers some advice for clinicians.

Introduction

Dilated cardiomyopathy (DCM) is an idiopathic functional abnormality of the myocardium causing left ventricular systolic dysfunction, cardiac chamber dilation and/or ventricular tachyarrhythmias. Definitive breed predispositions exist for the Doberman Pinscher, Great Dane, Standard Schnauzer and Irish Wolfhound, where a pattern of inheritance and/or genetic mutations are described **(Figure 1)**. As in cats, diet-associated DCM in dogs is also well-described, with clusters of cases being reported in the 1990s and into the first decade of this century; these were mainly characterized by taurine deficiency and were linked to diets low in protein or that contained lamb and/or rice (1-3). Several studies at that time investigated possible risk factors, which primarily appeared to negatively impact the dog's ability to synthesize adequate taurine to meet its metabolic needs (4-6).

After resulting modifications to various commercial diets, including those formulated to be lower in protein for management of specific diseases, diagnosis of DCM in dogs of non-genetically predisposed breeds apparently occurred only occasionally until late 2016, when clinicians began noticing more cases. The United States Food and Drug Administration (FDA) announced an investigation into the potential connection between diet and canine DCM in July 2018 and this was followed by updates in February and June 2019. The most recent update in September 2020 included over 1,100 reports of canine DCM suspected to be related to diet, in particular those marketed as grain-free and especially with diets containing legumes such as lentils and peas. This report also included detailed follow-up on a subset of the affected dogs, which showed that dietary change, most often together with taurine supplementation, resulted in complete or partial disease reversal (7).

Unsubstantiated claims that grains cause allergies and other negative health outcomes in dogs and cats have contributed to the popularity of grain-free pet foods. However, there is no evidence of an inherent safety risk of grains for pets, nor any medical or nutritional indication for grain-free diets *per se*. Descriptive and investigative research by multiple groups is ongoing or published, although the role of specific dietary characteristics in diet-associated DCM linked to grain-free foods remains unclear.

Figure 1. Certain dog breeds are well-recognized as being genetically prone to dilated cardiomyopathy, including the Doberman Pinscher and the Great Dane, but recent attention has focused on the possibility that certain dietary components can predispose dogs to heart disease.

Jennifer Larsen

DVM, PhD, Dip. ACVN, School of Veterinary Medicine, University of California, Davis (UCD), USA

Dr. Larsen earned her veterinary degree from UC Davis before completing a PhD in Nutritional Biology and attaining Diplomate status from the American College of Veterinary Nutrition. She is currently Professor of Clinical Nutrition at the UCD School of Veterinary Medicine and, as Chief of Service, she provides clinical nutritional consulting through the Nutrition Support Service at the UCD Veterinary Medical Teaching Hospital.

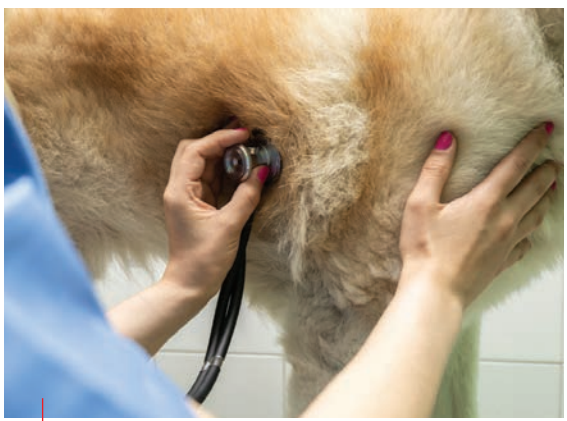
Joshua A. Stern

DVM, PhD, Dip. ACVIM (Cardiology), School of Veterinary Medicine, University of California, Davis, USA

Dr. Stern earned his veterinary degree from The Ohio State University in 2008, where he also undertook a small animal rotating internship. He then completed a cardiology residency at North Carolina State University and earned his PhD at Washington State University in cardiac genetics. He is currently Professor of Cardiology and Associate Dean for Veterinary Medical Center Operations at the UCD Veterinary Medical Teaching Hospital. His main areas of interest are in cardiac genetics and cardiac pharmacogenomics, with a particular focus on the role of individualized medicine in veterinary cardiology patients.

Clinical findings and diagnostic guidelines

While DCM can be occult (asymptomatic), clinical signs may include coughing, dyspnea, tachypnea, syncope, and occasionally ascites. A soft systolic murmur consistent with mitral valve regurgitation and/or a gallop sound (S_3) may be auscultated at the left apex. A tachyarrhythmia of sinus, supraventricular or ventricular origin may be noted. In some cases, a murmur or an arrhythmia may be the first sign of the occult form of the disease, and this should not be overlooked. Since primary valvular disease is relatively uncommon in young or middle-aged large breed dogs, and the detection of DCM before the development of congestive heart failure (CHF) may be beneficial in the long-term management of the case, identification of any new murmur, gallop, or tachyarrhythmia in suspect breeds may warrant a thorough cardiac work-up **(Figure 2)**.



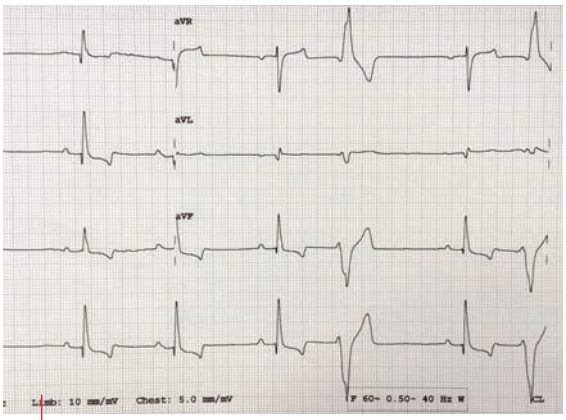
© Shutterstock

Figure 2. Routine clinical examination may pick up a new murmur, gallop, or cardiac arrhythmia in a patient. Any abnormal heart sound warrants further cardiac investigation which may include tests such as cardiac biomarkers, electrocardiogram, chest radiographs, or echocardiography.

Many dogs with DCM have normal electrocardiograms (ECGs), but in some cases atrial and/or ventricular enlargement patterns (R > 3.0 mV Lead II for the left ventricle) may be noted. Chamber enlargement patterns in dogs are specific but not sensitive findings, as many DCM cases have relatively normal ECG complex measurements. Sinus tachycardia, atrial fibrillation or ventricular arrhythmias are common **(Figure 3)**. In some cases, ventricular tachyarrhythmias can develop before any ventricular dilation or systolic dysfunction. Routine Holter monitoring may help detect these, and this technique has become a mainstay of the screening process for this disease, particularly in breeding populations.

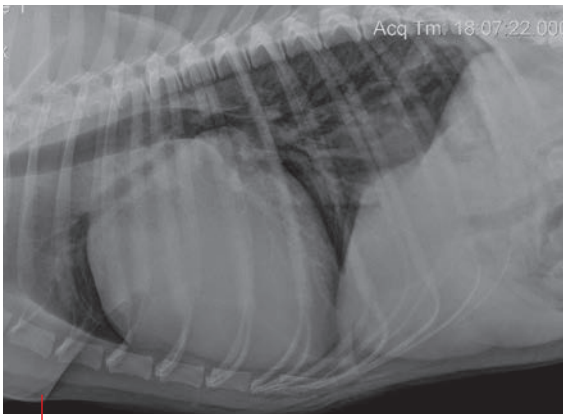
If the disease is diagnosed in the early stages, radiographic findings may be subtle. Therefore, depending on the stage of the disease, thoracic radiographs can be within normal limits or may indicate atrial and ventricular enlargement (typically left) with or without pulmonary venous distension and pulmonary edema **(Figure 4)**. Biatrial and biventricular enlargement may be noted in some cases. Echocardiography is not only the diagnostic test of choice for diagnosing canine DCM, it is also an important test for occult disease. Findings in the patient with overt disease should include left (and sometimes right) atrial and ventricular dilation and decreased systolic function.

Cardiac biomarkers are currently a major area of research for identification of occult heart disease. NT-proBNP is released when the ventricles are dilated, hypertrophic or subjected to increased wall stress or stretch, and concentrations of NT-proBNP



© Joshua Stern

Figure 3. An ECG is shown from a 2-year-old Golden Retriever with confirmed diet-associated DCM. Ventricular premature complexes are observed, which is a common feature of this disease and should prompt cardiac evaluation when observed.



© Joshua Stern

Figure 4. A lateral thoracic radiograph from a 2-year-old Golden Retriever that presented for evaluation of a cardiac arrhythmia and soft heart murmur. This dog had severe DCM on cardiac ultrasound, had a lifelong history of grain-free diet, and improved dramatically with diet change.

are typically increased in dogs with CHF and can be used to help diagnose or exclude CHF in dogs with cough or dyspnea. NT-proBNP may also be helpful in the identification of occult disease, but the frequency of false positives is a concern. Additionally, cardiac troponin-I is a cardiac biomarker that, when elevated, is consistent with occult cardiomyopathy, and although this test is specific for DCM, it is not sensitive enough to identify all cases. Interestingly, a recent study involving four breeds of apparently healthy dogs showed that cardiac troponin-I was elevated in those eating diets labeled as grain-free when compared to those that were fed a grain-inclusive diet (8).

Potential risk factors for diet-associated DCM

Various factors, including taurine deficiency, are postulated to have a role in recent cases of diet-associated DCM. However, many cases do not have taurine assessments performed as part of the work-up, or when tested, are shown not to have low plasma and/or whole blood taurine concentrations. It should be noted that most clinical cases of diet-associated canine DCM are identified once cardiac dysfunction is apparent, which may or may not be accompanied by CHF, and sulfur amino acid metabolism – and in particular taurine kinetics – under those circumstances have not been characterized. The disconnect between the results of taurine status assessments and clinical changes in the cardiac muscle may also be due to other factors. For example, it should be recognized that taurine has long been used as a readily analyzed marker for sulfur amino acid adequacy and, indirectly, the adequacy of general methyl donor status. However, other markers or assessments may enable a more complete clinical picture. Additional research is necessary to investigate the possible role of taurine in canine DCM, as well as fully characterize the interrelationships of other potential factors or nutrients that likely influence this disease.

Certain dietary characteristics have been linked to many cases of canine DCM, and notably in relation to particular ingredients. Specific ingredients such as peas or lentils, which are commonly used in grain-free diets, appear to be largely over-represented. Several studies have identified grain-free diets as a risk factor in this disease, but the reason for these findings is unclear (8,9,10,11). Legumes are a source of starch as well as significant amounts of fiber and protein; however, they are limited in sulfur amino acids, and some contain anti-nutritional factors that negatively impact protein digestibility and amino acid bioavailability. Proper manufacturing processes, including adequate cooking times and temperatures, are expected to largely destroy such anti-nutritional factors, but these procedures must be well defined by the manufacturer for any particular ingredient combination. In addition, some amino acids (especially lysine, cysteine, and methionine) undergo non-enzymatic reactions during processing that can result in decreased bioavailability even without negative effects on

"Whenever possible, whole blood and plasma taurine concentrations should be measured in any dog with DCM, given that low concentrations are very good indicators of disease risk and of nutritional inadequacy."

Jennifer Larsen

global protein digestibility. In summary, many characteristics of pet food impact the overall dietary amino acid balance as well as the microbiome (12). These are all likely to influence availability and utilization of sulfur-containing metabolites, pathway intermediates, methyl donors such as choline, and enzyme co-factors such as vitamins.

Commercial pet foods and DCM risk

Without a complete understanding of the underlying mechanisms, it is difficult to define specific recommendations for product modifications that will help prevent canine diet-associated DCM. Many balanced pet food products have been successfully formulated over the years using a range of ingredients, including potatoes and legumes, to provide the necessary protein and starch. However, it is apparent that commercial foods must better address the diversity of the canine population, so that all diets meet the needs of the many dogs that are not "average" in terms of energy and nutrient requirements. In addition, *in vivo* testing is essential, since amino acid bioavailability cannot be estimated by chemical analysis of the food (13).

Many manufacturers have initiated taurine supplementation in grain-free dog diets, with the implication or even assertion that this strategy will prevent DCM. However, this masks the ability to assess for poor sulfur amino acid bioavailability or deficiency. Although it is not unreasonable to add taurine to some diets where the protein level is intentionally restricted (such as in some veterinary therapeutic diets), maintenance of taurine adequacy in dogs is probably most appropriately accomplished by increasing the concentration of bioavailable methionine and cysteine (14). The use of high-quality, digestible protein sources and/or supplementation with purified methionine is recommended, together with consideration for overall amino acid balance and adequate provision of methyl donors necessary for metabolism of sulfur amino acids (15).

Assessment of suspected cases

Individualized nutritional assessment is a critical aspect of management of any patient. Evaluation of the complete diet along with the medical history, patient status, and physical examination findings will inform diagnostic and therapeutic plans, including dietary options. Current and historical body weights, body condition scores, and muscle condition scores should all be taken into account.

Whole blood and plasma taurine concentrations should be measured in all dogs (and cats) with DCM, given that low concentrations are very good indicators of disease risk and of nutritional inadequacy, even if additional factors may also influence the development of the condition (16). Due to the high-taurine content of granulocytes and platelets, sample clotting or hemolysis can result in falsely increased plasma taurine concentrations; however, whole blood taurine concentration is not confounded by these sampling and handling effects. As such, when plasma taurine concentrations are low, a diagnosis of taurine deficiency can be made; however, whole blood taurine concentrations may be used to substantiate a diagnosis of taurine deficiency when plasma concentrations are normal or equivocal. In addition, whole blood taurine concentrations are only slightly altered after meal consumption, whereas the plasma concentration may change substantially depending on taurine status and the composition of the pre-sampling meal relative to the longer-term diet.

Management recommendations

A dietary change is recommended if it is suspected of playing a role in the development of heart disease, especially if there is a large discrepancy between the predicted and actual calorie requirement. Notably, the FDA report from 2020 described disease resolution or improvement with diet change, and two recent studies have shown that dogs fed grain-free or non-traditional diets when diagnosed with DCM have prolonged survival times and improvement of heart function once a diet change is instituted as part of therapy (9,11). For owners that wish to discontinue commercial pet food entirely, it is recommended to pursue the formulation of a customized home-cooked diet recipe through consultation with a board-certified veterinary nutritionist. Internet or book recipes are not advised due to documented problems with adequacy and outdated strategies for disease management.

