# Diet and Skin Disease in Dogs and Cats[1]

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ABSTRACT Dietary factors have a major role in the maintenance of healthy coat and skin, and are significant in the etiology and therapy of certain skin diseases. Nutritional deficiencies are now uncommon as a result of the widespread feeding of complete and balanced pet foods. Deficiencies of (n-6) polyunsaturated fatty acids, zinc and vitamins, however, do arise in certain animal- or product-related instances. Supraphysiologic doses of vitamin A have been used in the management of vitamin A–responsive dermatosis in Cocker spaniels; other keratinization defects and seborrheic conditions may respond to retinoid therapy. Much interest has been paid to the therapeutic value of polyunsaturated fatty acid supplements in the management of dermatologic conditions associated with hypersensitivity reactions or keratinization defects. These studies have generally yielded disappointing results, which may reflect shortcomings in the design of some trials. Nevertheless, a placebo-controlled, double-blind, cross-over study has demonstrated a clear benefit of high dose (n-3) fatty acids in the management of pruritic skin disease. There is also preliminary experimental evidence that specific dietary (n-6):(n-3) fatty ratios are useful in the dietary management of inflammatory diseases. Although results of controlled clinical trials are awaited, the argument exists that it is the absolute amount of (n-3) fatty acid intake rather than ratio that is responsible for potential health benefits. J. Nutr. 128: 2783S–2789S, 1998.

KEY WORDS: • *dogs* • *cats* • *skin disease* • *fatty acids* • *vitamins*

The skin is a large, metabolically active organ with a high physiologic requirement for protein and other nutrients. It is not surprising, therefore, that subtle changes in its nutrient supply can have a marked effect on skin and coat condition. Dietary factors may play a role in the etiology and therapy of skin disease in three arenas, i.e., nutrient deficiency or imbalance, nutritional supplementation for therapeutic effect and dietary sensitivity.

## NUTRITIONAL DERMATOSES

Nutritional deficiencies are uncommon as a result of the widespread feeding of complete and balanced pet foods that meet the nutrient profiles specified by expert panels and regulatory bodies. However, deficiencies may arise when the animal's intake is reduced, when the diet is poorly formulated or stored, or when the animal is unable to digest, absorb or utilize the nutrient as a result of disease or genetic factors. Dietary interactions that reduce nutrient availability can result from errors in formulation, prolonged storage or injudicious oversupplementation of an otherwise balanced diet.

Although many nutrient deficiencies may be associated with skin disorders, most produce a similar range of clinical signs. In most cases, the skin develops seborrhea, which is characterized by abnormalities in sebum production and/or keratinization. Typical signs of a nutritional dermatosis include excessive scale, erythema, alopecia or poor hair growth and greasy skin, which may be accompanied by secondary bacterial infection and pruritus. It is generally accepted that signs become evident only after feeding deficient diets for several months.

## ESSENTIAL FATTY ACIDS

Dogs and cats are unable to synthesize linoleic acid; thus, a dietary source is essential in both species. In addition, cats exhibit low Δ-6 desaturase activity and cannot meet their physiologic requirement for arachidonic acid through biotransformation from linoleic acid (Rivers et al. 1975). Consequently, both linoleic acid and arachidonic acid are considered essential nutrients for cats (MacDonald et al. 1983). A dietary requirement for (n-3) polyunsaturated acids (PUFA) has not been documented for dogs and cats, nor have specific deficiency syndromes. However, it has been suggested that there may be a subtle dietary requirement for these fatty acids in certain physiologic states (Bauer 1997).

Essential fatty acids have a structural role in cell membranes, act as precursors for eicosanoids such as prostaglandins and leukotrienes, and are vital for maintaining normal skin structure and function. Of the (n-6) PUFA, linoleic acid [18:2(n-6)] is involved in the maintenance of the cutaneous water permeability barrier, and arachidonic acid [20:4(n-6)] regulates epidermal proliferation via prostaglandin $E_2$.

