CHAPTER

17

Nutritional Disorders

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OUTLINE

Providing nutrition to companion animals is relatively easy and safe; however, an adverse reaction to a diet or nutrient or exposure to a food hazard occasionally occurs. A complete and balanced diet is a diet that contains the appropriate ingredients in the appropriate amounts so that animals remain clinically healthy. It provides complete nutrition, and the nutrients are balanced with one another, especially the energy density of the diet. Nutritional disorders may result from imbalances in diet formulation or from specific food components.

FOOD COMPONENTS

Hazardous food components encompass dietary components that are present in the food. These may be components that should be present but are present in an unbalanced manner, or these may be components that should not be present. *Nutrient imbalances* may occur when there is a problem in the formulation or manufacture of a diet or if the owner supplements a complete and balanced diet with an incomplete and unbalanced food or supplements. Generic foods are more likely to be unbalanced and result in clinical disease.[26]

Excesses

Food components may be present in greater than recommended amounts. For example, consumption of energy in excess of expenditure is one potential mechanism for development of obesity (see Chapter 18). Other food components present in excess may pose serious health concerns and are discussed in the subsequent sections and in Table 17-1.

Hypervitaminosis A

Cats require preformed vitamin A in the diet because beta-carotene, the plant precursor of vitamin A, cannot be converted to vitamin A by cats (see Chapter 15).[6] Hypervitaminosis A is uncommonly seen but results in ankylosing spondylosis, particularly of the cervical vertebrae in cats; it can also induce growth retardation, abnormal dentition, and neurologic deficits as a result of nerve entrapment from hyperostoses.[10,16,44,51,52] It occurs when excessive vitamin A is present in the diet in the form of raw liver or cod liver oil or as a vitamin supplement.* Hyperostoses resulting from hypervitaminosis A include primarily cervical stiffness and forelimb lameness. Affected cats resist movement, particularly neck flexion. Clinical signs are attributed to new periosteal bone formation at sites of ligament and tendon attachment, which restrict joint movements and may impinge on nerves exiting vertebral foramina. With continued exposure to high levels of vitamin A, bony changes may extend to sternebrae, ribs, scapulae, other long bones, and pelvis. Ankylosis of cervical vertebrae and elbow joints may occur. Affected cats typically have an unkempt appearance because of an inability to groom. On presentation adult cats typically have a prolonged history of malaise, poor appetite, and a diet consisting mainly of liver or other concentrated source (or sources) of vitamin A. Physical examination often reveals muscle wasting, cutaneous hyperesthesia, inability to move neck, and a tendency to sit on the hindlimbs in a "kangaroo" position. Cervical radiography is diagnostic for ankylosing

*References 3, 10, 16, 44, 51, 52.

TABLE 17-1 Nutrient Excesses

Nutrient Class/Elements	Associated Diseases/Conditions
Energy	Obesity Increased risk for other diseases
Protein	May result in imbalanced or deficient diet
Carbohydrate	Lactose intolerance Diarrhea, bloating
Minerals	
Magnesium	Struvite-related urolithiasis and urethral plugs
Phosphorus	Secondary nutritional hyperparathyroidism
Sodium	Hypertension Congestive heart failure
Vitamins	
Vitamin A	Cervical osteocartilaginous hyperplasia
Vitamin D	Soft tissue calcification
Trace elements	
Iron	Vomiting, diarrhea, neurological signs
Copper	Chronic active hepatitis
Zinc	Hemolytic anemia
Iodine	Hyperthyroidism

spondylosis. Plasma vitamin A can be measured. Normal plasma vitamin A concentrations are 960 ± 770 ng/mL[2]; plasma concentrations of vitamin A in cats with hypervitaminosis A have been reported to be higher than 4500 ng/mL.[9,16,44,51] Treatment for hypervitaminosis A includes discontinuing the high vitamin A diet or supplement, changing the diet to one containing recommended vitamin A levels, and administering an analgesic and possibly antiinflammatory medication. If caught early, changing the diet may result in resolution of early ankylosis; however, when ankylosis has been present for some time, it will not resolve. Affected cats may have difficulty eating and drinking because of their inability to flex their neck. It may be necessary for the owner to provide food and water at a height that does not require neck flexion, or a feeding tube may be required.