In the United States, any suspected diet-associated cases of dilated cardiomyopathy (regardless of diet history) should be reported to the FDA.
For Doberman Pinschers, genetic testing is available (through North Carolina State University and other establishments) which may help

"One recent study showed that apparently healthy dogs eating grain-free diets had elevated cardiac troponin-I when compared to those on grain-inclusive diets, suggesting myocardial injury."

Joshua A. Stern

elucidate the etiology, but it should be remembered that more than one cause in an individual dog is possible.

Administration of angiotensin converting enzyme (ACE) inhibitors may be beneficial for the dog with early ventricular dilation, with or without systolic dysfunction. A major study in Doberman Pinschers with ventricular dilation showed that this drug prolongs the time period before the onset of CHF (17). Although this study was limited to evaluation of one breed, the use of ACE inhibitors (*e.g.*, enalapril at 0.5 mg/kg PO q12H) for other breeds with occult DCM may be considered. The same study also showed that once a certain level of heart enlargement and systolic dysfunction is reached, Doberman Pinschers benefit from oral pimobendan therapy (~0.3 mg/kg PO q12H) which prolongs the time to onset of CHF (17). Pimobendan is an inodilator drug that exerts its action through phosphodiesterase III inhibition and calcium sensitization. In the authors' practice, all dogs with occult cardiomyopathy receive both pimobendan and an ACE-inhibitor, with or without dietary change and taurine supplementation depending on breed, blood taurine levels, and diet history. Therapy for dogs with DCM and CHF is expanded to add furosemide (and often spironolactone) on top of the existing pimobendan and ACE-inhibitor treatment, with the treatment regime being frequently modified to address ventricular arrhythmias or atrial fibrillation as needed.

Taurine supplementation is safe and is specifically indicated in cases with confirmed deficiency (500-1500 mg per dog q12H). Carnitine supplementation can also be considered (50 mg/kg q8H), but the indication and benefits are difficult to assess, and it can be costly. Additionally, omega-3 fatty acids may be beneficial in cardiac disease, with a suggested total dose of 125 mg EPA+DHA/kg$^{0.75}$/day. Note that, when calculating the total intake, the clinician should consider the fatty acid contributions from all sources, including the base diet and any supplements.

Many dogs with diet-associated DCM improve with appropriate therapy including diet change and nutritional supplementation, and in some cases the improvement can be quite significant, including reversal of CHF and even successful withdrawal of medications. This degree of reversibility is similar to that seen in cats, and is one defining feature of diet-associated (compared to hereditary) canine DCM.

CONCLUSION

Although the possible links between diet and canine dilated cardiomyopathy (DCM) are currently far from clear, there is ample evidence to support that certain nutritional factors influence the disease. Any animal presenting with a heart murmur, gallop, or tachyarrhythmia should undergo a thorough cardiac work-up, including a detailed diet history and whole blood taurine measurements. Early intervention in dogs found to have DCM using appropriate therapy and – where indicated – dietary changes can have a significant effect on the outcome of such cases.

REFERENCES

1. Fascetti AJ, Reed JR, Rogers QR, *et al*. Taurine deficiency in dogs with dilated cardiomyopathy: 12 cases (1997-2001). *J. Am. Vet. Med. Assoc.* 2003;223:1137-1141.
2. Backus RC, Cohen G, Pion PD, *et al*. Taurine deficiency in Newfoundlands fed commercially available complete and balanced diets. *J. Am. Vet. Med. Assoc.* 2003;223:1130-1136.
3. 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. *J. Am. Vet. Med. Assoc.* 2001;62:1616-1623.
4. Backus RC, Ko KS, Fascetti AJ, *et al*. Low plasma taurine concentration in Newfoundland dogs is associated with low plasma methionine and cyst(e)ine concentrations and low taurine synthesis. *J. Nutr.* 2006;136:2525-2533.
5. Ko KS, Backus RC, Berg JR, et al. Differences in taurine synthesis rate among dogs relate to differences in their maintenance energy requirements. *J. Nutr.* 2007;137:1171-1175.
6. 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. (Berl.)* 2003;87(9-10):359-372.
7. www.fda.gov/animal-veterinary/cvm-updates/interdisciplinary-scientific-cooperation-will-lead-way-understanding-non-hereditary-dcm. Accessed 25th April 2021.
8. Adin D, Freeman L, Stepien R, *et al*. Effect of type of diet on blood and plasma taurine concentrations, cardiac biomarkers and echocardiograms in 4 dog breeds. *J. Vet. Intern. Med.* 2021;35(2):771-779.
9. Kaplan JL, Stern JA, Fascetti AJ, *et al*. Taurine deficiency and dilated cardiomyopathy in golden retrievers fed commercial diets. *PLOS One* 2018;13(12):e0209112.
10. Walker AL, DeFrancesco TC, Bonagura JD, *et al*. Association of diet with clinical outcomes in dogs with dilated cardiomyopathy and congestive heart failure. *J. Vet. Cardiol.* 2021 (epub ahead of print)
11. Freid KJ, Freeman LM, Rush JE, *et al*. Retrospective study of dilated cardiomyopathy in dogs. *J. Vet. Intern. Med.* 2021;35(1):58-67.
12. Kim SW, Rogers QR, Morris JG. Maillard reaction products in purified diets induce taurine depletion in cats which is reversed by antibiotics. *J. Nutr.* 1996;126(1):195-201.
13. Hendriks WH, Bakker EJ, Bosch G. Protein and amino acid bioavailability estimates for canine foods. *J. Anim. Sci.* 2015;93:4788-4795.
14. Backus RC. Could dietary taurine supplementation in dogs be masking a problem? In *Proceedings*, American College of Veterinary Internal Medicine Forum 2009; Montreal, Canada.
15. Mansilla WD, Marinangeli CPF, Ekenstedt KJ, *et al*. Special topic: The association between pulse ingredients and canine dilated cardiomyopathy: Addressing the knowledge gaps before establishing causation. *J. Anim. Sci.* 2019;97(3):983-997.
16. Zicker SC, Rogers QR. Use of plasma amino acid concentrations in the diagnosis of nutritional and metabolic diseases in veterinary medicine. In *Proceedings*, IV Congress of the International Society for Animal Clinical Biochemistry 1990:1-15.
17. Summerfield NJ, Boswood A, O'Grady MR, *et al*. Efficacy of pimobendan in the prevention of congestive heart failure or sudden death in Doberman Pinschers with preclinical dilated cardiomyopathy (the PROTECT Study). *J. Vet. Intern. Med.* 2012;26(6):1337-1349.

FAQs ABOUT CAT NUTRITION

Ana Luísa Lourenço

BVSc, PhD, Dip. ECVCN, University of Trás-os-Montes and Alto Douro, Villa Real, Portugal

Dr. Lourenço obtained both her animal science and veterinary degrees at the University of Trás-os-Montes and Alto Douro (UTAD), and remained there whilst researching for her PhD. She then completed an alternative residency in small animal nutrition at Ghent and Utrecht University. A recognized specialist, Dr. Lourenço is currently assistant professor at the School of Agricultural and Veterinary Sciences of UTAD and is board-certified in veterinary nutrition by the European College of Veterinary and Comparative Nutrition.

The feline species is unique in many ways, and none more so than in its nutritional requirements, as illustrated in this question-and-answer paper from Ana Lourenço.

KEY POINTS

1. Cats are obligate carnivores and it is extremely difficult, although in theory possible, to formulate a balanced maintenance diet for cats which is entirely free from animal products.
2. It is often said that cats should not be given milk due to a "lactose intolerance". This does not, however, mean that milk is forbidden at all times.
3. Carbohydrates are not contraindicated for cats, but the dietary carbohydrate source must be in a form that is easily digestible.
4. Cats have a remarkable ability to concentrate their urine if drinking water is scarce, but for optimal health they should be encouraged to drink freely wherever possible.

Introduction

Cats were first domesticated by humans some 10,000 years ago, and since then they have become one of our most popular companion animals. This has, not unnaturally, meant that we want to offer them the best possible care, and to this end the last few decades have seen much research effort focused on the feline species, and especially towards their nutritional requirements. It has gradually become apparent that the cat is an animal full of peculiarities, not least from the dietary point of view, and this article will explore the practical implications of some of these.

Q: Can a cat be fed a diet free from animal products?

A: Cats are often referred to as being strict or obligatory carnivores; in the wild they consume food that is almost entirely from animal sources, they are anatomically designed to hunt and eat prey, and their metabolism is adapted for their natural diet (1) **(Figure 1)**. Cats have various nutritional requirements, of which some must be supplied via their diet as they cannot be produced in sufficient quantities by their intrinsic metabolic pathways. Research shows that the cat has at least 45 nutrients which are essential for health (2), and because of their metabolic peculiarities they have higher requirements for protein, arginine, methionine,

© Shutterstock

Figure 1. Cats have evolved over many centuries to hunt, catch and eat prey, and their metabolism is based around an animal-based diet.

cysteine, taurine, arachidonic acid, vitamin A, vitamin D, niacin and pyridoxine when compared to omnivores (1,2).

Some essential dietary nutrients are primarily sourced from animal-derived ingredients; these include several amino acids (lysine, methionine, cysteine and taurine), some vitamins (A, D and B_{12}), and some fatty acids (arachidonic, eicosapentaenoic and docosahexaenoic acids). It can be challenging to obtain these from sources other than animals, so to formulate a feline diet that is free from animal products these nutrients must be provided by alternative means, either from chemically synthesized products or from specific non-animal sources. However, such options are not necessarily straightforward, and their use can raise other challenges. Firstly, it is not enough to simply know a given product contains the desired nutrient; it is essential to know the actual level of the required nutrient it contains. This is because the product may have a lower concentration of the substance than an equivalent animal-based product, or have a lower activity *(e.g.*, it may contain vitamin D_2 instead of D_3), so the bioavailability (*i.e.*, the amount of an ingested nutrient that is available for metabolism or storage within the body) needs to be quantified.

Secondly, some ingredients that are vegetable-based or derived from non-animal sources can also negatively impact a diet; they may alter the overall digestibility and bioavailability of its nutrients – for example, by adversely affecting the content and structure of the carbohydrate – and they can reduce the overall palatability.

And thirdly, the possible risks posed by such diets in terms of their potential impact on common diseases must also be evaluated – such as the effects on urinary pH, with the possibility that this can predispose to the formation of bladder calculi. In short, there are considerable challenges in producing an animal-free diet for cats that is not only complete and balanced when subject to chemical analysis, but proven to be adequate as a long-term maintenance food. As far as can be ascertained, these data are lacking for any of the commercially available "vegetarian" or "vegan" foods presently marketed for cats, and research to date shows that they often do not even meet the recommended essential nutrient levels (3-5). One study has suggested that in some cases the health of cats fed an animal-free diet was unaffected (3), but it was unclear if these cats had outdoor access where they could have hunted – and in addition the evaluation period may not have been long enough for individual animals to display any signs of deficiency.

The author is unaware of any major multinational pet food company that currently produces a feline diet that is free from animal ingredients. This in itself is significant; since these companies have the in-depth knowledge and the financial resources required to research and produce this type of diet, and given that this product would find a ready market with some pet owners, it can be argued that such foods pose, for now at least, an unnecessarily high risk for cats. Decades of research has revealed a great deal of unexpected information on feline nutrition – and we are still learning – so on this basis any formulation for cat food should be supported by good scientific evidence in order to be as safe as possible. The bottom line must be that, for now at least, cats should be fed a meat-based diet, as the alternatives raise the real possibility for sub-optimal long-term nutrition.

Q: Is feline heart disease linked to diet?

A: The short answer here is – in some situations, possibly. In the late 1980's taurine was identified as the key nutrient which could reduce the prevalence of dilated cardiomyopathy in cats (6). The mechanism by which low levels of taurine within cardiac muscle result in dilated cardiomyopathy and heart failure is not yet fully understood, although it is postulated to be due to disturbances in the calcium and energy metabolism of the myocardial muscle (7,8). Taurine

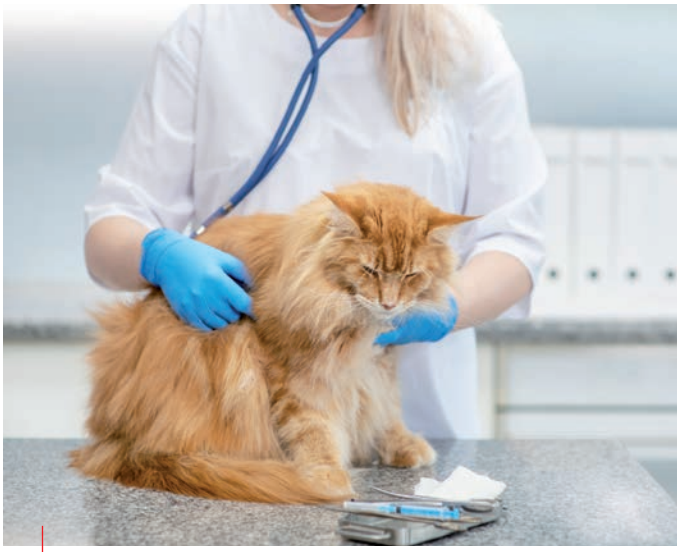
"Some vegetable-based and non-animal alternative sources can also negatively impact on a diet by affecting the digestibility and bioavailability of its nutrients and reducing diet palatability."

Ana Luísa Lourenço

is a non-protein ß-amino sulfonic acid which is abundant in the cat's natural prey. In contrast to most mammals, cats have low concentrations of cysteine dioxygenase and cysteine sulfinic acid decarboxylase, key enzymes in the metabolic pathway of taurine synthesis (9). This idiosyncrasy means that cats are unable to efficiently synthesize taurine using methionine and cysteine, and therefore are dependent on their diet to provide the taurine they need as a metabolic substrate.

Once taurine was recognized as an essential nutrient, manufactures started to incorporate it in commercial petfood diets, and what was once a frequent cause of cardiomyopathy in cats became a rare occurrence. Most cases are nowadays identified in cats fed homemade diets, but it should be stressed that it is not just this species that may be susceptible. A recent Food and Drug Administration review in the USA has highlighted a possible link between certain diets and the development of dilated cardiomyopathy, with most of the recently reported cases being in dogs, although a small number of cats were also affected (10). Various factors are still unclear, but the investigation is centered around the diets that were fed to affected animals and, more specifically, a suspicion that the diets had low bioavailability levels of taurine. It should be stressed that there are diverse factors involved in the etiology of dilated cardiomyopathy, and that more robust data collection will be needed before any conclusions can be drawn from this review.