Dietary deficiencies of essential fatty acids are uncommon but may occasionally occur in dogs and cats that are fed poor quality, low fat dry foods or inappropriately formulated home-

[1] Presented as part of the Waltham International Symposium on Pet Nutrition and Health in the 21st Century, Orlando, FL, May 26–29, 1997. Guest editors for the symposium publication were Ivan Burger, Waltham Centre for Pet Nutrition, Leicestershire, UK and D'Ann Finley, University of California, Davis.

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prepared diets. Levels of PUFA may also be depleted in food after oxidative damage resulting from prolonged storage or in cases in which antioxidants such as vitamin E are included in inadequate amounts. Rarely, fatty acid deficiency may occur in association with fat malabsorption due to hepatic, pancreatic or gastrointestinal disease.

Cutaneous signs may be apparent within 2–3 mo when a deficient diet is fed. Initially, surface lipid production is decreased to produce a dull, dry coat with accompanying fine scale. Prolonged deficiency results in alopecia, a greasy skin particularly on the ears and between the toes, and secondary pyoderma.

In cases in which the deficiency is uncomplicated by other factors, a response to fatty acid supplementation is visible within 3–8 wk (Scott et al. 1995). Correction of the deficiency may be achieved by changing to a higher fat, premium quality diet, by the addition of food oils to the diet or by the administration of proprietary fatty acid supplements. Vegetable oils, such as sunflower oil, are a rich source of linoleic acid, but arachidonic acid is found in quantity only in animal fats. One teaspoon (5 mL) of a mixture of vegetable oil and animal fat or fish oil per can or cup (225 g) of food is an effective supplement (Scott et al. 1995). However, increasing the dietary PUFA content simultaneously increases the requirement for vitamin E and may also increase the requirement for other vitamins and minerals involved in fatty acid utilization. In most cases, it is preferable to feed a better quality prepared pet food or to provide a balanced veterinary supplement containing essential fatty acids, vitamin E and zinc (Harvey 1993a).

In addition to correcting keratinization defects due to absolute or relative deficiency states, essential fatty acid supplements may also have a therapeutic role in the treatment of a number of other dermatoses. Some cases of canine idiopathic seborrhea demonstrate changes similar to those of essential fatty acid–deficient animals and may respond to supplementation with sunflower oil (Campbell 1993). Manipulation of dietary PUFA may also alter the balance of pro- and anti-inflammatory eicosanoid production and has been used therapeutically for the management of some inflammatory skin disorders, particularly those associated with hypersensitivity reactions. Conditions that may respond to essential fatty acid supplementation include canine atopy, flea-allergic dermatitis and feline miliary eczema. It has also been suggested that dogs with atopic dermatitis may have an impaired ability to convert linoleic acid to the longer-chain (n-6) PUFA and their derivatives, and may benefit from dietary fatty acid supplementation (Harvey 1993a).

## PROTEIN

Hair is composed of ~95% protein, which is rich in the sulfur-containing amino acids, methionine and cystine. Normal growth of hair and keratinization of the skin thus create a high demand for protein and may account for between 25 and 30% of the animal's daily protein requirement (Scott et al. 1995). Failure to meet this demand results in the cutaneous manifestations of protein malnutrition including brittle, depigmented hair, which is easily shed and slow to regrow, excessive scaling and thin, inelastic and hyperpigmented skin.

Protein deficiency is rare in clinical practice but is occasionally encountered after starvation, disease-induced inappetence or the prolonged feeding of a poorly formulated or inappropriate diet. Primary protein deficiencies are most likely to occur when requirements are increased as in young, growing animals and in pregnant or lactating females. Alternatively, protein malnutrition may be associated with excessive protein loss, which may occur in certain chronic illnesses such as pancreatic disease or the protein-losing nephro- or enteropathies. Dietary correction involves supplementation with high quality protein sources such as meat, eggs and milk, but the prognosis may be complicated by the presence of underlying disease.