Hypervitaminosis D

The dietary vitamin D requirement of adult cats is fairly low, although cats require a dietary source because sunlight is not required for activation of vitamin D.[46] Hypervitaminosis D is uncommon but may occur if complete diets are supplemented with vitamin D or when manufacturing errors occur. In 2006 a major pet food manufacturer recalled canine and feline canned diets because of excessive levels of vitamin D_3 contained in the vitamin–mineral premix. Affected cats developed gastrointestinal signs, hypercalcemia, and renal disease. More commonly, hypervitaminosis D results from ingestion of vitamin D containing rodenticides and causes an acute disease manifested as hypercalcemia, polyuria–polydipsia, muscle fasciculations, vomiting, diarrhea, anorexia, seizures, and possibly renal failure (see Chapter 31). Chronic hypervitaminosis D results in musculoskeletal deformities, although cats appear to be relatively resistant.[53]

Excessive Intake of Polyunsaturated Fatty Acids

Steatitis, a painful inflammatory condition of adipose tissue, may result from excessive intake of polyunsaturated fatty acids or ingestion of rancid fat. Hard, painful masses can be palpated in adipose tissue of affected cats.* Although this condition is uncommon, it may occur if the antioxidant activity of the food is not adequate, if the food is fed beyond the effective time of included antioxidants, or with homemade diets that are stored for long periods without added antioxidants. Fish oil is particularly susceptible to oxidation and requires higher levels of antioxidants compared with vegetable or animal fat sources. Treatment involves analgesic therapy, antioxidant supplementation, and possibly surgical excision of necrotic fat.

Deficiencies

Diets may be deficient in required nutrients, including macronutrients and micronutrients. Important deficiencies are described in the subsequent sections and in Table 17-2.

Thiamin

Thiamin is a B vitamin that is involved with neurologic function. Classic thiamin deficiency occurs with ingestion of large amounts of raw fish that contain thiaminase, an enzyme that destroys thiamin. Cooking the fish destroys thiaminase and eliminates the problem. Thiamin deficiency has been reported with sulfur dioxide preservation of dietary meat and in cats fed commercial cat food.[11,30,31,55,57] A small cluster of thiamin-deficiency cases associated with commercial cat foods occurred in the eastern part of the United States in 2009, and another limited precautionary recall of canned cat foods owing to inadequate thiamin content occurred in 2010. Clinical signs of thiamin deficiency include decreased food intake, hypersalivation, ventral flexion of the neck (see Figure 26-19), and seizures. Myocardial degeneration has also been associated with thiamin deficiency.[1] Fundic examination may reveal retinal venous dilation and hemorrhages. Treatment includes discontinuing the offending diet, changing to a complete and balanced cat food, and supplementing with thiamin (5 mg orally or 1 mg parenterally). Thiamin supplementation results in resolution of clinical signs, usually within 24 hours.

*References 6, 17, 27, 34, 37, 38, 62, 67.

TABLE 17-2 Nutrient Deficiencies

Nutrient Class/ Elements	Associated Diseases/Conditions
Energy	Malnutrition Poor growth and body condition
Protein	Poor hair coat Hypoproteinemia Edema/ascites Vacuolar hepatopathy
Taurine	Dilated cardiomyopathy Retinal degeneration Poor reproductive performance
Fat: linoleic, arachidonic	Poor hair coat Fat-soluble vitamin deficiencies
Minerals	
Calcium, phosphorus	Nutritional secondary hyperparathyroidism
Magnesium	Calcium oxalate urolithiasis (?) Cardiac dysfunction
Sodium	Poor appetite
Potassium	Polymyopathy
Vitamins	
Vitamin A	Dermatologic and ophthalmologic disease
Vitamin D	Rickets
Thiamine	Seizures
Niacin	Pellagra/black tongue
Biotin	Poor hair coat
Vitamin E	Pansteatitis
Trace elements	
Iron	Anemia
Copper	Anemia Depigmentation of skin
Zinc	Parakeratosis, poor hair coat
Iodine	Goiter, alopecia
Selenium	Muscular weakness

Vitamin E

Ingestion of large quantities of raw fish may also result in vitamin E deficiency. Fish contains polyunsaturated fatty acids that are easily oxidized. Vitamin E is an antioxidant that prevents oxidation of fatty acids in cell membranes. Signs of vitamin E deficiency include pansteatitis (discussed previously), decreased appetite, hyperesthesia, fever, and myositis.[13,15,64] Treatment consists of changing the diet with vitamin E supplementation (vitamin E acetate 100 mg/kg daily by mouth). Glucocorticoids may help decrease inflammation.