If dietary taurine deficiency is identified as the likely cause in a case of feline dilated cardiomyopathy, with tests demonstrating low levels of the amino acid in plasma and whole blood **(Figure 2)**, supplementing the diet with taurine usually results in an immediate improvement of heart function and – assuming that the cat survives the immediate critical period – the condition should resolve within 6 months. So whilst it is true that certain diets can cause heart disease in cats, a well-formulated commercial diet is unlikely to predispose to cardiomyopathy.



© Shutterstock

Figure 2. It is advisable to measure taurine plasma levels if dilated cardiomyopathy is suspected in a cat.

© Shutterstock

Figure 3. Most cats will enjoy drinking milk – but this does not mean that it is good for them, especially if given to excess.

Q: Should a cat be given milk to drink?

A: Most cats enjoy drinking milk, but this does not necessarily mean it is healthy for them **(Figure 3)**. Perhaps a better question is "how much milk is appropriate to give a cat – and what problems can be linked to milk consumption?" Certainly cats, like all other mammals, naturally survive exclusively on milk in their first weeks of life, and even after they start to ingest solid food they will partially rely on their mother's milk until they are fully weaned **(Figure 4)**. The lactase activity in a cat's intestine declines with age, and with it the capacity to digest lactose, the main sugar present in milk. If the amount of lactose ingested by a cat surpasses its capacity to digest it, the remaining lactose will ferment in the gastrointestinal tract, leading to clinical signs such as vomiting and diarrhea. An adult cat's capacity to digest lactose is therefore less than that of a kitten, but research has confirmed that an adult cat can cope with (at least) 1.3 g lactose per kg bodyweight on a daily basis (11). It is notable that the lactose concentration in milk is remarkably constant, both between different species *(e.g.*, cow, sheep or goat's milk) and across different types of milk (*i.e.*, skimmed, low-fat or whole milk), with an average maximum of 5% (12). This means that a cat should be able to cope with ingesting up to 25 mL per kg bodyweight per day; so if a 4 kg cat was to drink less than 100 mL of milk each day, it is unlikely to develop any clinical signs of lactose intolerance. This does not exclude the fact that a few individuals can have a particularly low digestive capacity for lactose, so in some cases clinical signs can develop even if a cat consumes a small quantity of milk.

© Shutterstock

Figure 4. A female cat's milk will provide a complete diet for her kittens in the first few weeks of life.

However, quite apart from the lactose intolerance aspect, if a cat is given milk regularly it is important that the overall nutritional profile of the diet and the amount of energy ingested is also taken into account. A queen's milk is a very complete food for her kittens, containing all the essential nutrients, but it does not constitute a balanced diet for a cat after weaning. Milk is also energy dense, so if offered to a cat on a regular basis it should either be regarded as an integral component of a complete and balanced diet, or considered to be an extra treat; with this scenario the amount offered should not exceed 10% of the energy content of the basic diet. Given that whole bovine milk has an energy density of 69 kcal/100 mL (or around half of this for skimmed milk) (12) and that a castrated 4 kg cat has an average daily energy requirement of 130-190 kcal/day (52-75 kcal/kg$^{0.67}$), the daily amount of milk to be offered as a treat would be 20-30 mL, or around double this amount if skimmed milk is used. At this level, the lactose intake is well below the maximum.

Finally, another aspect to consider is the possibility that some cats can be allergic to casein. Although the literature does not report this as being prevalent in cats (13), obviously such individuals should not be fed milk at all.

In summary, it can be said that milk in moderation is acceptable for most cats, but excessive amounts can be detrimental in the long term.

Q: Can a cat digest and metabolize carbohydrates?

A: The metabolism and physiology of the feline species has evolved on a diet based on small prey (such as mice and birds) which provide very limited amounts of carbohydrate (14), and when given the choice domestic cats will still select diets that have a low-carbohydrate content (15). These facts lead to the assumption that a cat's digestive system and metabolism cannot cope with carbohydrates. In fact, glucose (one of the simplest carbohydrates) is just as essential for the feline carnivore as for omnivores or herbivores. Glucose is the main or only source of energy for brain and red blood cells, leukocytes and some specific cells within the renal medulla, testes and eye (16). Glucose is also required for the synthesis of some non-essential amino acids, vitamin C and nucleic acids, and (in lactating queens) to manufacture lactose (16). Nevertheless, carbohydrates are not mandatory in the cat's diet, as they can synthesize the glucose that they require from other sources.

Blood glucose concentrations in the cat will rapidly return to normal baseline levels after intravenous glucose administration, and the fasting glucose blood levels in this species are very similar to that of other mammals who have different dietary needs (16), so it is clear that cats can indeed metabolize carbohydrates. Although they have evolved to have a more limited digestive capacity for complex carbohydrates than other domesticated species *(e.g.*, dogs or pigs (17)), cats can digest and absorb carbohydrates efficiently as long as they are properly processed (*i.e.*, ground and/or cooked) and are not ingested in excessive amounts (18). So the question is not so much "should cats be fed carbohydrates?" but rather "what needs to be considered when including carbohydrates in a cat's diet?" – which is why cats should not be fed carbohydrates that are raw or in amounts that exceed their digestive capacity.

Q: Can a cat become diabetic if fed a high-carbohydrate diet?

A: Diabetes mellitus is a relatively common disease in cats, with some studies indicating a prevalence as high as 1.25% (16), and owners of affected cats may ask if the carbohydrate component in their pet's diet has contributed to development of the disease **(Figure 5)**. Firstly, it is appropriate to consider why, since a cat's natural diet typically has very low levels of carbohydrates (at around 2% on a metabolizable energy (ME) basis (14)), most commercial cat foods

© Shutterstock

Figure 5. Cat owners may ask if excess dietary carbohydrates contribute to diabetes but there is not enough evidence to support this theory.

contain relatively high levels of carbohydrates. This is not simply because this class of nutrient is cheaper or more sustainable than protein or fat, or that it is a technological requirement for making kibble. It is also because carbohydrates have various beneficial properties; they are extensively digested and absorbed in the gastrointestinal tract, they can partially replace fat and protein in the diet as an energy source, and they also have a metabolic-sparing effect on amino acids.

Since feline diabetes is an endocrine disorder that is caused mainly by an intolerance to glucose due to insulin resistance, it can be difficult not to assume that the carbohydrate content of a cat's diet is the cause of the disease, but the evidence to support this hypothesis is very weak (16). There are some data that suggest offering affected cats lower carbohydrate diets can result in better glycemic control and higher clinical remission scores (19), and diabetic cats can certainly benefit from such diets, but this does not necessarily mean that carbohydrates are the cause of the condition.

Blood glucose levels rise after a meal, followed by a physiological insulin release from the pancreas to counteract this effect. If there was a connection between higher levels of dietary carbohydrates and diabetes, it would be expected that this mechanism would be altered in some way, resulting in lower glucose tolerance and/or lower insulin sensitivity. Some studies have found changes in glucose tolerance when feeding cats high-carbohydrate/low-protein diets when compared to high-protein/low-carbohydrate diets, but other studies have not confirmed this (20). At least one study has failed to establish any relation between the carbohydrate content of the diet and insulin sensitivity (21), and it is also true that even diets with high levels of starch do not usually cause hyperglycemia and glycosuria in cats.

In addition, the idea that raised blood glucose levels secondary to high-carbohydrate diets in cats are responsible for excessive insulin secretion from pancreatic ß-cells (which could then lead to destruction of the cells and subsequently diabetes mellitus), has been refuted by a study that showed that feline pancreatic ß-cells are more responsive to amino acids and less responsive to glucose than the ß-cells of omnivorous species (22). This suggests that there is more to the etiology of feline diabetes than the carbohydrate content of a diet. Nonetheless, studies have shown that the feline species has a lower glucose clearance rate than in dogs or humans, and that a chronic hyperglycemia state (30 mmol/L over a 10-day period, achieved by glucose infusion) can cause dysfunction and loss of ß-cells which impairs insulin secretion (16). It is, however, important to note that this scenario does not mimic a cat's physiological response to food intake, so in conclusion there is no solid evidence that currently supports a hypothesis that the carbohydrate content of a diet can cause diabetes in the cat. Obesity, due to inactive lifestyle and excessive caloric intake, and advancing age remain the greatest risk factors for diabetes mellitus (16).

© Shutterstock

Figure 6. Although most cats have unrestricted access to water, many of them may not drink a great deal.

Q: Can a cat be healthy without drinking water?

A: Cats cannot survive without water, but they will satisfy their fluid requirements both by exogenous means (from drinking water and the water content of their food) **(Figure 6)**, and by endogenous processes (from water produced from oxidation of carbohydrates, fats, and protein). They will lose water in their urine, faces and via evaporation processes, and whilst various factors (including disease, and the ambient temperature and humidity) can increase these losses, the typical average daily water requirement of a cat is about 50 mL per kg body weight (23).

When necessary, cats have an incredible ability to concentrate their urine by reabsorbing water within the kidneys. Although this is probably a valuable evolutionary adaptation to aid survival in arid environments, it has been suggested that this ability, along with a low water intake, may contribute to the development of some of the urinary tract disorders which are so prevalent in this species (24). To reduce this risk, any measure or strategy that encourages a cat to ingest more water is to be encouraged – so for example, by providing water fountains or extra water bowls around the house **(Figure 7)**. However, at the same time it is important to avoid anything that may upset the cat, as stress can be a major contributor to some feline health issues. The factors that help maximize a cat's water intake whilst minimizing any element of stress need to be tailored for each individual animal (24), and the best approach is simply to allow the cat to decide how, where, and when to drink.

But drinking water is not the only option available; the water contained within its food is also a good option to fulfil a cat's fluid requirements. Food is a good source of water for cats in nature, since the

© Shutterstock

Figure 7. A drinking fountain can be one way to encourage a cat to increase its water intake in a stress-free way.

prey they catch to survive in the wild (small rodents and birds) will have a water content of around 70% (14). Offering a diet with a high water content is obviously both very effective and stress-free, as long as the cat enjoys this type of food, and many years ago it was shown that the cats could meet their water requirements solely from a diet of fish or meat (25). More recent research has shown that a cat's daily water intake and the volume of urine it produces is significantly higher when wet food (which contains around 75-80% water) is offered rather than dry food (which is about 8% water) (26). Therefore, while any stress-free method that encourages a cat to drink is to be welcomed, offering a high-moisture content food may, for many cats, be the easiest way to ensure sufficient water intake for good homeostasis.

CONCLUSION

The cat is unique in many ways, and its dietary requirements and peculiarities underline the adage that "a cat is not a small dog" – the nutritional needs of the feline species can vary quite markedly from the canine requirements, and it is always worthwhile devoting a significant period of any consultation to discussing a cat's dietary needs. Good pet health starts with good communication in the consult room, and it is essential for the small animal clinician to have excellent nutritional knowledge in order to deliver the best advice to owners.

REFERENCES

1. Morris JG. Idiosyncratic nutrient requirements of cats appear to be diet-induced evolutionary adaptations. *Nutr. Res. Rev.* 2002;15:153.
2. National Research Council. Nutrient Requirements of Dogs and Cats. Washington, D.C.: *National Academies Press;* 2006;361-370.
3. Engelhard R. Feldstudie zur vegetarischen Ernährung von Hunden und Katzen. *Thesis*, Ludwig-Maximillians-Universität München, 1999.
4. Kanakubo K, Fascetti AJ, Larsen JA. Assessment of protein and amino acid concentrations and labeling adequacy of commercial vegetarian diets formulated for dogs and cats. *J. Am. Vet. Med. Assoc*. 2015;247:385-392.
5. Zafalon RVA, Risolia LW, Vendramini THA, *et al*. Nutritional inadequacies in commercial vegan foods for dogs and cats. *PLOS One* 2020;15:1-17.
6. Pion PD, Kittleson MD, Rogers QR, *et al*. Myocardial failure in cats associated with low plasma taurine: a reversible cardiomyopathy. *Science* 1987;237:764-768.
7. Schaffer S, Sayed-Mozaffari M, Kramer J, *et al*. Effect of drug-induced taurine depletion on cardiac contractility and metabolism. *Biochem. Pharmacol.* 1986;35:985-989.
8. Huxtable RJ. From Heart to Hypothesis: a mechanism for the calcium modulatory actions of taurine. In: Huxtable RJ, Franconi F, Giotti A. (eds*) The Biology of Taurine. Advances in Experimental Medicine and Biology* Vol 217. Boston, MA. Springer 1987;371-387.
9. Park T, Jerkins AA, Steele RD, *et al*. Effect of dietary protein and taurine on enzyme activities involved in cysteine metabolism in cat tissues. *J. Nutr.* 1991;121:181-182.
10. Food and Drug Administration. FDA Investigation into Potential Link between Certain Diets and Canine Dilated Cardiomyopathy. 2019;1-17.
11. Kienzle E. Carbohydrate metabolism of the cat (3); Digestion of sugars. *J. Anim. Physiol. Anim. Nutr*. 1993;69:203-210.
12. Pereira PC. Milk nutritional composition and its role in human health. *Nutrition* 2014;30:619-627.
13. Mueller RS, Olivry T, Prélaud P. Critically appraised topic on adverse food reactions of companion animals (2): Common food allergen sources in dogs and cats. *BMC Vet. Res*. 2016;12:10-13.
14. Plantinga EA, Bosch G, Hendriks WH. Estimation of the dietary nutrient profile of free-roaming feral cats: possible implications for nutrition of domestic cats. *Brit. J. Nutr*. 2011;106 Suppl:S3-48.
15. Hewson-Hughes AK, Hewson-Hughes VL, Miller AT, *et al*. Geometric analysis of macronutrient selection in the adult domestic cat, *Felis catus*. *J. Exp. Biol.* 2011;214:1039-1041.
16. Verbrugghe A, Hesta M. Cats and carbohydrates: The carnivore fantasy? *Vet. Sci.* 2017;4:1-22.
17. Verbrugghe A, Hesta M, Daminet S, *et al*. Nutritional modulation of insulin resistance in the true carnivorous cat: A review. *Crit. Rev. Food Sci. Nutr*. 2012;52:172-182.
18. De-Oliveira LD, Carciofi AC, Oliveira MCC, *et al*. Effects of six carbohydrate sources on diet digestibility and postprandial glucose and insulin responses in cats. *J. Anim. Sci.* 2008;86:2237-2246.
19. Behrend E, Holford A, Lathan P, *et al*. 2018 AAHA Diabetes Management Guidelines for Dogs and Cats. *J. Am. Anim. Hosp. Assoc.* 2018;54:1-21.
20. Perez-Camargo G. Cat nutrition: What is new in the old? *Comp. Cont. Educ. Pract. Vet*. 2004;26(Suppl 2A):5-10.
21. Hoenig M. Comparative aspects of diabetes mellitus in dogs and cats. *Mol. Cell. Endocrino*l. 2002;197:221-229.
22. Curry DL, Morris JG, Rogers QR, *et al*. Dynamics of insulin and glucagon secretion by the isolated perfused cat pancreas. *Comp. Biochem. Physiol. Part A: Physiol*. 1982;72:333-338.
23. Zentec J. Untersuchungen zum Mineralstoffhaushalt der Katze unter besonderer Berücksichtigung des Magnesiums. *Thesis*, *Tierärztliche Hochschule,* Hannover, 1987.
24. Handl, S, Fritz J. The water requirements and drinking habits of cats. *Vet. Focus* 2018;28.3:32-40.
25. Prentiss PG, Wolf AV., Eddy HA. Hydropenia in cat and dog; ability of the cat to meet its water requirements solely from a diet of fish or meat. *Am. J. Physiol.* 1959;196:625-632.
26. Zentek J, Kaufmann D, Pietrzak T. Digestibility and effects on fecal quality of mixed diets with various hydrocolloid and water contents in three breeds of dogs. *J. Nutr.* 2002;132:1679S-1681S.