## ZINC

Zinc plays a critical role in regulating many aspects of cellular metabolism, a number of which are concerned with the maintenance of a healthy coat and skin. Zinc is an integral component of a wide range of metalloenzymes and, as a cofactor for RNA and DNA polymerases, its presence is of particular importance in rapidly dividing cells, including those of the epidermis. Zinc is also essential for the biosynthesis of fatty acids, participates in both the inflammatory and immune systems and is involved in the metabolism of vitamin A.

In the adult, signs of zinc deficiency are confined mainly to the skin, but these may be accompanied by growth and other abnormalities in young animals. Appetite may be depressed in affected animals as a result of a diminished sense of taste and smell; prolonged deficiency can result in weight loss, impaired wound healing, conjunctivitis and keratitis. Generalized lymphadenopathy is also a common feature, particularly in young animals.

Cutaneous signs are characterized by focal areas of erythema, alopecia, crust and scale, which develop symmetrically around the extremities, mucocutaneous junctions and pressure points of the limbs. Areas of crust with underlying suppuration are particularly evident around the mouth, eyes and ears and may also form on the vulva, scrotum and prepuce. Thick crusts are also common over the elbow, hock and other pressure points and, in some cases, the footpads may become thickened with deep fissures. The hair coat is typically dull and harsh, and secondary skin infections with bacteria or *Malassezia pachydermatitis* are common.

Absolute dietary deficiencies of zinc are considered rare in dogs and have not been reported in cats. However, a relative deficiency may occur in some dogs when the availability of dietary zinc is reduced through nutrient interactions or cases in which intestinal absorption of zinc is impaired as a result of disease or genetic factors. A defect in zinc metabolism has been implicated in lethal acrodermatitis of English bull terriers, which gives rise to severe systemic as well as cutaneous signs that resemble experimental zinc deficiency. This disease is inherited as an autosomal recessive trait, is unresponsive to zinc supplementation and is usually fatal, with an average survival time of 7 mo for affected puppies (Jezyk et al. 1986).

Absorption of zinc can be inhibited by excessive levels of dietary calcium, iron and copper, which compete with zinc for intestinal absorption sites. Dietary phytate, which is found in cereal-based diets, chelates zinc, and high levels may also hinder intestinal zinc absorption. Historically, most cases of zinc-responsive dermatosis in dogs were associated with the feeding of poor quality, cereal-or soy-based dry foods, whose effects may have been exacerbated in some animals with a simultaneous inherent defect of zinc absorption. Prolonged enteritis or other malabsorption syndromes may also prevent normal zinc absorption and may precipitate clinical signs of deficiency, particularly when combined with other predisposing factors.

Clinically, two zinc-responsive dermatologic syndromes are recognized, although there is considerable overlap between the two. Syndrome I is associated with defective intestinal absorption of zinc and occurs predominantly in Alaskan malamutes

and Siberian huskies, although other breeds may also be affected. A genetic disorder of decreased capacity for zinc absorption has been identified in Alaskan malamutes. In some Siberian huskies, hypothyroidism and low serum zinc may occur concurrently, although the significance of this is not clear (Kunkle 1980, Scott et al. 1995). The appearance of lesions frequently coincides with the onset of adulthood and during periods of stress; it may be linked to higher metabolic requirements in these dogs. Although dietary interactions may limit zinc absorption in some affected animals, the condition occurs in many cases despite feeding a nutritionally complete and balanced diet. Oral zinc supplementation, together with dietary correction, where appropriate, brings rapid resolution of signs in most cases. Supplementation with zinc sulfate [10 mg/(kg · d)] or zinc methionate [1.7 mg/(kg · d)] is usually adequate, but lifelong therapy is normally required, and the dosage may be adjusted for long-term maintenance (Scott et al. 1995). Some cases, especially Siberian huskies, do not respond to oral supplementation and may require the intravenous administration of zinc sulfate (10–15 mg/kg) at weekly intervals for 4 wk, followed by maintenance injections every 1–6 mo to prevent a relapse.