Taurine

Taurine is a beta-sulfonic amino acid that is an essential nutrient for cats, and deficiency has been associated with heart disease. Cats cannot make taurine from other amino acids and lose taurine in bile. Taurine is found primarily in animal-based products, and commercial cat foods have added taurine. Taurine deficiency occurs in cats when loss exceeds intake. This usually occurs when homemade vegetarian diets are fed or when cats are fed primarily dog food. In cats taurine deficiency is associated with retinal degeneration and blindness (see Figures 29-61 and 29-62), dilated cardiomyopathy (see Figure 18-1), reproductive problems, and abnormal skeletal development in kittens.* In pregnant queens taurine deficiency is associated with abortions, stillbirths, and birthing of kittens that do not survive. If neonatal kittens are born live to queens that are taurine deficient, they often have skeletal abnormalities, such as curved spines and small stature.

With dilated cardiomyopathy the diagnosis of taurine deficiency is made by radiography and echocardiography (see Chapter 20). In cats with retinal degeneration, a thorough ophthalmic examination should be performed (see Chapter 29). If reproductive problems are present, evaluation for other causes of decreased conception rates or stillbirths is undertaken (see Chapter 40). In kittens with skeletal abnormalities, radiography should be performed. In cats plasma taurine concentration can decrease below the normal range after less than 24 hours of fasting; therefore whole blood is preferred for evaluation of taurine levels.[22,36,54] Although taurine deficiency is now a rare cause of dilated cardiomyopathy in cats, taurine is inexpensive and safe and is often empirically administered (250 to 500 mg orally every 12 hours) for 8 weeks to patients with presumed deficiency. Commercial dry cat foods are required to contain 0.1% taurine on a dry matter basis, and canned foods are required to contain 0.2% on a dry matter basis. Canned diets are required to contain more taurine than dry diets because canned diets promote bacterial growth in the intestines that degrade taurine.

Inappropriate Food Components

Occasionally, foods and ingredients that are not toxic to other species may induce toxicity in cats. Examples include onion and garlic ingestion, which can cause Heinz body hemolytic anemia.[5,47] The primary toxic principle in members of the *Allium* genus is n-propyl disulfide, which causes oxidative damage to erythrocytes. Toxicoses have been reported with fresh, cooked, dried, or powdered material. Feeding commercial baby food containing onion powder has also been reported to cause toxicity in cats. Clinical signs include vomiting, weakness, and pallor. Affected cats may also have hemoglobinuria. Therapy involves inducing emesis and administering activated charcoal if ingestion is recent, along with supportive care. Patients with severe anemia may require whole blood transfusions, and fluid diuresis is recommended for cats with hemoglobinuria.

*References 19-21, 24, 42, 43, 48-50, 58-61.

FOOD CONTAMINANTS

Occasionally, food may become contaminated. This may occur if the manufacturer uses contaminated foodstuffs, or the food may become contaminated after production. Bacterial contamination with *Salmonella*, *Campylobacter*, and *Escherichia coli* have been reported in cats, and clinical signs usually include vomiting, diarrhea, and inappetence.[12,23,56]

In 2007, melamine and cyanuric acid contamination of cat food resulted in renal failure, death, and the largest cat and dog food recall in U.S. history, involving about 150 brands of food.[8,29,40,45] Melamine and cyanuric acid were likely added to increase the nitrogen content of imported wheat flour or wheat gluten. Although it is illegal to add melamine to human or animal food in the United States, it is a common additive in other countries, such as China. Clinical signs varied in severity and included inappetence with or without vomiting, polydipsia, polyuria, dehydration, vomiting, lethargy, and anorexia. Abdominal palpation often revealed renomegaly. Affected cats became azotemic, with decreased urine specific gravity within 2 weeks of ingesting the contaminated food. Although some cats appeared to recover renal function, others developed chronic renal disease. Cats with severe azotemia, hyperphosphatemia, and hyperkalemia were unlikely to survive. Microscopic examination of the urine of some cats revealed goldish brown crystals, and at necropsy kidneys were found to contain such crystals. Renal tubular necrosis was also present. Kidney and urine samples contained melamine and cyanuric acid.