PET OBESITY: NEW CHALLENGES, NEW SOLUTIONS

Alexander J. German

BVSc, PhD, CertSAM, Dip. ECVIM-CA, SFHEA, FRCVS, School of Veterinary Science, University of Liverpool, UK

Professor German qualified from the University of Bristol in 1994 and spent two years in mixed practice before returning to Bristol to undertake a PhD, and then a residency in small animal internal medicine. He moved to the University of Liverpool in 2002 where he is currently the Royal Canin Professor of Small Animal Medicine. His clinical and research interests include gastroenterology and comparative obesity biology.

Obesity in our pets is nothing new – but Alex German offers some new ideas as to how the problem can be best tackled.

KEY POINTS

1. Obesity is not a new problem, but the prevalence of obesity in companion animals seems to be increasing inexorably in recent years.
2. New strategies are required to help owners ensure that their pets lose excess fat and maintain a healthy adult weight.
3. Better preventative measures are also required in an effort to prevent obesity developing in the first place.
4. "Weight stigma" must be avoided when dealing with obese animals, and good communication is essential to improve owner compliance.

Introduction

Obesity can be defined as "a disease in which body fat has accumulated to the point that the health of an individual is adversely affected" and – in both pets and humans – it is associated with negative effects on wellbeing and health, a shortened lifespan and a poorer quality of life. Despite much recent scientific and media interest, the prevalence of obesity continues to increase, and the problem shows no signs of abating (1,2). This article considers the current challenges with pet obesity before looking at possible options that may help improve the management of this chronic disease.

What are the challenges?

Increasing overall prevalence of obesity

Numerous studies over the last 30 years have reported the incidence of obesity in pets, and although comparisons between studies should be made cautiously, there is an apparent trend towards an increasing prevalence in both dogs and cats. In studies using a body condition score (BCS) of 1 to 9, the number of dogs rated as 8/9 or 9/9 increased from 10% in 2007 to 19% in 2018, and for cats in the same period from 19% to 34% (2).

Increased prevalence in growing animals

There is an even more concerning trend in growing animals. A recent study reported the prevalence of overweight and obesity in cats at 12-13 months of age to be 7% (3) – which may be an underestimate, given that this employed data from owners (rather than veterinarians) on weight status. More concerning is the situation with young dogs; one study found that of 516 juvenile (< 24 months) dogs, 190 (37%) were either in overweight or obese body condition, with the prevalence increasing steadily during the growth phase, from 21% (21/100) in dogs under 6 months of age to 52% (16/31) in dogs 18-24 months of age (4).

Changing breed popularity

Recent changes in demographics, with the increased popularity of small-breed (particularly brachycephalic) dogs are also significant. Historically, it was typically medium-to-larger breed dogs that were predisposed to obesity, but a recent survey (2) noted the greatest prevalence was now mostly in small and toy breeds of dog **(Figure 1)**.

Owner assessment

Owners often underestimate the true body condition of their pet, assuming them to be slimmer than they are. This may be because their overall perception of body shape is incorrect, or it may be that their assessment is linked to constant exposure to overweight and obese pets, and exacerbated by media images that depict animals perceived to be "perfect" examples of their breed; for example, in a recent observational study 26% of animals photographed at a national dog show were judged overweight (5). This might explain why veterinary and owner estimations of body condition can differ, and why owners can distrust professional assessments regarding obesity (6).

Increasing number of animals "beyond the scale"

Of major concern is the fact that whilst the 9-point BCS scale is useful, 9/9 depicts an animal that is 40% above its ideal weight, but this arguably does not reflect the degree of adiposity seen in the current generation of pets **(Figure 2)**, many of which are now "beyond the scale". A long-term survey has noted an average of 46% of patients to be > 40% above ideal weight (author's unpublished data), with the most recent years (2015-2020) showing 59% of animals to be past this marker.

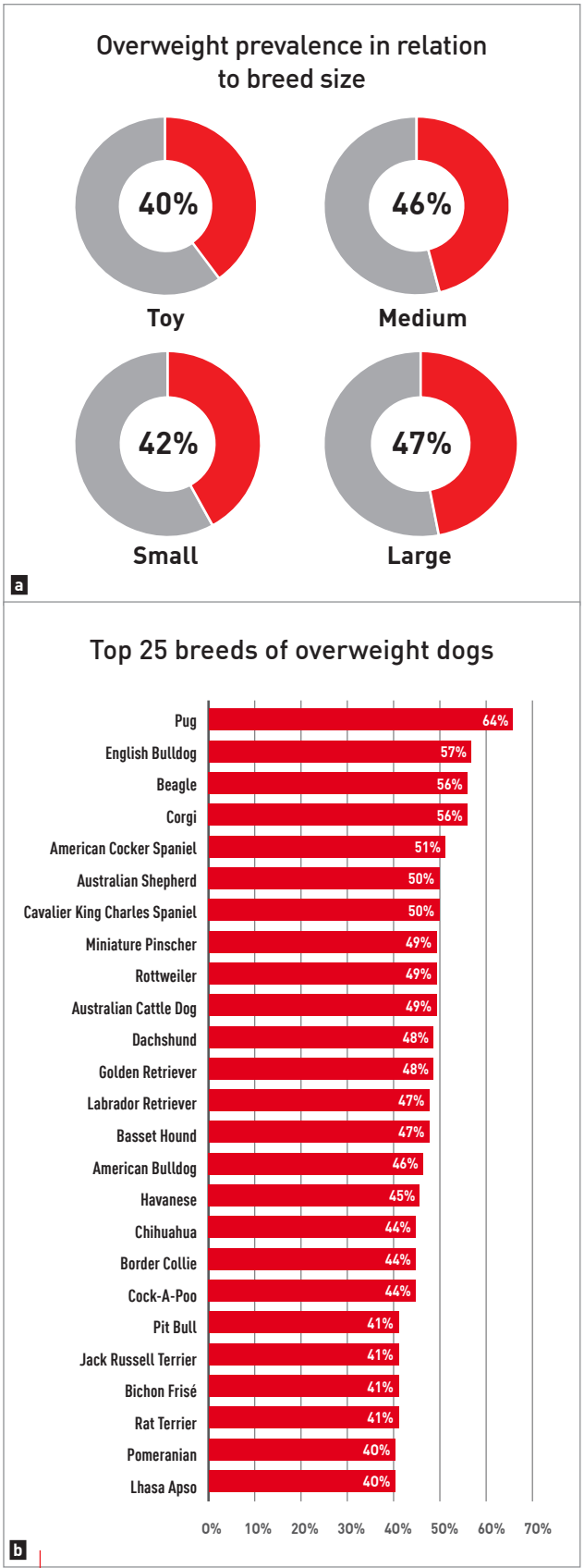
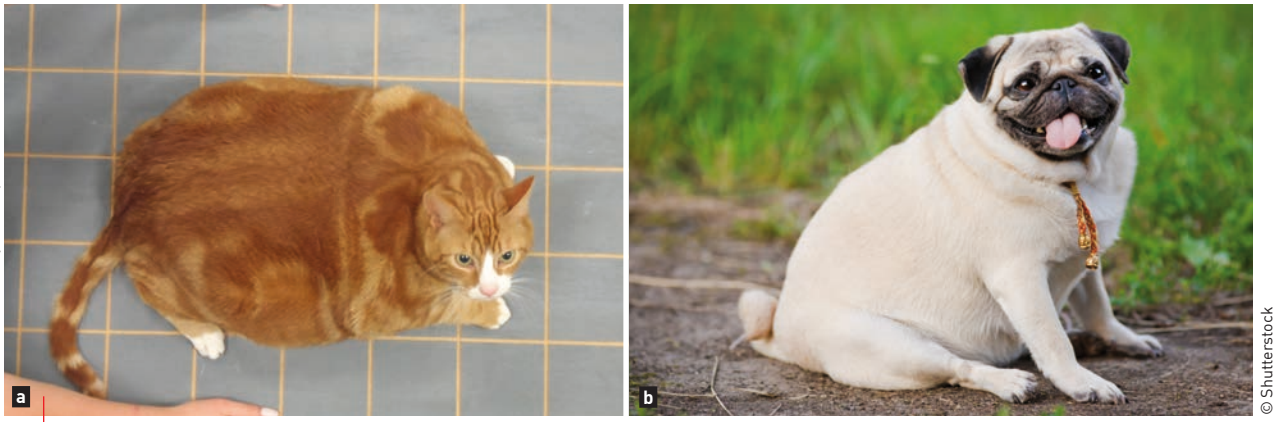


Figure 1. Prevalence of overweight body condition in different dog breeds as seen at Banfield Pet Hospital. Although overweight prevalence increased with breed size **(a)**, smaller breeds were the most commonly affected **(b)**, and Pugs topped the list, with 64% identified as being overweight.

Efficacy of weight management protocols

Given the negative correlation between the percentage of body fat mass and the success of weight management programs, there is a



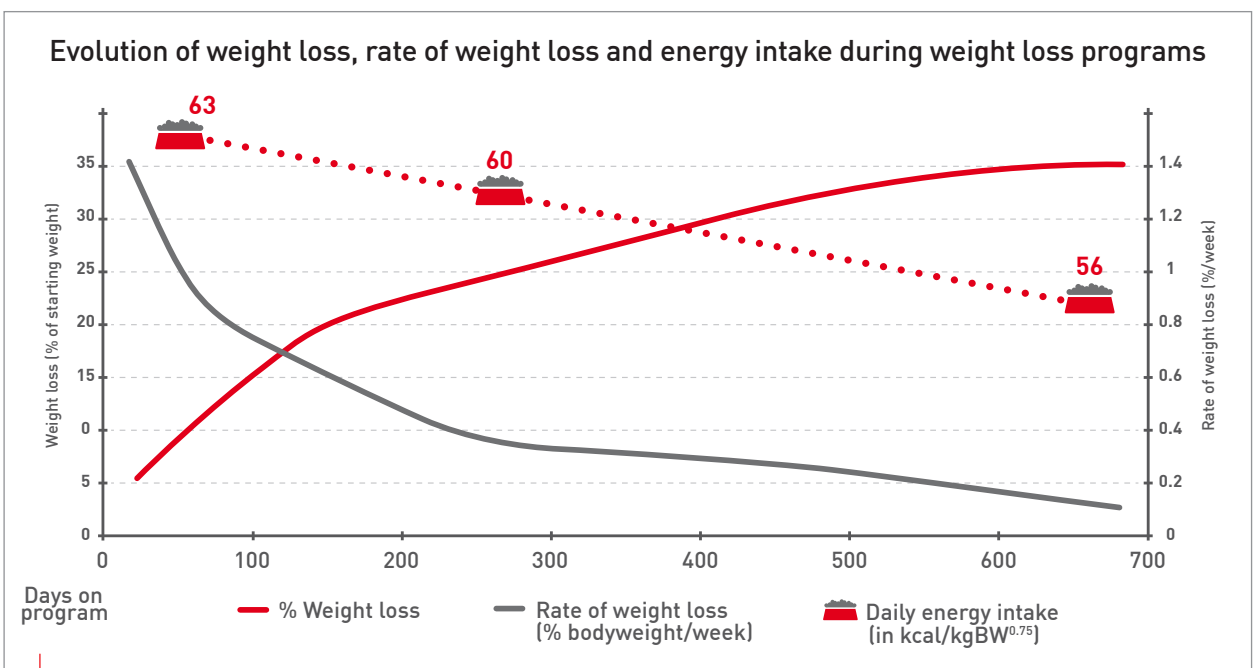
© A. German/University of Liverpool

© Shutterstock

Figure 2. A cat **(a)** and dog **(b)** with severe obesity, which is defined as having a body condition significantly greater than 40% above their ideal weight. As a consequence, their body condition is beyond the characteristics defined in the conventional 9-unit BCS scale.

significant challenge with returning obese pets to their ideal weight. Some professionals have claimed that weight management is easy (7), but this is a misperception, perhaps because many early studies were often in colonies where the dogs were young, healthy and often only modestly (< 20%) overweight, and where owner influence was absent. Such studies are not typical of pets with obesity, and more recent research has shown that even with marked dietary energy restriction the rate of weight loss is typically less than 1% per week (8,9). The reasons for this include a more variable pet population (in terms of age, neuter status and breed), the fact that pets are often very severely overweight, and often have comorbidities (10). However, owner factors, such as the feeding of table scraps and treats, are also important (9). This suggests that, in contrast to the findings from colony studies, achieving weight loss in obese pets is extremely challenging.

The figures for overall success in dieting schemes are often disappointing. In one study only 53% of dogs with obesity enrolled in a 6-month weight loss program completed the course, and educating owners about nutrition made little difference (8). In a second study examining compliance with weight management (10), 61% of dogs successfully reached their target weight, whilst in a similar study in cats with obesity, just 45% reached their weight goal (11). A key factor associated with success or failure is the severity of the obesity, *i.e.*, the greater the body fat mass, the more likely it is for the animal to fail to complete the program. Importantly, attrition from a weight program is not uniform; compliance is usually good in the first 12 weeks, with >80% of animals still enrolled and averaging over 8% of body weight loss **(Figure 3)**, but very few will have reached their target weight in this period and more will discontinue the program in subsequent weeks.