Syndrome II is usually seen in rapidly growing puppies, particularly of the giant breeds, and may correspond with a high metabolic requirement for zinc in affected animals. The condition occurs when the diet is absolutely or relatively deficient in zinc, and is most likely to be seen when the diet is high in phytate or is oversupplemented with calcium. Lesions resolve within 2–6 wk after dietary correction, but the response time can be hastened by oral supplementation with zinc. In these cases, supplementation may be discontinued once the clinical signs are in remission.

## VITAMIN A

Vitamin A (retinol and its derivatives) has many physiologic functions and is involved in the regulation of cellular growth and differentiation. It is essential to maintain the integrity of epithelial tissues and is particularly important for the keratinization process. Both deficiency and excess of vitamin A can give rise to cutaneous lesions of hyperkeratinization and scaling, alopecia, poor hair coat and increased susceptibility to microbial infections (Scott et al. 1995). Hyperkeratinization of the sebaceous glands can result in occlusion of their ducts and the formation of firm, papular eruptions.

Cats require a dietary source of preformed retinol because, unlike dogs, they are unable to utilize the retinol precursor, $\beta$-carotene (Brewer 1982). Nevertheless, vitamin A deficiency is rare in companion animals, and a toxicity state, with its accompanying skeletal changes, is more likely to occur. Hypervitaminosis A is seen predominantly in cats that are fed large amounts of liver or after prolonged oversupplementation of the diet with vitamin A or cod liver oil.

Vitamin A–responsive dermatosis is a rare condition that is seen almost exclusively in Cocker spaniels even when fed an apparently nutritionally adequate diet (Ihrke and Goldschmidt 1983, Parker et al. 1983). Affected animals exhibit a generalized defect in keratinization with scaling, greasy skin, alopecia, pruritus and secondary pyoderma, and characteristic hyperkeratotic plaques that project above the skin surface. The condition is refractory to other antiseborrheic treatment but responds slowly to oral supplementation with vitamin A (retinol) at 10,000 IU/d. Clinical improvement is observed within 5–8 wk, although lifelong maintenance therapy is usually required. This dose is in excess of the normal dietary requirement for vitamin A in dogs and it is important, therefore, that other causes of seborrhea be eliminated before therapy is initiated. In true deficiency syndromes, vitamin A therapy should not exceed 400 IU/(kg · d) orally or a single injection of 6000 IU/kg, which need not repeated for over 2 mo (Scott et al. 1995).

## VITAMIN E

Vitamin E is a natural antioxidant and, together with selenium, is important for maintaining stability of cell membranes. As a free radical scavenger, it protects cells from the potentially damaging effects of toxic oxygen radicals, whose major source is lipid metabolism. The dietary requirement of vitamin E, therefore, is linked to the dietary intake of PUFA, and high fat diets can induce a relative deficiency of vitamin E. Similarly, levels of vitamin E may be depleted after the oxidation of fat during processing or prolonged storage of food.

Pansteatitis is associated with a deficiency of vitamin E in cats that are habitually or exclusively fed high fat diets, particularly canned red tuna or other oily fish (Harvey 1993a). In these cases, low levels of vitamin E relative to the PUFA content of the diet result in the accumulation of ceroid, a product of lipid peroxidation, in subcutaneous and intra-abdominal fat. Necrosis and subsequent inflammation of the affected tissues produce firm, nodular masses, and the cat may exhibit considerable pain on palpation and movement. Treatment consists of dietary correction together with vitamin E supplementation [10 mg/(kg · d)] and supportive therapy, but the prognosis for untreated or severely affected cats is poor.

There is no record of naturally occurring vitamin E deficiency in dogs. However, supraphysiologic doses of vitamin E have been used in the treatment of canine discoid lupus erythematosis and primary acanthosis nigricans with variable successes reported (Werner and Harvey 1995).