In 1996 an outbreak of food-associated peripheral neuropathy in cats occurred in the Netherlands and Switzerland.[41,63] The outbreak was related to feeding two brands of dry food contaminated by the coccidiostatic drug salinomycin. Affected cats had acute onset of lower motor neuron signs of variable severity. The clinical signs varied from paraparesis to tetraplegia, were symmetric and bilateral, and progressed from the pelvis to thoracic limbs. Dyspnea occurred in some severely tetraplegic cats. The majority of reported cases recovered with withdrawal of the diet and supportive care, although the most severely affected cats died or were euthanized.

FOOD HYPERSENSITIVITY

An *adverse reaction* to food is defined as a clinically abnormal response attributed to an ingested food or food substance. It may be immunologic or nonimmunologic.

A food hypersensitivity or allergic reaction is an immunologically mediated reaction to an ingested food or food ingredient. This is different from food intolerance, which is a nonimmunologically mediated adverse reaction. Food hypersensitivity reactions appear to cause primarily dermatologic and gastrointestinal clinical signs.

Throughout their lives cats are exposed to a variety of potential dietary allergens; however, after a variable period of time, some animals may develop an immune response against a particular food that activates one or more immunopathogenic pathways. After development of this response, subsequent ingestion of this foodstuff results in clinical signs. These dietary antigens do not normally cause problems because the intestinal mucosa forms a barrier that limits absorption of macromolecules, but this mechanism is imperfect. There is evidence that antigens are absorbed through both normal and abnormal gut. Indeed, antibodies to food allergens, usually IgG, are often demonstrable in normal individuals, but they do not result in clinical disease. On initial presentation of the antigen to the gut mucosa, there is generally an immune response involving IgA. This reduces the amount of antigenic material that is absorbed. Immune complexes of antigen and IgA antibody are transported across hepatocytes, into bile, and are recirculated to the intestine. This local IgA response may be followed by a transitory systemic immune response, but immunologic tolerance follows. Thus there is an apparent paradox of a vigorous local immune response followed by a systemic tolerance. Absorption of macromolecules can be altered in either direction by local immunity. Decreased uptake has been demonstrated experimentally after oral or parenteral immunization in rats, and increased absorption occurs in IgA-deficient human beings. Absorption is also enhanced by vasodilation in the gut mucosa, such as that resulting from a local allergic reaction. In this case the patient becomes caught in an immunologic vicious circle because local hypersensitivity reactions favor access of allergens, which in turn heightens the antibody response.[14,33,65]

Factors that lead to the development of hypersensitivity to ingested antigens are the subject of speculation. Those most frequently implicated are heat- and acid-stable glycoproteins with molecular weights of 18,000 to 30,000 daltons. Hypersensitivity reactions involved in food allergies have been shown to involve types I, III, and IV reactions. However, studies indicate that IgE is implicated in most instances, and the reactions involved include both the classic, immediate type I reaction and the late-phase IgE-mediated reactions.[4,39] The factors that determine the extent of absorption of allergens by the intestine are not fully understood, although local vasodilation is clearly facilitatory. Once local vasodilation is stimulated by local reactions, the cycle feeds on itself. What initiates the original immunologic reaction is not clear. Certainly, if clinical or subclinical gastrointestinal disease occurs that alters mucosal integrity, absorption of antigenic proteins may occur, which may initiate the

FIGURE 17-1 Facial pruritus and excoriation in a cat with presumed food allergy.

processes. Inflammatory mediators involved in food allergy may include interleukins, platelet-activating factor, histamine and other products of mast cells and basophils, and cytokines.

No age, gender, or breed predisposition has been identified for food hypersensitivities in cats. Clinical signs of food hypersensitivity reaction in cats include dermatologic and gastrointestinal signs.* Dermatologic signs include nonseasonal pruritus; alopecia and erythema, particularly around the face and ears (Figure 17-1); and secondary bacterial pyoderma. Other dermatologic signs include eosinophilic granuloma complex and miliary dermatitis. Gastrointestinal signs usually include vomiting, but diarrhea and inappetence may be present. Whether food hypersensitivity reaction is involved with feline asthma, cholangitis–cholangiohepatitis complex, and idiopathic cystitis is subject to speculation.

Most basic food ingredients have the potential to induce an allergic response, although proteins cause the majority of reactions. Dietary components reported to cause food sensitivity in cats include cow's milk, beef, mutton, pork, chicken, rabbit, horse meat, fish, eggs, oatmeal, wheat, corn, soy, rice flour, potatoes, kidney beans, canned foods, cod liver oil, dry food, pet treats, and food additives.