© Royal Canin / Redrawn by Sandrine Fontègne

Figure 3. Weight loss in dogs is most marked in the first 12 weeks or so after starting a weight management program.

A further challenge is the fact that many animals will subsequently regain weight. In recent studies, 48% and 46% of dogs and cats respectively regained weight after successfully reaching their target (12,13). This is disappointing and emphasizes that managing obesity is a lifelong process which provides a major challenge to pet owners.

So a combination of slow percentage of weight loss, poor completion rate and risk of subsequent weight regain underlines the fact that successful weight management is testing, and that in reality only a minority of pets on a program will ever reach their target. However, most concerning is the fact that few dogs and cats with obesity ever undergo any sort of weight management at all. It is estimated that over half of all pet dogs and cats are either overweight or have obesity (1) yet only 1.4% of veterinary health records mention the pet's weight status (14). Given that so few veterinarians will formally identify this disease, it is unsurprising that weight management success is so poor.

Attitudes about obesity

When considering the cause of disease, society tends to assign "responsibility and blame" – *i.e.*, those affected are either considered to be victims or perpetrators. So-called "disease stigma*" occurs when an individual is blamed for having a particular condition because of a moral or other failing (15), and this includes human obesity, with the predominant societal opinion being that people with the condition are personally responsible either because they are lazy, or because they overeat, or both. Recent research also suggests that such attitudes are used to justify weight discrimination, despite evidence that obesity is a complex chronic disease with multiple risk factors beyond the control of the individual (16).

*https://implicit.harvard.edu/implicit/selectatest.html

"The best strategy when dealing with a pet with obesity is to modify our approach, maximizing the benefits and minimizing the failures, in the hope of improving the quality of life of as many animals as possible."

Alexander J. German

This weight stigma is also commonly found in many healthcare professionals (15), and the issue has recently been investigated within the veterinary profession. In one study, veterinarians admitted to using stigmatizing terms to describe excess weight in dogs, and reported feeling blame, frustration, and disgust towards both dogs with obesity and their owners (17). Veterinarians also expressed the belief that owners with obesity were responsible for causing obesity in their dogs, and were pessimistic about such individuals complying with treatment recommendations. There is also indirect evidence of weight stigma in other studies; for example, most veterinarians believe that "owner-related" factors are the main reason for development of obesity in pets (18). These opinions are at odds with the fact that multiple risk factors have been identified for companion animal obesity, including genetic factors.

There is an idea that weight stigma is positive, because it incentivizes people with obesity to lose weight – yet evidence actually suggests that it can negatively influence such individuals by decreasing their chances of successful weight loss, as well as affecting their mental health (19). It is tempting to speculate that weight stigma might affect the care that dogs and cats with obesity receive. Could these attitudes be associated with the fact that very few veterinarians record the terms "overweight" or "obese" in clinical records (14), and explain why they are reluctant to hold conversations with owners about obesity? In the opinion of the author, until we address the attitudes of veterinarians and wider society towards obesity – in both pets and people – we will continue to struggle to manage this disease effectively.

Where are the solutions?

Rethink weight management strategies

The author believes that the best strategy is to modify our approach, maximizing the benefits and minimizing the failures, in the hope of improving the quality of life of as many animals as possible. The first aspect is to consider the goals of weight loss. Currently, much of the focus is on the "numbers" – such as the ideal weight and the percentage or rate of weight loss. Instead, goals should relate to the benefits that weight loss can bring, such as improving metabolic health, mobility and quality of life. Prior to agreeing a weight loss plan, it helps to have an in-depth conversation with the owner regarding their concerns and priorities. For example, an owner may be worried that their dog has severe osteoarthritis, and their key priority is to improve its mobility. Here, rather than the outcome for weight loss being to lose a particular percentage of body weight, it should arguably be better mobility and less chronic pain, and the amount of actual weight lost is simply a path towards the overall goal.

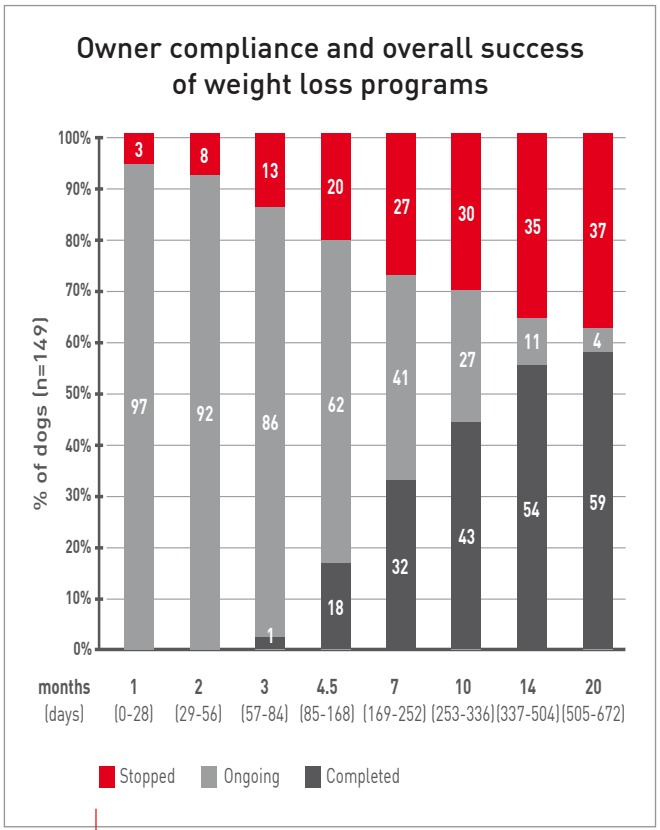


Figure 4. A bar chart showing owner compliance and overall success of a weight management program; compliance decreases markedly by 5 months after starting such a program.

Another consideration is knowing when failure in weight loss programs occurs. As explained above, compliance is typically very good in the first few weeks, with a reasonable rate of weight loss, typically ~1% per week **(Figure 3)**; however, beyond this time the process becomes more challenging, with the rate of weight loss slowing and compliance issues setting in **(Figure 4)**. Therefore, rather than designing a program to return a pet to its ideal weight, a standard "time-limited" protocol may be more useful. This can be likened to the use of chemotherapy, where a set procedure often involves standardized doses and timings, and a defined length of therapy. Outcomes are then reviewed at this time point, and further treatments and protocols are recommended accordingly. Weight loss programs could be approached in a similar way; 12 weeks is a sensible length for such a protocol, given that outcomes are best during this time irrespective of the amount of weight that the patient must lose, and the primary focus can be in helping the client to ensure completion. At this point the outcomes can be appraised, not just in terms of the weight lost but also by reviewing the health benefits that have been achieved. Agreement can then be made as to the next stage; this might be further weight loss, or a shift to weight maintenance, where the priority becomes preventing rebound.

There are many potential benefits of such an approach. Firstly, greater emphasis can be placed on factors that matter, especially for the owner, such as improvements in quality of life, with less focus on achieving a target weight. Reviewing the goals that have been agreed before the program commences (*e.g.*, improved mobility, decreased use of analgesic medication for concurrent osteoarthritis) after 12 weeks can then inform the need for further cycles of weight loss. Secondly, a pre-defined endpoint gives better certainty for the owner in terms of what they are committing to – so although they may find the weight loss phase difficult, the end is always in sight. Thirdly, it maximizes the period when weight loss is most successful (which in turn maximizes compliance); and, finally, it recognizes that success is not determined by reaching a nominal "target weight" but rather that even modest weight loss can lead to improved quality of life. In this respect, studies have demonstrated that a loss in the region of 10% of the starting weight is possible during a 12-week period (20), and this is generally associated with notable improvements in mobility and quality of life.

Focus more on obesity prevention

Given that most dogs and cats never start a weight management program in the first place, veterinary professionals should place greater emphasis on obesity prevention. This has three main components, namely identifying "at-risk" individuals before obesity has developed, proactively monitoring at-risk individuals for life, and promoting maintenance of a healthy weight and lifestyle

Identifying "at-risk" individuals before obesity has developed

Using known risk factors **(Box 1)** to identify dogs and cats at risk of obesity enables preventive measures to be best targeted. Some of the most significant risk factors are as follows:

- **Patterns of growth.** In children, future risk of obesity is associated with certain patterns of growth, including rapid growth and catch-up growing, and a similar phenomenon has been reported in both cats (21) and dogs (22).

- **Breed.** This is particularly the case where obesity is more prevalent in certain breeds due to genetic influences (*e.g.*, Labrador Retrievers, Golden Retrievers, Pugs). In cats, mixed-breed cats (domestic shorthair and longhair) are at greatest risk, with genetic factors also being a risk factor for obesity.

- **Neutering.** Neutering is an important risk factor in both dogs and cats, probably because alterations in sex hormones can lead to changed

behavior, most specifically increased food-seeking and decreased physical activity. Because neutering is pre-planned, there should be no excuse for implementing prevention strategies afterwards.

- **Comorbidities.** Many other co-existing diseases can alter energy flux, either by increasing energy intake or decreasing expenditure, and these can predispose to inappropriate weight gain.

- **Food and feeding behavior.** There are varying opinions with respect to the associations between obesity and food, but arguably the most consistently identified risk is giving extra food in the form of table scraps and treats. In addition, certain feeding behaviors in cats are implicated as possible risk factors for obesity.

- **Environment and activity.** Animals living indoors (especially apartments) have a greater risk of developing obesity than those with outdoor access. Cats living either with dogs or with one or two other cats are also predisposed to becoming obese.

- **Owner factors.** A number of owner factors have also been implicated in the development of obesity in dogs and cats.

Box 1. Various risk factors that can predispose to excess weight gain and obesity in dogs and cats.

Medical effects on energy flux
• Polyphagia associated with hyperadrenocorticism (dog)
• Polyphagia as side effect of drugs, *e.g.*, corticosteroids, anticonvulsants (dog)
• Neutering (cat, dog)
• Decreased physical activity due to musculoskeletal disease (cat, dog)
• Decreased basal metabolic rate associated with hypothyroidism (dog)
Dietary associations
• "Grocery store" foods (dog)
• "Premium" foods (cat)
• Dry food (cat)
• Dietary fat (but not dietary carbohydrate) content (cat)
• Free-choice feeding (cat)
• *Ad libitum* feeding (dog)
• Number of meals and snacks (dog)
• Table scraps (cat, dog)
• Animal present during food preparation (cat, dog)
Owner factors
• Lower average income (dog)
• Body mass index of owner (dog, cat)
• Not viewing obesity as a disease (dog)
• Not believing that obesity has health risks (dog)
• Lesser interest in preventive health (cat)
• Frequency and/or duration of walks (dog)
• Less time spent playing with pet (cat)
• Over-humanization by owner (dog)
• Pet used as human companion substitute (cat)
• Close observation of feeding behavior (cat, dog)
• A stronger owner-animal bond (cat, dog)
• Seeing pet as a baby (dog)
• Allowing pet to sleep on bed (dog)

Proactively monitoring at-risk individuals for life

The above factors should enable a veterinary professional to determine an individual's risk of obesity and allow best targeting of prevention strategies, and these should be implemented before the disease has developed (*e.g.*, at or before 12 weeks of age), and continued for life.

A key strategy is to monitor bodyweight from the time of initial vaccinations, throughout the growth phase and into adulthood. Although BCS is a useful means of determining weight status in adult dogs, existing methods have not been properly validated in growing animals. Instead, monitoring of bodyweight, facilitated by the use of growth charts (23), can be useful. Evidence-based growth charts have recently been developed for puppies (https://www.waltham.com/resources/puppy-growth-charts), and their use can allow rapid identification of abnormal patterns of growth, not least those that are associated with the risk of obesity (24). Puppies should be weighed monthly until 6 months of age and then at least every 3 months until they reach adult weight. This maximizes the likelihood of a puppy reaching skeletal maturity in ideal body condition, and at this point the BCS can be used to confirm optimal condition, with the weight recorded in the patient's health records as its "healthy weight". The aim from then on is to ensure that this is maintained (to within ±5%) for the rest of the pet's adult life. Ideally, animals should be weighed every 6 months, and no less frequently than once a year (*i.e.*, at annual vaccination), but with more frequently weight checks introduced during the senior phase of life, for example every 3 months. Ideally, animals should attend the veterinary practice for weighing, since the same set of calibrated electronic scales can be used; in addition, body condition can be assessed, and any other minor health concerns addressed. However, where this is difficult (for example, with nervous cats), home weight checks can be employed coupled with phone consultations, with owners either using bathroom scales or luggage scales (*e.g.*, weighing the cat in its transport box). However determined, the current weight should be compared against the pet's healthy weight, and where a deviation of 5% or more is flagged, strategies can be implemented to help restore the healthy weight.

Promoting maintenance of a healthy weight and lifestyle

Various strategies can be used to prevent obesity in at-risk individuals, which broadly involve either controlling energy intake or increasing energy use.

© Shutterstock

Figure 5. Measuring cups are often utilized by owners to measure their pet's food, but although quick and easy to use, they lead to variable portion sizes and predispose to overfeeding.

- **Controlling energy intake.** This centers around the main meal offered. A nutritionally complete and balanced food should be fed, appropriate for the life stage of the animal. The choice is a matter for discussion between owner and veterinary professional, and the clinician is directed towards various reputable textbooks for further detail. Many pets exhibit strong food-seeking behaviors which owners often find difficult to refuse. In such cases, using foods with increased amounts of protein and fiber will improve satiety, thereby reducing voluntary food intake and food-seeking behaviors. Other strategies include decreasing the energy density of food by expanding its volume, either by adding water (or switching to a wet food if cost effective) or expanding a kibbled food with air. Finally, choosing a kibbled diet with a shape that requires more chewing will help slow food intake.

  Whatever diet is selected, it is important to feed the correct amount, which will vary according to the food and life stage. This can be determined by calculating the pet's maintenance energy requirements, or from the manufacturer's guidelines, adjusted to individual circumstances (*e.g.*, body weight, breed, sex, neuter status, activity level). The daily amount should be measured out accurately (see below) and fed for two weeks before reviewing. If weight has been lost in this time, food intake should be increased by 10%, or decreased by 10% if weight has been gained. Further cycles of weighing and adjustment should be continued until the body weight is stable. Thereafter, continue to weigh the animal at regular intervals to ensure that its weight remains on track.