## VITAMIN B

The B-complex vitamins are involved as cofactors in many metabolic functions, especially energy metabolism and synthetic pathways. Because they are water soluble, they are not stored in the body; however, the animal's daily requirements can normally be met from a combination of dietary sources and intestinal microbial biosynthesis. Deficiencies may occur, nevertheless, after prolonged oral antibiosis, anorexia or when water loss is increased as in polyuric conditions or enteritis. Occasionally, deficiencies of individual B-group vitamins arise as a result of interaction with other dietary components.

In general, skin lesions associated with deficiencies of B-group vitamins include dry, flaky seborrhea and alopecia. Biotin deficiency produces a characteristic alopecia around the face and eyes with crusting in severe cases. This condition may occur in the unusual circumstance of feeding large amounts of raw egg whites which contain avidin, a protein that binds biotin and prevents its gastrointestinal absorption. Riboflavin deficiency produces cheilosis in addition to seborrhea but will not occur if the diet contains meat or dairy products. Niacin is synthesized from tryptophan by all animals except cats (Scott et al. 1995), and a deficiency is possible only when the diet is low in animal protein and high in corn or other cereals that are a poor source of tryptophan. A deficiency results in pellagra (humans) or "black tongue" (dogs), with ulceration of mucous membranes, diarrhea and emaciation and, occasionally, in a pruritic dermatitis of the hind legs and ventral abdomen (Scott et al. 1995). Pyridoxine deficiency may cause a dull, waxy, unkempt coat with fine scales and patchy alopecia but has been reproduced only in experimental studies.

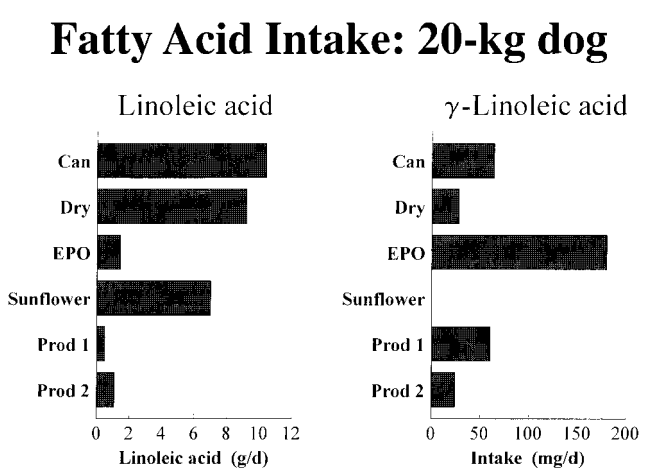


**FIGURE 1** Estimated daily intakes of linoleic and γ-linolenic acid for a 20-kg dog fed typical canned (Can) and dry (Dry) dog foods, compared with fatty acids supplied by supplementation with evening primrose oil (EPO, 2.2 mL), sunflower oil (10 mL), and two commercially available fatty acid preparations (Prod 1 and Prod 2) fed according to manufacturer's recommendations.

Treatment of B-vitamin deficiency involves dietary correction, where appropriate, and supplementation with the entire vitamin B group to compensate for reduced intake or increased losses. Oral supplementation with brewers yeast and/or parenteral administration of B-vitamins is usually effective. In addition, biotin supplementation has been used with some success in the treatment of various dermatoses and disorders of keratinization.

## NUTRITIONAL SUPPLEMENTATION FOR THERAPEUTIC EFFECT

Supraphysiologic doses of nutrients have been used in the management of certain skin diseases. In such cases, nutrient supplementation is likely to have a pharmacologic effect rather than merely correcting a deficiency. Although there has been interest in the use of vitamins A and E for certain conditions, as discussed in the previous section, the efforts of veterinary nutritionists and dermatologists have concentrated largely on dietary PUFA.