Diagnosis of food hypersensitivity involves first ruling out other potential causes of the clinical signs (Box 17-1). This may be complicated by concurrent diseases with similar clinical signs (e.g., the cat with flea allergy dermatitis and food hypersensitivity). Intradermal skin testing and serologic testing are unreliable for the diagnosis of food allergies in cats. The most useful and reliable aid in diagnosis of dietary sensitivity is the procedure of feeding a restricted or elimination diet followed by dietary challenge with a test meal; however, this is difficult in cats, especially in multicat households.

*References 25, 32, 35, 65, 66, 68, 69.

BOX 17-1
Diagnosis of Food Hypersensitivity

1. Rule out other potential causes for the clinical signs; start with a minimum database (CBC, chemistry panel, urinalysis [and total T_4 for senior cats]) as well as basic system-specific tests such as fecal parasite analysis and skin scrapings; further testing is performed as required.
2. Obtain a full dietary history, including all treats and table food; include food used to administer medications, such as Greenies Pill Pockets, and any flavored medications or supplements.
3. Choose an elimination diet (home prepared or commercial); ideally, the diet should contain a single protein and carbohydrate source to which the cat has not been exposed in the past; introduce the diet gradually (see Box 18-5), and counsel the owner to be patient because some cats will require several weeks to respond.
4. Once the clinical signs have resolved, attempt to identify the offending antigen by introducing foodstuffs one at a time to the elimination diet; if the owner is unwilling to attempt identification, the cat can be fed a commercially prepared limited-protein-source diet that does not provoke clinical signs.

CBC, Complete blood count; *T_4,* thyroxine.

Elimination diets must be individualized on the basis of previous dietary exposure and may be commercial or home-prepared diets. By definition, elimination diets contain protein and carbohydrate sources to which the cat has not been exposed. A detailed study of the cat's diet (including all treats, table food, and flavored medications and supplements) will allow identification of foods that have not been fed before and that could be used to formulate a nutritionally balanced elimination diet that will be hypoallergenic.[18,28,32] As the variety of protein sources in commercial diets has expanded over the years, it has become more difficult to find a novel protein. If it is not possible to formulate a suitable elimination diet, then a restricted diet may be used that contains only one or two potential allergens, preferably ones that the animal has not eaten in the preceding month. A canned diet may be preferable to a dry diet. Many homemade diets that are used as elimination diets are not complete and balanced (e.g., cottage cheese and rice, or chicken and rice) and therefore are not suitable for long-term use (i.e., more than 4 weeks). Supplementation of a homemade diet with vitamins and minerals is encouraged, but use of supplements that contain potentially offending foodstuffs (e. g., beef or pork) is not recommended. In one study use of a homemade diet resulted in better resolution of clinical signs than did

commercially available diets.[28] The owner should also be instructed not to give treats and table food to the cat and restrict the cat's access to food intended for other pets in the home. Good client education and communication are critical to the success of a diet trial, especially in multicat homes. Scheduling follow-up telephone calls and a follow-up visit in the first 1 to 2 weeks may help identify problems that can be corrected early. Gastrointestinal signs may subside in 3 to 5 days, but if the signs are chronic, this may take 4 to 6 weeks or longer. Resolution of dermatologic signs may take 8 weeks or longer. Once clinical improvement is noted, the veterinarian should try to identify the offending antigen by introducing foodstuffs one at a time to the elimination diet. Most cats will relapse within a few days or within 2 weeks when the triggering allergen is reintroduced. However, not all owners are willing to perform this step because it is time-consuming and tedious, and the owner may be reluctant to risk recurrence of the cat's clinical signs.

Because proteins with molecular weights over 18,000 daltons are incriminated as being antigenic, modification of proteins to compounds having lower molecular weight may be of benefit. Protein modification is a process that alters the physical characteristics of protein molecules, presumably reducing the antigenicity and rendering them less able to elicit an immune response. By reducing the average weight of the protein molecule, this process can result in a protein that may be truly hypoallergenic. To be effective, it must reduce the molecular weight of the protein below 18,000 daltons.[7,18] Recently, several commercially available diets containing protein hydrolysates have been introduced. These diets appear to be effective as elimination diets, and they have the advantage of being complete and balanced.