- **Accurate portion size measurement.** Food portions must be measured out as precisely and accurately as possible, especially with dry diets, as their energy density means that even small over-estimates can mean significant overfeeding. Measuring cups to estimate the amount fed are simple but unreliable, leading to variable portion allocations and predisposing to overfeeding (9), so electronic scales are to be preferred **(Figure 5)**. Accurate portion measurement should become easier in future with the development of "smart bowls" and computer-controlled food hoppers, which will automatically measure out the correct daily portion. Some devices may also monitor food consumption throughout the day, enabling owners to chart patterns of food intake and making it easier to spot disruptions which might indicate a health issue **(Figure 6)**.

- **Responsible feeding of additional food items.** Although there is an association between the development of obesity and the feeding of additional food items such as table scraps and treats, owners are often unaware as to how much additional energy these food items contribute to their pet's daily intake, so a prevention program should include control over this. If dry food is fed, part of the daily ration can be reserved and used as treats. If other food is involved, the amount should be limited to a maximum of 10% of the total daily calorie ration, with the main meal portion reduced accordingly.

- **Method and pattern of feeding.** Most dog owners, and many cat owners, feed their pet 1-2 meals per day from a bowl, but this allows for rapid consumption and consequently many hours without food, which can lead to hunger and increased food-seeking behavior. Some cat owners leave (usually dry) food out all day, either as an alternative to, or in addition to, meal feeding, but this is a known risk factor for the development of obesity. Veterinary professionals should recommend the use of puzzle feeders or modified feeding bowls which slow food intake;

© Shutterstock

Figure 6. Electronic food hoppers which automatically measure out the correct daily portion can help with weight control, and some devices can also monitor food consumption throughout the day, allowing an owner to chart their pet's feeding pattern.

this can reduce the risk of over-eating (since the physiological "satiety signals" from the gastrointestinal tract take time to be released), and makes the feeding period last longer and more enjoyable for the pet.

- **Increasing energy expenditure.** Part of any prevention program will usually involve a review of physical activity levels, but in fact this only moderately affects an animal's overall energy expenditure. On average, walking an extra 1000 steps only increases energy expenditure by approximately 1 kcal per $kg^{0.75}$, although there are other benefits, such as improving cardiovascular and musculoskeletal fitness and strengthening the owner-pet bond. The exact amount of physical activity should be tailored to the individual, and must take into account any concurrent medical concerns. For dogs, at least one daily walk of 30 minutes is recommended, but longer and more frequent is better, and extra play sessions can also be beneficial. If possible and safe to do so, allowing a cat access to outdoors is recommended, and they will also benefit from short play sessions, typically 1-2 minutes at a time at least twice daily.

Have better obesity conversations

Many veterinary professionals are reluctant to hold conversations about obesity with pet owners, possibly because it is a highly stigmatized condition. It is therefore important to tackle the issue of weight stigma within the profession and, in so doing, improve communication regarding obesity. Because weight stigma can be unconscious, a veterinarian may approach a case without being aware of the effect that this bias can have on the outcome, and it may be useful for clinic personnel to quickly check their own implicit weight bias (25). This may help with obesity management, for example by enabling the clinician to actively adjust both the advice they give to owners of pets with obesity and the way they communicate with them to ensure that their clinical recommendations are consistent. Indeed, a key aspect of addressing weight stigma is to focus on better communication with owners whose pets have obesity. Appropriate training is vital for such situations, and all conversations should be supportive and non-judgmental, using empathic non-stigmatizing terms. Care should be taken not to assign (or appear to assign) blame to the owner, as this is likely to be counterproductive, and "toxic" terms such as "obese" and "fat" must be avoided, since owners can find them uncomfortable and might be offended, which will do little to encourage them to address the issue. A "patient-first language" is recommended in human medicine, and a similar strategy can be used for pet obesity, so the choice of words is all-important. As the name suggests, the clinician should ensure that the patient comes first in the conversation, and should not refer to it as "being obese" or use phrases such as "an obese dog" or saying a dog "is obese". One would not refer to a "cancerous dog" or say a dog "is cancer" so it is better to refer to a pet "with obesity" or say a pet "has obesity". Although this change might appear to be trivial, this phraseology avoids labelling the patient.

Nonetheless, it can be tricky to find a way of introducing the topic during a consultation, not least if the owner has brought their pet for another reason (*e.g.*, an unrelated illness or a routine vaccination). One strategy is to "talk about something else", perhaps broaching the subject in terms of changes in weight and body condition. For example, if the clinic regularly records body weight (as discussed above in terms of obesity prevention), deviations from the animal's "healthy adult weight" can be highlighted to the owner rather than discussing "obesity" – and again the choice of words is significant. Another strategy would be to use body condition (especially if BCS charts are displayed in the consult room) with the owner invited to assess their dog, along with guidance from the veterinarian. Talking about something else enables weight management to be discussed without ever using the term "obesity", even though both owner and professional will know that this is the issue at stake.

However, it is raised, and prior to discussing the topic in terms of causes and solutions, it is sensible to consider first asking permission to ascertain that the owner is comfortable discussing the topic (*e.g.*, *"We have identified that Fluffy is currently above a healthy weight. Would you be comfortable with us discussing this and what we can do to help?"*). Such a strategy emphasizes that the owner is in control and can facilitate acceptance of a weight management plan.

"In one study, veterinarians admitted to using stigmatizing terms to describe excess weight in dogs, and reported feeling blame, frustration, and disgust towards both dogs with obesity and their owners."

Alexander J. German

Classifying obesity as a disease

There have been recent moves to classify obesity as a disease, not least because it fits the formal definitions of disease (26). Although some argue that obesity is a normal physiological response to excess energy intake (7), there is significant scientific evidence to suggest that it is actually a pathological process, with over 20 national and international veterinary organizations supporting such a formal categorization. Classifying obesity as a disease may have a positive effect on how veterinary professionals manage pets with obesity – for example, when discussing causes of obesity, rather than only focusing on owner factors, the complexity of the disease pathogenesis can be emphasized, including other aspects such as genetic factors. This makes it easier for the clinician to be non-judgmental in their discussions with the client, thereby gaining their trust and increasing the chances that they will be receptive to weight management advice.

CONCLUSION

Pet obesity is currently a growing concern, with a changing picture in terms of the demographics of patients that are affected. Although weight management protocols are well-established, these are far from perfect and are often not even implemented by veterinary professionals. This is not helped by the fact that obesity is a highly stigmatized condition and often an awkward topic for discussion. Although there are no simple solutions, clinicians can be more effective in managing this problem, by considering short-term weight loss plans, by preventing obesity in young animals, and by more effective conversations about obesity. As with the human condition, formally classifying obesity as a disease could be a trigger to kick-start more supportive and effective obesity care from the veterinary profession.

REFERENCES

1. Association for Pet Obesity Prevention. Pet Obesity survey results 2017-2018. [accessed 6th July 2021]. Available from: https://petobesityprevention.org
2. Banfield® Pet Hospitals. Obesity in dogs and cats – state of pet health report [accessed July 6th 2021]. Available from: https://www.banfield.com/state-of-pet-health/obesity
3. Rowe E, Browne W, Casey R, *et al*. Risk factors identified for owner-reported feline obesity at around one year of age: dry diet and indoor lifestyle. *Prev. Vet. Med.* 2015;121:273-281. DOI: 10.1016/j.prevetmed.2015.07.011
4. German AJ, Woods GRT, Holden SL, *et al*. Dangerous trends in pet obesity. *Vet. Rec.* 2018;182:25. DOI: 10.1136/vr.k2
5. Such ZR, German AJ. Best in show but not best shape: a photographic assessment of show dog body condition. *Vet. Rec.* 2015;177:125: DOI: 10.1136/vr.103093
6. White GA, Hobson-West P, Cobb K, *et al*. Canine obesity: is there a difference between veterinarian and owner perception. *J. Small Anim. Pract.* 2011;52:622-666. DOI:10.1111/j.1748-5827.2011.01138.x
7. Hargreaves R. 'We shouldn't classify pet obesity as a disease'. *Vet. Rec.* 2019;185:667.
8. Yaissle JE, Holloway C, Buffington CA. Evaluation of owner education as a component of obesity treatment programs for dogs. *J. Am. Vet. Med. Assoc*. 2004;224:1932-1935.
9. German AJ, Holden SL, Mason SL, *et al*. Imprecision when using measuring cups to weigh out extruded dry kibbled food. *J. Anim. Physiol. Anim. Nutr. (Berl.)* 2011;95:368-373. DOI: 10.1111/j.1439-0396.2010.01063.x.
10. German AJ, Titcomb J, Holden SL, *et al*. Cohort study of the success of controlled weight loss programs for obese dogs. *J. Vet. Intern. Med*. 2015;29:1547-1555. DOI: 10.1111/jvim.13629
11. O'Connell EM, Williams M, Holden SL, *et al*. Factors associated with overweight cats successfully completing a diet-based weight loss program: an observational study. *BMC Vet. Res.* 2018;14:397. DOI: 10.1186/s12917-018-1740-5
12. German AJ, Holden SL, Morris PJ, *et al*. Long-term follow-up after weight management in obese dogs: the role of diet in preventing regain. *Vet. J*. 2012;192:65-70.
13. Deagle G, Holden SL, Biourge V, *et al*. Long-term follow-up after weight management in obese cats. *J. Nutr. Sci*. 2014;3:e25; DOI: 10.1017/jns.2014.36
14. Rolph NC, Noble PJM, German AJ. How often do primary care veterinarians record the overweight status of dogs? *J. Nutr. Sci.* 2014;3:e58. DOI: 10.1017/jns.2014.42
15. Puhl RM, Heuer CA. Obesity stigma: important considerations for public health. *Am. J. Public Health* 2010;100:1019-1028.
16. Riveros-McKay F, Mistry V, Bounds R, *et al*. Genetic architecture of human thinness compared to severe obesity. *PLOS Genetics* 2019; doi.org/10.1371/journal.pgen.1007603
17. Pearl RL, Wadden TA, Bach C, *et al*. Who's a good boy? Effects of dog and owner body weight on veterinarian perceptions and treatment recommendations. *Int. J. Obes. (Lond.)* 2020; Pub online ahead of print; DOI: 10.1038/s41366-020-0622-7.
18. Bland IM, Guthrie-Jones A, Taylor RD, *et al*. Dog obesity: owner attitudes and behaviour. *Prev. Vet. Med.* 2010;92:333-340.
19. Sutin AR, Stephan Y, Terracciano A. Weight discrimination and risk of mortality. *Psychol. Sci.* 2015;26:1803-1811.
20. Flanagan J, Bissot T, Hours MA, *et al*. Success of a weight loss plan for overweight dogs: the results of an international weight loss study. *PLOS One* 2017;12:e0184199. DOI: 10.1371/journal.pone.0184199
21. Serisier S, Feugier A, Venet C, *et al*. Faster growth rate in *ad libitum*-fed cats: a risk factor predicting the likelihood of becoming overweight during adulthood. *J. Nutr. Sci.* 2013;2;e11. DOI:10.1017/jns.2013.10
22. Salt C, Morris PJ, Butterwick RF, *et al*. Comparison of growth patterns in healthy dogs and dogs in abnormal body condition using growth standards. *PLOS One* 2020;15:e0238521. DOI: 10.1371/journal.pone.0238521
23. https://www.waltham.com/resources/puppy-growth-charts Accessed 6th July 2021
24. Salt C, Morris PJ, Wilson D, *et al*. Association between life span and body condition in neutered client-owned dogs. *J. Vet. Intern. Med.* 2019;33:89-99. DOI: 10.1111/jvim.15367
25. https://implicit.harvard.edu/implicit/ Accessed 6th July 2021
26. American Medical Association House of Delegates. Recognition of obesity as a disease. Resolution 420 (A-13). www.npr.org/documents/2013/jun/ama-resolution-obesity.pdf?t=1544195051473

CALCIUM AND PHOSPHORUS – GETTING THE BALANCE RIGHT

It is critical to ensure that a puppy is neither over- nor under-supplied with certain nutrients and minerals during the growth phase, but this can be more difficult than it first appears, as discussed in this paper.

KEY POINTS

1. An adequate supply of calcium and phosphorus (both absolute and relative to each other) is crucial to ensure a dog's healthy skeletal development.
2. Regular weighing and comparison with recommended growth curves is the gold standard to monitor optimal energy supply in puppies, as body condition scoring is not suitable.
3. Calcium and phosphorus requirements for growing dogs depend on the expected mature body weight and age of the individual.
4. Excess protein intake in puppies does not cause problems, but energy supply should be restricted to ensure optimal growth rates and to help avoid developmental orthopedic disease.

Introduction

Developmental orthopedic diseases (DODs) such as osteochondroses, joint dysplasia, osteodystrophy or bone deformities are frequently seen in dogs, with fast-growing large and giant breeds being at particular risk. The maturation phase is a major life stage for all species, but is of critical importance in fast-growing young animals that have significant skeletal vulnerability during this period, and dogs come into this category. Feeding a balanced diet that provides adequate amounts of energy and nutrients is the basis for a healthy musculoskeletal system, as deficiencies can exacerbate some conditions that are not primarily nutrition-related. Signs of DOD can vary from mild and transient lameness through to severe movement disorders, and can also involve painful deviation of one or more limbs and swelling of the joints. Malnutrition in the first few months of life can lead to lifelong impairment of an animal´s health.

How much is too much?

A major problem when feeding a growing puppy is to determine the optimal amount of energy required for that individual. Excessive energy intake leads to an animal being acutely overweight (*i.e.*, a growth rate that is faster than recommended), which places an additional burden on the growing skeleton (1). Even with some orthopedic disorders that have a hereditary basis, for example hip dysplasia **(Figure 1)**, energy restriction at the puppy stage can reduce the prevalence of these conditions (2). It is now known that a puppy which grows rapidly due to excess energy intake has a much greater risk of being overweight or obese in adulthood (3). Several factors, such as breed, activity level, housing and health status, will influence daily energy requirements, so the required correct calorie level can therefore vary considerably between individuals. In adult dogs, the recommended way to monitor if excess or insufficient energy is being supplied is to use body condition scoring (BCS); identifying an adult dog with a high BCS and excess body fat will indicate that the animal's diet has a disproportionately high energy content. This is not the case during the puppy stage, where a high-calorie diet can lead to an increased growth rate, and the dog can be overweight without having additional fat deposits. In fact, such puppies may even appear thin and undernourished, yet still weigh too much. The littermates in **Figure 2** impressively demonstrate the impact of energy supply during growth: both dogs have the same body

Linda Böswald

Dip. ECVCN, Ludwig-Maximilian University, Munich, Germany

Dr. Böswald studied veterinary medicine at Munich's Ludwig-Maximilian University and remained there after qualifying to specialize in animal nutrition. She completed her doctoral thesis on comparative aspects of mammalian calcium and phosphorus metabolism in 2018, and is currently continuing her research in this area. Following an internship and residency she gained her Diploma from the European College of Veterinary and Comparative Nutrition (ECVCN) in 2020.