The primary indications for therapeutic dietary fatty acid supplementation in dogs and cats are the pruritic skin diseases associated with hypersensitivity reactions, namely, flea allergic dermatitis, atopic dermatitis, hypersensitivity to certain foods and idiopathic pruritus, as well as the eosinophilic granuloma complex in cats. The other major indication concerns conditions associated with abnormalities in fatty acid metabolism, potentially hypothyroidism, atopic dermatitis and defects of keratinization. Linoleic acid is indicated in certain keratinization defects in dogs because of its effect on cutaneous permeability and the ability of its metabolite arachidonic acid to regulate epidermal proliferation. Supplementation with γ-linolenic acid, generally in the form of evening primrose oil, and eicosapentaenoic acid, as marine fish oils, has been studied in both dogs and cats with pruritic skin diseases.

The potential benefit of fatty acid supplementation was assessed in a survey of North American veterinary dermatology specialists (Logas 1995). In this survey, >90% of respondees believed dietary fatty acid supplements to be useful, with nearly 50% using them in >75% of pruritic cases. However, the efficacy of such supplements was judged as only "mildly effective" by the majority of respondees. This apparent contradiction between perceived or potential benefit and true clinical efficacy is also evidenced by the results of clinical trials of fatty acid supplementation. The majority of these trials were reported between 1988 and 1993 and have already been reviewed by Campbell (1993) and Harvey (1993b). These studies evaluated monotherapy with either evening primrose oil or marine fish oil, combination therapy with both oil types and certain commercially available products with combinations of both (n-6) and (n-3) fatty acids. The results of these trials were generally disappointing, with success rates ranging from 10 to 80% in uncontrolled studies and no significant treatment effect in placebo-controlled studies.

The lack of clear benefit in these studies may be explained by the fact that such dietary supplements have no significant effect. Although this could be the case, there are a number of other reasons why the studies have failed to prove benefit; these include inadequate duration of treatment or dose of supplement and failure to take into account differences in background dietary fatty acid consumption. In the earlier studies, fatty acid supplements were fed for periods ranging from only 1–2 wk compared with 6–12 wk in later studies, which is now generally accepted to be the time lag to clinical response (Harvey 1993a). Another explanation may be simply that the doses of fatty acid used were not sufficient to produce an effect. This is particularly important because there was no attempt to measure or control the levels of fatty acids consumed in the background diet in any of the studies reviewed by Harvey (1993b) and Campbell (1993). This pitfall is clearly demonstrated when a supplemental fatty acid intake is compared with fatty acids supplied in typical canned and dry pet foods (**Figs. 1–4**). These data demonstrate two key features. First, the variability in background fatty acid intake can be at least as great as the level of supplemental fatty acids provided; second, the levels of fatty acids in commercial preparations, when fed according to the manufacturers' instructions, are likely to be swamped by those in certain background diets, thus providing no additional benefit. This hypothesis is supported by the study of Sture and Lloyd (1995) in which a commercially available evening primrose oil/marine fish oil combination was shown to have no significant benefit over placebo (olive oil) in dogs with atopic dermatitis. Adequate