If the client has been cooperative, the presumptive food antigen has been identified. If this has occurred, or if it is not possible to identify the antigen, then long-term management procedures should be instituted. If the elimination diet is a commercially prepared, complete, and balanced diet, it can be used long term. If the elimination diet is a home-prepared diet, it should be changed to a commercially prepared diet of selected protein or a protein hydrolysate diet. This not only provides a nutritionally balanced and complete diet but also is more convenient for owners. There are many single-protein source diets available including diets that contain duck, venison, lamb, rabbit, and kangaroo. If the cat continues to do well, the owner should be strongly advised not to feed the cat table scraps or treats and not to switch the diet even if clinical signs have not recurred. A few cats with dietary hypersensitivity may eventually develop sensitivities to ingredients in the new diet, and the procedure to identify the offending ingredient must be repeated. Dietary hypersensitivity is poorly responsive to corticosteroid therapy, and because of the risks of long-term corticosteroid treatment, emphasis should be placed on dietary management rather than drug therapy.

FOOD INTOLERANCE

Food intolerance is a nonimmunologic abnormal physiologic response to a food item and may involve toxic, pharmacologic, or metabolic reactions or dietary idiosyncrasies, in which the animal is unable to digest or otherwise process a dietary component. Examples of food intolerance include lactose intolerance, gluten intolerance, reactions to vasoactive amines in diet, reactions to histamine-containing foods or foods that stimulate histamine release, reactions to foods that contain opiates or additives, and toxic reaction to food substances. For diagnosis and management of food intolerances, the veterinarian should follow the same steps as for food hypersensitivities.

References

1. Anderson WI, Morrow LA: Thiamine deficiency encephalopathy with concurrent myocardial degeneration and polyradiculoneuropathy in a cat, *Cornell Vet* 77:251, 1987.
2. Baker H, Schor SM, Murphy BD et al: Blood vitamin and choline concentrations in healthy domestic cats, dogs, and horses, *Am J Vet Res* 47:1468, 1986.
3. Bennett D: Nutrition and bone disease in the dog and cat, *Vet Rec* 98:313, 1976.
4. Bircher AJ, Van Melle G, Haller E et al: IgE to food allergens are highly prevalent in patients allergic to pollens, with and without symptoms of food allergy, *Clin Exp Allergy* 24:367, 1994.
5. Botha CJ, Penrith ML: Potential plant poisonings in dogs and cats in southern africa, *J S Afr Vet Assoc* 80:63, 2009.
6. Burger I, Edney A, Horrocks D: Basics of feline nutrition, *In Practice* 9, 1987.
7. Cave NJ: Hydrolyzed protein diets for dogs and cats, *Vet Clin North Am Small Anim Pract* 36:1251, 2006.
8. Cianciolo RE, Bischoff K, Ebel JG et al: Clinicopathologic, histologic, and toxicologic findings in 70 cats inadvertently exposed to pet food contaminated with melamine and cyanuric acid, *J Am Vet Med Assoc* 233:729, 2008.
9. Clark L: Hypervitaminosis A: A review, *Aust Vet J* 47:568, 1971.
10. Clark L, Seawright AA: Skeletal abnormalities in the hindlimbs of young cats as a result of hypervitaminosis A, *Nature* 217:1174, 1968.
11. Davidson MG: Thiamin deficiency in a colony of cats, *Vet Rec* 130:94, 1992.
12. Deming MS, Tauxe RV, Blake PA et al: Campylobacter enteritis at a university: transmission from eating chicken and from cats, *Am J Epidemiol* 126:526, 1987.
13. Dennis JM, Alexander RW: Nutritional myopathy in a cat, *Vet Rec* 111:195, 1982.
14. Farhadi A, Banan A, Fields J et al: Intestinal barrier: An interface between health and disease, *J Gastroenterol Hepatol* 18:479, 2003.
15. Gershoff SN: Nutritional problems of household cats, *J Am Vet Med Assoc* 166:455, 1975.
16. Goldman AL: Hypervitaminosis A in a cat, *J Am Vet Med Assoc* 200:1970, 1992.
17. Griffiths RC, Thornton GW, Willson JE: Eight additional cases of pansteatitis ("yellow fat") in cats fed canned red tuna, *J Am Vet Med Assoc* 137:126, 1960.