Britta Dobenecker

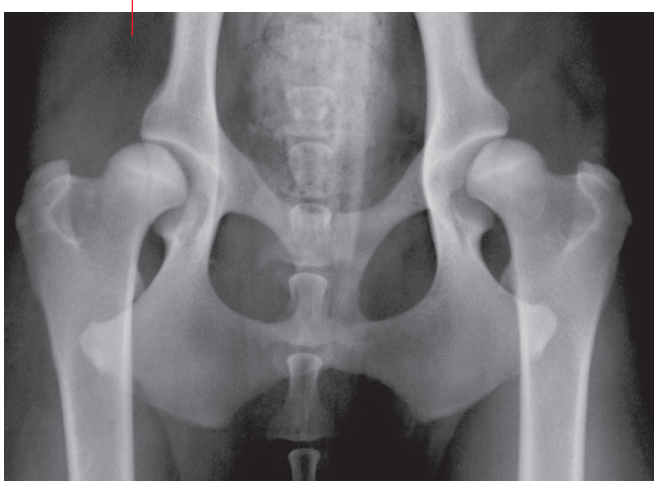
Dip. ECVCN, Ludwig-Maximilian University, Munich, Germany

After graduating from Hannover's School of Veterinary Medicine, Dr. Dobenecker studied for a doctoral thesis at the Institute of Physiological Chemistry in Hannover and the Institute of Animal Nutrition in Munich. She is a board-certified specialist in animal nutrition and dietetics (national level, European College of Veterinary and Comparative Nutrition (ECVCN)). She currently is an Academic director at the Institute of Animal Nutrition and Dietetics at the Ludwig-Maximilians University, with her main research interests centered around calcium and phosphorus and their impact on skeletal and renal health in dogs and cats.

condition score, but the animal on the left, which received a higher energy intake from the age of 8 weeks onwards, is clearly larger yet still lean in appearance.

An increased growth rate and the extra weight this brings represent additional stress for the developing skeleton, so the gold standard for determining an optimal growth rate is to monitor a puppy's body weight through regular weighing (*e.g.*, weekly) and compare with a growth chart (4). Optimal growth curves will differ depending on the anticipated ideal weight of the adult dog, therefore it is crucial to estimate this as accurately as possible.

Figure 1. A radiograph showing severe bilateral hip dysplasia in a 14-month-old Hovawart. Although many orthopedic problems in young dogs have a hereditary basis, restricting energy intake at the puppy stage has been shown to reduce the incidence of such conditions.



It is important to note that on-pack pet food recommendations for daily energy allowances can be rather high, and in addition calculating the calorie requirements using the 2006 equation from the US National Research Council (NRC) (5) will tend to give an overestimate. Recent studies have demonstrated significantly lower energy requirements in both growing colony dogs and household puppies (4,6) and the latest updated recommendations take this into account[1]. The average daily energy requirement (metabolizable energy, ME) for growing dogs can be estimated as follows (4):

$$\text{ME intake [MJ]} = 1.063\text{-}0.565 \times \frac{\text{actual BW}}{\text{expected mature BW}} \times \text{actual BW}^{0.75}$$

Dietary calcium and phosphorus

The two major constituents of bone, calcium and phosphorus, are linked by a functional and regulatory relationship, and are best considered together. The absolute requirements of both minerals, as well as the ratio (the recommended range is between 1:1 and 2:1), should be carefully calculated, as an excess or a deficiency of one or both minerals during the developmental phase can be a major factor in terms of nutritionally induced DODs. In retrospective evaluations of cases seen by the authors at Ludwig-Maximilian University, the majority of growing dogs with signs of DOD were found to have either an excessive or deficient intake of calcium and/or phosphorus, although interestingly the distribution has changed over the years. The first survey (in 1998) revealed that 61% of affected puppies had experienced a calcium excess and only 20% had a deficient intake of this

[1] https://fediaf.org/self-regulation/nutrition.html

© B. Dobenecker

Figure 2. Two growing Foxhound-crossbred littermates; the male dog (right) had been fed with a dietary energy supply to allow for growth according to the recommended growth curve, whilst the female (left) was given a diet that had excess energy levels. The BCS in both cases was 5/9.

element (1). A second survey in 2018 showed that the majority of DOD patients (58%) were fed a diet deficient in calcium, with only 21% of cases receiving excessive amounts of calcium prior to the orthopedic diagnosis (7).

It can be questioned as to why poor nutrition is such an issue for canine growth when it is much less of a problem in humans? One explanation is that in contrast to humans, maturation occurs much earlier in dogs, with almost the entire growth phase being realized within a single year.

"A number of factors, such as breed, activity level, housing and health status, influences daily energy requirements, so the required correct calorie level can therefore vary considerably between individuals."

Britta Dobenecker

Consequently, even limited periods of inadequate nutrient supply pose a disproportionately greater risk for puppies than for children and teenagers. But another difference between dogs and other species such as humans must be taken into account: adaptive mechanisms to compensate for low or excessive calcium and phosphorus supply are virtually non-existent in the canine species (8). Rather than intestinal absorption of both minerals being the major regulatory pathway, the skeletal system is employed to store and mobilize calcium and phosphorus as necessary (9,10). Viewed from the idea that the canine species evolved as a predator, this makes perfect sense: either there was prey available, supplying abundant energy and minerals, or both energy and minerals were scarce, so that energy-dependent up-regulation of intestinal absorption was not feasible. This bone-based regulation is one factor in the development of clinical signs linked to calcium and phosphorus malnutrition in growing and adult dogs.

Deficient calcium intake during the growth phase can cause secondary nutritional hyperparathyroidism, with reduced overall bone formation resulting in various clinical signs, including pathological fractures from normal physical exertion (11). In this context it is important to note that an increased fecal dry matter excretion (due either to a low dietary digestibility or a high food intake) also increases the fecal losses of calcium and phosphorus (12-14) and therefore increases the daily requirements for the minerals. As noted above, there is a considerable and increasing number of puppies diagnosed with DOD that are fed a calcium deficient diet. However, calcium excess receives more attention in the literature: several publications report that DOD can be caused experimentally in growing Great Danes by feeding excess dietary calcium (15,16) – although in most of these trials only the calcium supply was increased above the recommended intake; this led to reduced utilization of other elements crucial to skeletal development, such as phosphorus, zinc and copper. Consequently, such DODs may be caused, or at least be aggravated by, a secondary deficiency of these minerals, and in particular phosphorus (17). This tallies with the results of a study that examined the effects of excess dietary calcium when surplus phosphorus was also provided (to allow for a reduced digestibility of this element) in medium and large dog breeds, with no clinical signs of DOD being noted (18). On the contrary, a low phosphorus intake has been shown to have substantial (but reversible) adverse effects on skeletal health (19,20) **(Figure 3)** and other developmental disorders described in various case reports may also be linked to dietary phosphorus deficiency (21,22). It is also worth noting that, although supplying dietary protein at levels above the recommended daily allowance is often suspected to have a detrimental impact on skeletal development, this can be classified as a myth. Studies have demonstrated that the level of protein fed to growing dogs has no effect on their skeletal development (23,24).

© B. Dobenecker

Figure 3. A Foxhound-crossbred puppy after 6.5 weeks of low dietary phosphorus intake with bowed legs and splayed paws. The abnormalities were painless and completely reversible after a couple of weeks of adequate phosphorus supply.

How are daily nutrient requirements calculated?

When recommending daily allowances which will meet the requirements of the majority of the population, the basic approach is to determine the net requirements of each nutrient at various life stages, along with how bioavailability varies depending on the nutrient source. There are several ways to estimate these requirements. Experiments to study the dose-effect relationship of a particular nutrient can elucidate what happens with insufficient, adequate and excessive supply, but such trials need to be highly standardized, with careful selection of suitable target parameters to assess the effect of the nutrient being studied. For calcium and phosphorus, defining the parameters is rather difficult. On the one hand, there may be a prolonged latency period between onset of malnutrition and the appearance of specific clinical signs. The homeostasis of calcium and phosphorus is tightly controlled, so serum concentrations are maintained for a long time, even during severe deficiency. On the other hand, factors such as the ratio of dietary calcium to phosphorus, energy intake, growth rate and any genetic predisposition to skeletal disease are known to exacerbate the clinical signs. The calcium and phosphorus requirements for growing dogs, as defined by the NRC (5), are based on research using large and giant breeds and mainly in puppies younger than 6 months of age, with the trial data then extrapolated to cover the entire growth period of dogs of all sizes. The aim was to "meet or exceed" the requirement of all growing dogs, so it made sense to deduce the recommendations from the group with the highest requirement, *i.e.*, young giant breed puppies. However, this method does not take into account individual differences, and the critical period of maximum growth intensity differs in duration between small and large breeds of dog. While large and giant breeds grow up to 12-15 months of age, small breeds may have reached their final bodyweight by 7-8 months, so extrapolation of data is likely to overestimate the requirement of small breeds. In addition, deducing the requirement with data from the early growth period will overestimate the requirement for the later period of growth, where daily gains have slowed.

Nutritional calculations

Another approach for determining the optimal daily supply of a nutrient is to perform a factorial calculation, which summates the required amounts for maintenance and performance. The advantage with this method is that it takes into account the precise individual requirements for a selected life stage, and it has been used successfully with farm animals, for example to determine the optimal rations for milk production in dairy cattle and for fattening pigs. In growing dogs, the factors to be determined are the tissue gain during growth and the maintenance requirement for the current body weight (25). The maintenance requirement is the amount of a nutrient needed to compensate for inevitable endogenous losses from feces, urine, sweat, skin, fur, etc.; when the endogenous losses are quantified, this gives the "net maintenance requirement" of a nutrient. However, because not 100% of the ingested amount of a nutrient are taken up by the body, the actual availability needs to be taken into account in order to obtain the "gross maintenance requirement" or feeding recommendation (26).

"The calcium and phosphorus requirements for growing dogs are based on research using large and giant breeds and mainly in puppies younger than 6 months of age, with the data generated from those trials being extrapolated to cover the complete growth period of dogs of all size groups."

Linda Böswald

Table 1. Requirement for calcium and phosphorus for growing dogs relative to metabolic body weight ($kg^{0.75}$) (adapted from 26).

Mature body weight (kg)	10	35	60
Current age (weeks)	Calcium (mg/$kg^{0.75}$)		
9	550	–	–
13	436	634	776
17	361	512	610
22	339	479	565
26	335	466	542
31	316	444	519
35	251	350	405
39	217	300	348
44	213	294	342
48	193	266	306
52	187	258	296
Current age (weeks)	Phosphorus (mg/$kg^{0.75}$)		
9	352	–	–
13	197	291	362
17	158	225	269
22	151	213	251
26	152	210	244
31	141	196	227
35	124	172	197
39	121	166	190
44	125	171	196
48	116	158	180
52	114	155	177

The equation is as follows:

$$\text{Feeding recommendation for maintenance} = \frac{e}{\text{availability [\%]}} \times 100$$

(where e = the endogenous losses)

Obviously, the overall feeding recommendation is strongly influenced by the availability of a nutrient; if the average availability is reduced by half, the feeding recommendation doubles. Mineral availability differs between sources and overall diet composition, so to ensure adequate intake levels a safety margin is built into the average availability.

Tissue gain during growth requires an additional nutrient allowance to be factored into the equation, as follows;

$$\text{Net requirement for growth} = \text{average daily gain} \times \text{nutrient concentration in gained tissue}$$

Box 1. Calculating the required calcium **(Ca)** and phosphorus **(P)** levels for "Max", a 22-week-old German Shepherd puppy with a current body weight of 20 kg and an estimated adult body weight of 35 kg.

1.

For Max at 22 weeks and with a predicted mature body weight of 35 kg, table one provides a daily recommendation of 479 mg Ca/$kg^{0.75}$ and 213 mg P/$kg^{0.75}$. Using his current weight of 20 kg, the absolute calcium and phosphorus requirements are calculated as follows:

- Recommended daily Ca intake: 479 mg x $20^{0.75}$ kg = 479 mg x 9.46 kg = 4,530 mg (or 4.5 g)
- Recommended daily P intake: 213 mg x $20^{0.75}$ kg = 213 mg x 9.46 kg = 2,014 mg (or 2.0 g)

2.

If using a commercial dry diet, it is essential to check whether the calcium and phosphorus content of the chosen diet is adequate for the puppy at his current growth period. Assuming a daily energy requirement of 7.0 MJ ME (1,673 kcal) (4), the required mineral content in the diet can be calculated as follows:

- Calcium content 4.5 g/7 MJ ME = 0.64 g Ca per MJ ME (2.69 g Ca/1000 kcal ME)
- Phosphorus content 2.0 g/7 MJ ME = 0.29 g P per MJ ME (1.20 g P/1000 kcal ME)

3.

The owner has chosen a balanced, complete dry diet for growing large breed dogs which contains 1.6 MJ ME (382 kcal) per 100 g, 1.1% calcium and 0.7% phosphorus, with a Ca:P ratio of 1.6:1. The first step is to calculate the daily amount of the diet that will provide the ME requirement for Max, as follows;

- 7 MJ ME/1.6 MJ ME x 100 g = 438 g

4.

The calcium and phosphorus supplied with this amount of diet then needs to be compared to the requirement. This can be calculated either relative to the dietary ME content (i), or as absolute values for the individual dog (ii).

(i) Relative to ME, the mineral content is calculated as follows:

- 1.1 g Ca per 100 g → 1.1 g Ca/1.6 MJ ME = 0.68 g Ca/MJ ME (2.88 g Ca/1000 kcal ME)
- 0.7 g P per 100 g → 0.7 g P per 1.6 MJ ME = 0.43 g P/MJ ME (1.83 g P/1000 kcal ME)

So the diet in this example meets the required levels of calcium and phosphorus per MJ ME (as calculated in step 2).