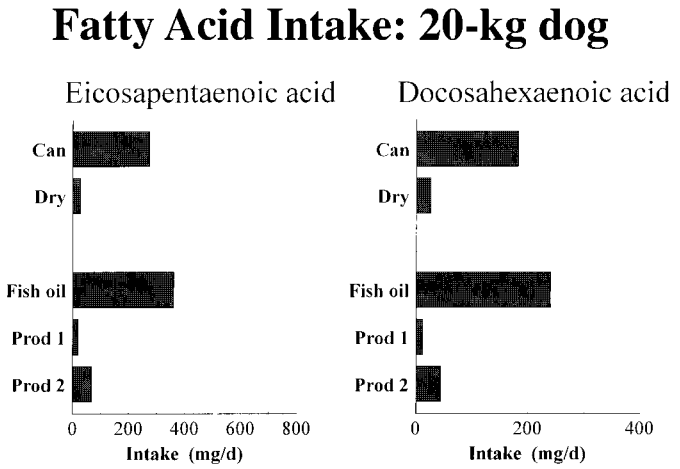


**FIGURE 2** Estimated daily intakes of eicosapentaenoic and docosahexaenoic acid for a 20-kg dog fed typical canned (Can) and dry (Dry) dog foods, compared with supplementation with marine oil (2 mL) and two commercially available fatty acid preparations (Prod 1 and Prod 2) fed according to manufacturer's recommendations.

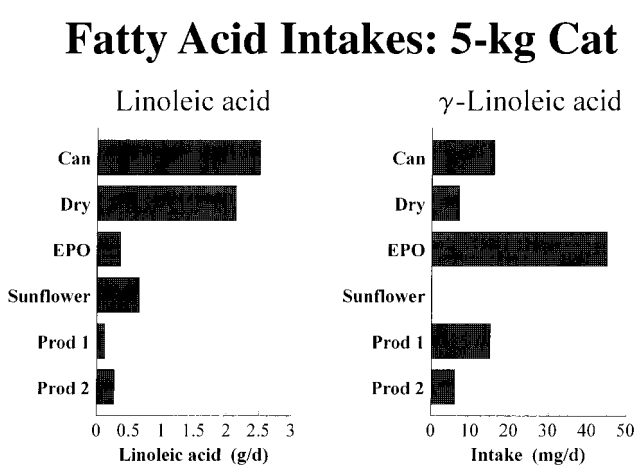


**FIGURE 3** Estimated daily intakes of linoleic and γ-linolenic acid for a 5-kg cat fed typical canned (Can) and dry (Dry) dog foods, compared with fatty acids supplied by supplementation with evening primrose oil (EPO, 0.5 mL), sunflower oil (1 mL), and two commercially available fatty acid preparations (Prod 1 and Prod 2) fed according to manufacturer's recommendations.

clinical response was achieved, however, in 73% of the dogs when the level of supplementation was increased and the mean dosage was ~1.4 times the initial dose.

In the midst of this confusion, one well-designed study has shown a clear benefit from supplementation with fatty acids in dogs with pruritic skin disease (Logas and Kunkle 1994). In this study, a relatively high dose of marine fish oils (18% eicosapentaenoic acid, 12% docosahexaenoic acid; 1 mL/4.55 kg body weight) was fed to 16 dogs with pruritic skin disease for 6 wk in a placebo-controlled, double-blind, cross-over study. The response was assessed with respect to pruritus and skin and coat character, and compared with a corn oil placebo at the same dose. Supplementation with marine fish oil was associated with significant decreases in pruritus (−38%) and alopecia (−45%) and increases in coat character (+57%), whereas there was no significant response to corn oil. The improvements in alopecia and coat condition were attributed to a reduction in pruritus and self-trauma. Overall, 11 of the 16 owners reported that their dog's condition was significantly improved when they consumed marine fish oils.