18. Guilford WG, Matz ME: The nutritional management of gastrointestinal tract disorders in companion animals, *N Z Vet J* 51:284, 2003.
19. Hayes KC: Nutritional problems in cats: Taurine deficiency and vitamin a excess, *Can Vet J* 23:2, 1982.
20. Hayes KC, Carey RE, Schmidt SY: Retinal degeneration associated with taurine deficiency in the cat, *Science* 188:949, 1975.
21. Hayes KC, Trautwein EA: Taurine deficiency syndrome in cats, *Vet Clin North Am Small Anim Pract* 19:403, 1989.
22. Heinze CR, Larsen JA, Kass PH et al: Plasma amino acid and whole blood taurine concentrations in cats eating commercially prepared diets, *Am J Vet Res* 70:1374, 2009.
23. Hill SL, Cheney JM, Taton Allen GF et al: Prevalence of enteric zoonotic organisms in cats, *J Am Vet Med Assoc* 216:687, 2000.
24. Hilton J: The biosynthesis, function and deficiency signs of taurine in cats, *Can Vet J* 29:598, 1988.
25. Hirt R, Iben C: Possible food allergy in a colony of cats, *J Nutr* 128:2792s, 1998.
26. Huber TL, Laflamme DP, Medleau L et al: Comparison of procedures for assessing adequacy of dog foods, *J Am Vet Med Assoc* 199:731, 1991.
27. Kolata RJ: Feline steatitis (a case report), *Vet Med Small Anim Clin* 66:1028, 1971.
28. Leistra M, Willemse T: Double-blind evaluation of two commercial hypoallergenic diets in cats with adverse food reactions, *J Feline Med Surg* 4:185, 2002.
29. Lewin-Smith MR, Kalasinsky VF, Mullick FG et al: Melamine-containing crystals in the urinary tracts of domestic animals: sentinel event? *Arch Pathol Lab Med* 133:341, 2009.
30. Loew FM, Martin CL, Dunlop RH et al: Naturally-occurring and experimental thiamin deficiency in cats receiving commercial cat food, *Can Vet J* 11:109, 1970.
31. Malik R, Sibraa D: Thiamine deficiency due to sulphur dioxide preservative in "pet meat"—a case of deja vu, *Aust Vet J* 83:408, 2005.
32. Medleau L, Latimer KS, Duncan JR: Food hypersensitivity in a cat, *J Am Vet Med Assoc* 189:692, 1986.
33. Merchant SR, Taboada J: Food allergy and immunologic diseases of the gastrointestinal tract, *Semin Vet Med Surg (Small Anim)* 6:316, 1991.
34. Merchant SR, Taboada J: Systemic diseases with cutaneous manifestations, *Vet Clin North Am Small Anim Pract* 25:945, 1995.
35. Messinger LM: Therapy for feline dermatoses, *Vet Clin North Am Small Anim Pract* 25:981, 1995.
36. Muhlum A, Meyer H: Influence of taurine intake on plasma taurine values and renal taurine excretion of cats, *J Nutr* 121:S175, 1991.
37. Munson TO, Holzworth J, Small E et al: Steatitis (yellow fat) in cats fed canned red tuna, *J Am Vet Med Assoc* 133:563, 1958.
38. Niza MM, Vilela CL, Ferreira LM: Feline pansteatitis revisited: hazards of unbalanced home-made diets, *J Feline Med Surg* 5:271, 2003.
39. Orhan F, Karakas T, Cakir M et al: Prevalence of immunoglobulin e-mediated food allergy in 6-9-year-old urban schoolchildren in the eastern black sea region of turkey, *Clin Exp Allergy* 39:1027, 2009.
40. Osborne CA, Lulich JP, Ulrich LK et al: Melamine and cyanuric acid-induced crystalluria, uroliths, and nephrotoxicity in dogs and cats, *Vet Clin North Am Small Anim Pract* 39:1, 2009.
41. Pakozdy A, Challande-Kathman I, Doherr M et al: Retrospective study of salinomycin toxicosis in 66 cats, *Vet Med Int* 147:142, 2010.
42. Pion PD, Kittleson MD, Rogers QR et al: Myocardial failure in cats associated with low plasma taurine: a reversible cardiomyopathy, *Science* 237:764, 1987.
43. Pion PD, Kittleson MD, Rogers QE et al: Taurine deficiency myocardial failure in the domestic cat, *Prog Clin Biol Res* 351:423, 1990.