(ii) Alternatively, the absolute dietary supply with both minerals can be calculated as follows:

- 1.1% Ca in 438 g diet → 1.1 x 438/100 = 4.818 g calcium supplied with this amount
- 0.7% P in 438 g diet → 0.7 x 438/100 = 3.066 g phosphorus supplied with this amount

The values should be compared to the absolute requirement for Max (as calculated in step 1) and can be seen to be met in this case.

For the feeding routine, the daily ration can be weighed out and kept in a small container; this means that should the owner want to give Max treats for training, kibble from the measured portion can be used without the risk of additional energy supply **(Figure 4)**.

So the feeding recommendation for growth can be calculated from this, as follows;

$$\frac{\text{(e + net requirement for growth)}}{\text{availability [\%]}} \times 100$$

The canine species shows a uniquely high variation in body size and weight, so to generate the growth curve for an individual puppy it is important to have a good estimate of its future adult body weight (based on the ideal weight of the same-sex parent or, if this is not available, mean weight according to breed standard). There are also differences in growth rate between small, medium, large and giant breeds, so it is necessary to categorize dogs on their adult body weight to better define recommendations for calcium and phosphorus supply. In addition, since growth is not a linear process, using age groups allows further differentiation for the recommendations. The percentage requirement for tissue gain is highest during early growth; this then declines as the dog approaches its adult body weight and the maintenance requirement accounts for most of the total energy and nutrient requirements. The factorial calculation for calcium and phosphorus needs to take both aspects into consideration.

There are different ways to express feeding recommendations. The NRC supplies the values in relation to metabolizable energy (ME). The absolute value calculated using this reference depends on the energy requirement, which may differ considerably from the average for an individual case. It can be predicted, for example, that a Newfoundland puppy will have a lower ME requirement than a Great Dane puppy of the same age and body weight due to differences in temperament and activity. As an alternative to the reference ME, it is possible to relate the recommended daily intake to metabolic body weight ($kg^{0.75}$). This has the advantage of calculating the exact requirements – and hence enabling a precisely balanced diet – for an individual dog, but it does not directly inform about the required nutrient content in a diet. This needs to be calculated for each case using the individual ME requirement.

© Shutterstock

Figure 4. To help Max with his training, the owner should reward him with kibble taken from his calculated daily allowance.

© B. Dobenecker

Figure 5. There are a bewildering number of multivitamin and mineral supplements available, and the veterinarian should guide the owners to select a product appropriate to their dog's needs.

Table 2. A potential homemade diet formulated to meet the requirement of a 22-week-old German Shepherd puppy currently weighing 20 kg and with an estimated mature bodyweight of 35 kg.

Feed item	Amount [g/d]	Energy [MJ ME]	Ca [mg]	P [mg]
Beef	600	4.8	24	1188
Cooked potatoes	250	0.5	15	115
Vegetables	100	0.2	34	30
Fruit	100	0.2	11	16
Plant oil	15	0.5	0	0
Fish oil	5	0.2	0	0
Dried tripe as chew	30	0.6	27	54
Supplement	20	0	4500	2100
Total in ration		**7.0**	**4611**	**3503**
Recommended intake		**7.0**	**4530**	**2014**

The calcium and phosphorus recommendations for various ages and mature body weight groups required for the calculations are given in **Table 1**, and a worked example for a young puppy fed a commercial diet can help explain the different steps **(Box 1)**.

Should an owner wish to feed a homemade diet, it must be appreciated that most recipes need to be supplemented with minerals and vitamins to meet the daily requirements, and because there are various supplements available, the nutrient supply from all components of the diet must be added together and compared to the requirement to allow selection of a suitable product **(Figure 5)**. An example of a homemade ration is shown in **Table 2**, from which it can be seen that 20 g of a mineral supplement which contains 22.5% calcium and 10.5% phosphorus will meet the daily recommended allowance for these minerals. Note also that the dietary Ca/P ratio at 1.3/1 is in the ideal range (recommended range 1/1–2/1). However, in order to meet the requirements, the amount of daily supplement offered must be increased if the nutrient concentration is lower, or decreased if the nutrient supply is higher.

CONCLUSION

Metabolizable energy and nutrient requirements change during growth, so a proper overview of a puppy's nutritional requirements requires regular ration adaptations. Whilst two-monthly reviews are supposedly adequate for dogs growing according to the recommended growth curve, all puppies should be weighed weekly, and the body weight compared to the optimal growth curve to monitor progress. If there is a deviation from the ideal body weight, the diet – and especially the ME supplied – needs to be adapted immediately. For simplicity, this article considers only metabolizable energy, calcium and phosphorus, but in general the requirement for all minerals, trace elements and vitamins should be included when reviewing the nutritional needs of a patient, and whilst the calculations are not difficult, they do illustrate that consideration of all the factors required to achieve a balanced diet is necessary.

REFERENCES

1. Dobenecker B, Kienzle E, Köstlin R, *et al*. Mal- and over-nutrition in puppies with or without clinical disorders of skeletal development. *J. Anim. Physiol. Anim. Nutr.* 1998;80(1-5):76-81.
2. Kealy RD, Olsson SE, Monti KL, *et al*. Effects of limited food consumption on the incidence of hip dysplasia in growing dogs. *J. Am. Vet. Med. Assoc.* 1992;201:857-857.
3. German AJ. Promoting healthy growth in pets. (Abstract) International Nutritional Science Symposium, Atlanta, USA, 2016.
4. Klein C, Thes M, Böswald LF, *et al*. Metabolisable energy intake and growth of privately owned growing dogs in comparison with official recommendations on the growth curve and energy supply. *J. Anim. Physiol. Anim. Nutr.* 2019;103(6):1952-1958.
5. National Research Council (NRC). Nutrient Requirements of Dogs and Cats. National Academic Press, Washington D.C. 2006
6. Dobenecker B, Endres V, Kienzle E. Energy requirements of puppies of two different breeds for ideal growth from weaning to 28 weeks of age. *J. Anim. Physiol. Anim. Nutr.* 2013;97(1):190-196.
7. Böswald L, Elmiger C, Dobenecker B. Nährstoffversorgung in der Aufzucht von Hunden – tierschutzrelevante Befunde und Fallbeispiele. In *Proceedings*, DVG Tierschutztagung München 2018.
8. Mack JK, Alexander LG, Morris PJ, *et al*. Demonstration of uniformity of calcium absorption in adult dogs and cats. *J. Anim. Physiol. Anim. Nutr.* 2015;99(5):801-809.
9. Böswald LF, Dobenecker B, Clauss M, *et al*. A comparative meta-analysis on the relationship of faecal calcium and phosphorus excretion in mammals. *J. Anim. Physiol. Anim. Nutr.* 2018;102(2):370-379.
10. Schmitt S, Mack J, Kienzle E, *et al*. Faecal calcium excretion does not decrease during long-term feeding of a low-calcium diet in adult dogs. *J. Anim. Physiol. Anim. Nutr.* 2018;102(2):e798-e805.
11. Becker N, Kienzle E, Dobenecker B. Calcium deficiency: a problem in growing and adult dogs: two case reports. *Tierarztliche Praxis. Ausgabe K, Kleintiere/heimtiere* 2012;40(2):135-139.
12. Kienzle E, Dobenecker B, Wichert B, *et al*. Effect of fecal water and dry matter excretion on fecal mineral excretion in dogs studied in a fiber model. *J. Nutr.* 2006;136(7):2001S-2003S.
13. Kienzle E, Brenten T, Dobenecker B. Impact of faecal DM excretion on faecal calcium losses in dogs eating complete moist and dry pet foods – food digestibility is a major determinant of calcium requirements. *J. Nutr. Sci.* 2017;6(3):1-3.
14. Kienzle E, Hofmann C, Dobenecker B. The increase of faecal calcium excretion with increasing faecal dry matter excretion in dogs is an independent *per se* effect of faecal dry matter excretion. In: *Proceedings*, Congress of the European Society of Veterinary and Comparative Nutrition Turin, Italy, 2019.
15. Hazewinkel HAW, Goedegebuure SA, Poulos PW, *et al*. Influences of chronic calcium excess on the skeletal development of growing Great Danes. *J. Am. Anim. Hosp. Assoc.* 1985;135:305-310.
16. Schoenmakers I, Hazewinkel HAW, Voorhout G, *et al*. Effect of diets with different calcium and phosphorus contents on the skeletal development and blood chemistry of growing Great Danes. *Vet. Rec.* 2000;147(23):652-660.
17. Dobenecker B. Factors that modify the effect of excess calcium on skeletal development in puppies. *Br. J. Nutr.* 2011;106(S1):S142-S145.
18. Dobenecker B, Kasbeitzer N, Flinspach S, *et al*. Calcium-excess causes subclinical changes of bone growth in Beagles but not in Foxhound-crossbred dogs, as measured in X-rays. *J. Anim. Physiol. Anim. Nutr.* 2006;90(9-10):394-401.
19. Kiefer-Hecker B, Kienzle E, Dobenecker B. Effects of low phosphorus supply on the availability of calcium and phosphorus, and musculoskeletal development of growing dogs of two different breeds. *J. Anim. Physiol. Anim. Nutr.* 2018;102(3):789-798.
20. Kiefer-Hecker B, Bauer A, Dobenecker B. Effects of low phosphorus intake on serum calcium, phosphorus, alkaline phosphatase activity and parathyroid hormone in growing dogs. *J. Anim. Physiol. Anim. Nutr.* 2018;102(6):1749-1758.
21. Fritz J, von Rosenberg S, Bensinger K, *et al*. Two cases of malnutrition associated with locomotor problems in growing puppies without alterations of x-ray density of long bones. In: *Proceedings*, Congress of the European Society of Veterinary and Comparative Nutrition, Vienna, Austria, 2008.
22. Cetinkaya MA, Yardimci C, Sağlam M. Carpal laxity syndrome in forty-three puppies. *Vet. Comp. Orthop. Traumatol.* 2007;2(02):126-130.
23. Nap RC, Hazewinkel HA, Voorhout G, *et al*. Growth and skeletal development in Great Dane pups fed different levels of protein intake. *J. Nutr.* 1991;121(suppl_11):S107-S113.
24. Nap RC, Hazewinkel HA, Voorhout G, *et al*. The influence of the dietary protein content on growth in giant breed dogs. *Vet. Comp. Orthop. Traumatol.* 1993;6(01):1-8.
25. Kienzle E, Zentek J, Meyer H. Body composition of puppies and young dogs. *J. Nutr.* 1998;128(12):2680S-2683S.
26. Böswald LF, Klein C, Dobenecker B, *et al*. Factorial calculation of calcium and phosphorus requirements of growing dogs. *PLOS One* 2019;14(8):e0220305.

veterinary focus #32.1

The worldwide journal for the companion animal veterinarian

© Shutterstock © Shutterstock © Shutterstock © Smadar Tal/ Koret School of Veterinary Medicine

COMING UP...

In our next issue, we will look at various start of life topics.

- DHA (docosahexaenoic acid) and neural development in puppies
  Russ Kelley, USA
- Kitten kindergartens
  Kersti Seksel, Australia
- Milk replacements – when, what and how
  Emmanuel Fontaine, USA
- Antibiotic use in young animals
  Scott Weese, Canada
- Vaccination and immune response in kittens
  Kelly St. Denis, Canada
- Growth charts: how to use them
  Caitlin Grant, Canada
- Kitten-friendly consults
  Liz O'Brien, Canada
- Amniocentesis in dogs
  Smadar Tal, Israel

ROYAL CANIN®

veterinary focus #31.3

Origine du papier : VIRTON (Belgique)
Taux de fibres recyclés : 0%
Certification : 100% PEFC
Impact sur l'eau : 0.012 P tot kg/tonne

PEFC 10-31-1668

Deputy publisher: Buena Media Plus
Chairman: Julien Kouchner;
CEO: Bernardo Gallitelli
11-15, quai De Dion-Bouton
92800 Puteaux, France
Phone: +33 (0) 1 76 21 91 78
Editor-in-chief: Ewan McNeill, BVMS, Cert VR, MRCVS
Editorial secretary
- Laurent Cathalan (laurent.cathalan@1health.fr)

Artwork
- Pierre Ménard

Printed in the European Union
ISSN 2430-7874
Legal deposit: November 2021
Cover: Royal Canin
Authors portraits: Manuel Fontègne
Veterinary Focus is published in Brazilian Portuguese, English, French, German, Italian, Polish, Russian, Spanish and Korean.
Find the most recent issues on: https://vetfocus.royalcanin.com and www.ivis.org.

The licensing arrangements for therapeutic agents intended for use in small animal species vary greatly worldwide. In the absence of a specific license, consideration should be given to issuing an appropriate cautionary warning prior to administration of any such drug.

We welcome offers to write ideas for papers and suggestions for topics and authors, which should be directed to the editor. *Veterinary Focus* is fully covered by copyright. No part of this publication may be reproduced, copied or transmitted in any form or by any means (including graphic, electronic or mechanical), without the written consent of the publishers © Royal Canin SAS 2021. Proprietary names (trademarks) have not been specially identified. It cannot, however, be conducted from the omission of such information that they are non-proprietary names and as such can be used by everyone. The publishers cannot take any responsibility for information provided on dosages and methods of application. Details of this kind must be checked for correctness by the individual user in the appropriate literature. While every effort has been made by the translators to ensure the accuracy of their translations, no responsibility for the correctness of the original articles and thus no resulting claims against professional negligence can be accepted in this connection. Views expressed by authors or contributors do not necessarily reflect the views of the publishers, editors or editorial advisors.

33rd

EUROPEAN VETERINARY DERMATOLOGY CONGRESS

ORGANIZED BY ESVD-ECVD

PORTO - PORTUGAL

29 SEPTEMBER - 1 OCTOBER 2022

SAVE THE DATE

29 SEPT - 1 OCT 2022

PORTO - PORTUGAL

SCIENTIFIC AND CONTINUING EDUCATION PROGRAMME

FREE COMMUNICATIONS AND POSTERS

ESVD European Society of Veterinary Dermatology

ECVD THE EUROPEAN COLLEGE OF VETERINARY DERMATOLOGY

DRN Quality without Compromise · Elanco · ICP Evolution of Knowledge · ROYAL CANIN · zoetis

THE LONG-TERM PARTNERS OF ESVD/ECVD

WWW.ESVD-ECVDCONGRESS.COM