44. Polizopoulou ZS, Kazakos G, Patsikas MN et al: Hypervitaminosis A in the cat: a case report and review of the literature, *J Feline Med Surg* 7:363, 2005.
45. Puschner B, Poppenga RH, Lowenstine LJ et al: Assessment of melamine and cyanuric acid toxicity in cats, *J Vet Diagn Invest* 19:616, 2007.
46. Rivers JP, Frankel TL, Juttla S et al: Vitamin D in the nutrition of the cat, *Proc Nutr Soc* 38:36a, 1979.
47. Robertson JE, Christopher MM, Rogers QR: Heinz body formation in cats fed baby food containing onion powder, *J Am Vet Med Assoc* 212:1260, 1998.
48. Schmidt SY: Biochemical and functional abnormalities in retinas of taurine-deficient cats, *Fed Proc* 39:2706, 1980.
49. Schmidt SY, Berson EL, Hayes KC: Retinal degeneration in the taurine-deficient cat, *Trans Sect Ophthalmol Am Acad Ophthalmol Otolaryngol* 81:Op687, 1976.
50. Schuller-Levis G, Mehta PD, Rudelli R et al: Immunologic consequences of taurine deficiency in cats, *J Leukoc Biol* 47:321, 1990.
51. Seawright AA, English PB: Hypervitaminosis A and deforming cervical spondylosis of the cat, *J Comp Pathol* 77:29, 1967.
52. Seawright AA, Hrdlicka J: Severe retardation of growth with retention and displacement of incisors in young cats fed a diet of raw sheep liver high in vitamin A, *Aust Vet J* 50:306, 1974.
53. Sih TR, Morris JG, Hickman MA: Chronic ingestion of high concentrations of cholecalciferol in cats, *Am J Vet Res* 62:1500, 2001.
54. Sisson DD, Knight DH, Helinski C et al: Plasma taurine concentrations and m-mode echocardiographic measures in healthy cats and in cats with dilated cardiomyopathy, *J Vet Intern Med* 5:232, 1991.
55. Steel RJ: Thiamine deficiency in a cat associated with the preservation of "pet meat" with sulphur dioxide, *Aust Vet J* 75:719, 1997.
56. Stiver SL, Frazier KS, Mauel MJ et al: Septicemic salmonellosis in two cats fed a raw-meat diet, *J Am Anim Hosp Assoc* 39:538, 2003.
57. Studdert VP, Labuc RH: Thiamin deficiency in cats and dogs associated with feeding meat preserved with sulphur dioxide, *Aust Vet J* 68:54, 1991.
58. Sturman JA: Dietary taurine and feline reproduction and development, *J Nutr* 121:S166, 1991.
59. Sturman JA, Gargano AD, Messing JM et al: Feline maternal taurine deficiency: effect on mother and offspring, *J Nutr* 116:655, 1986.
60. Sturman JA, Messing JM: Dietary taurine content and feline reproduction and outcome, *J Nutr* 121:1195, 1991.
61. Sturman JA, Messing JM: High dietary taurine effects on feline tissue taurine concentrations and reproductive performance, *J Nutr* 122:82, 1992.
62. Summers BA, Sykes G, Martin ML: Pansteatitis mimicking infectious peritonitis in a cat, *J Am Vet Med Assoc* 180:546, 1982.
63. van der Linde-Sipman JS, van den Ingh TS, van Nes JJ et al: Salinomycin-induced polyneuropathy in cats: morphologic and epidemiologic data, *Vet Pathol* 36:152, 1999.
64. van Vleet JF, Ferrans VJ: Etiologic factors and pathologic alterations in selenium–vitamin E deficiency and excess in animals and humans, *Biol Trace Elem Res* 33:1, 1992.
65. Verlinden A, Hesta M, Millet S et al: Food allergy in dogs and cats: A review, *Crit Rev Food Sci Nutr* 46:259, 2006.
66. Wasmer ML, Willard MD, Helman RG et al: Food intolerance mimicking alimentary lymphosarcoma, *J Am Anim Hosp Assoc* 31:463, 1995.
67. Watson AD: More on feline pansteatitis, *Can Vet J* 21:321, 1980.
68. Zoran D: Is it IBD? Managing inflammatory disease in the feline gastrointestinal tract, *Vet Med* 95:128, 2000.
69. Zoran DL: Nutritional management of feline gastrointestinal diseases, *Top Companion Anim Med* 23:200, 2008.