An alternative approach to fatty acid supplementation is the concept of dietary (n-6) to (n-3) fatty acid ratios. This is based on the assumption that eicosanoids derived from (n-6) fatty acids are proinflammatory, whereas those derived from (n-3) fatty acids are anti-inflammatory or have little or no proinflammatory activity. Support for this concept came from an elegant experimental study in which five groups of six healthy beagle dogs each were fed diets with (n-6): (n-3) fatty acid ratios between 5:1 and 100:1 (Vaughn et al. 1994). Each group received a single diet for 12 wk; activities of leukotriene ($LTB_4$) and $LTB_5$ were measured in skin biopsies and circulating neutrophils at 0, 6 and 12 wk. Dogs receiving diets with lower ratios (5:1 and 10:1) demonstrated a significant reduction in the activity of proinflammatory $LTB_4$ and a concomitant increase in the activity of $LTB_5$, which has little or no inflammatory activity, compared with diets with ratios of 25:1 and greater. Although controlled clinical trials of this concept have not yet been published, preliminary results from two single-blind studies were presented to 12th Annual Congress of the European Society of Veterinary Dermatology (Schick et al. 1995, Scott 1995). In the first study, 18 dogs with atopy were fed a commercial lamb and rice diet with an (n-6):(n-3) fatty acid ratio <10:1; the response was judged as good to excellent in eight dogs (44%). In the second study, 31 dogs with either confirmed atopy, adverse reactions to foods or a combination of both diseases were fed a commercial fish and potato diet also with an (n-6):(n-3) fatty acid ratio <10:1; the response was judged as good to excellent in 14 of the 28 dogs that completed the study (50%). Unfortunately, due to the absence in both studies of a placebo diet without an amended (n-6):(n-3) fatty acid ratio, it remains unclear to what extent this response was due to the dietary constituents and the restricted protein sources (lamb and rice or fish and potato) in particular, as opposed to the fatty acid ratio.

A further complication in interpreting these studies is the level of certain fatty acids in products with low (n-6):(n-3) fatty acid ratios. Marine fish oils were added to achieve the low ratios in the study of Vaughn et al. (1994) with the effect of significantly increasing the levels of (n-3) fatty acids in those diets compared with those with higher ratios. The levels of (n-3) fatty acids in the experimental diets with ratios of 5:1 and 10:1, when fed to dogs, are in fact similar to the intakes that would have been achieved in the study of marine fish oil supplementation of Logas and Kunkle (1994) (**Fig. 5**). Thus it is possible that any beneficial effects of diets with a low (n-6):(n-3) fatty acid ratio are simply due to their (n-3) fatty acid, specifically eicosapentaenoic acid, content. This concept is supported by a recent study in humans showing that it is the absolute amounts of fish oils, and not the ratio of fish to vegetable oil, that determine the magnitude of changes in cell membrane phospholipid fatty acid content and hence physiologic response (Hwang et al. 1997).

## DIETARY SENSITIVITY

The term "dietary sensitivity" describes any adverse reaction to food and may be further classified as either food intolerance or true food allergy (hypersensitivity). True dietary hypersensitivity is an immune-mediated phenomenon, whereas food intolerance denotes any other clinically abnormal response to a dietary component. Food intolerance can result from an impaired ability to digest the food or from pharmacologic, metabolic or toxic reactions. In practice, however, a distinction is seldom made between

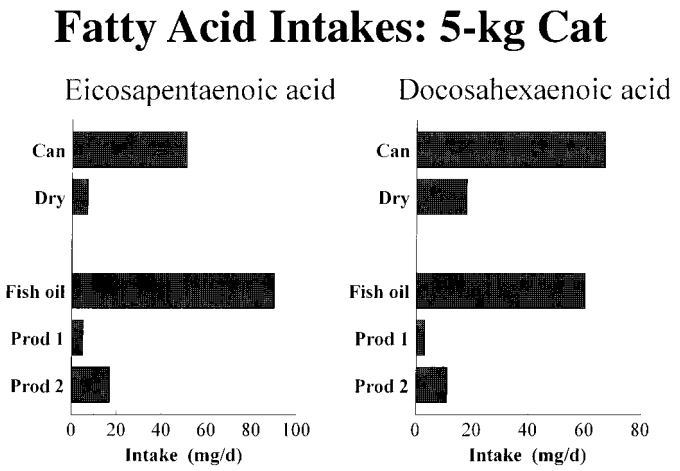


**FIGURE 4** Estimated daily intakes of eicosapentaenoic and docosahexaenoic acid for a 5-kg cat fed typical canned (Can) and dry (Dry) dog foods, compared with supplementation with marine oil (0.5 mL) and two commercially available fatty acid preparations (Prod 1 and Prod 2) fed according to manufacturer's recommendations.

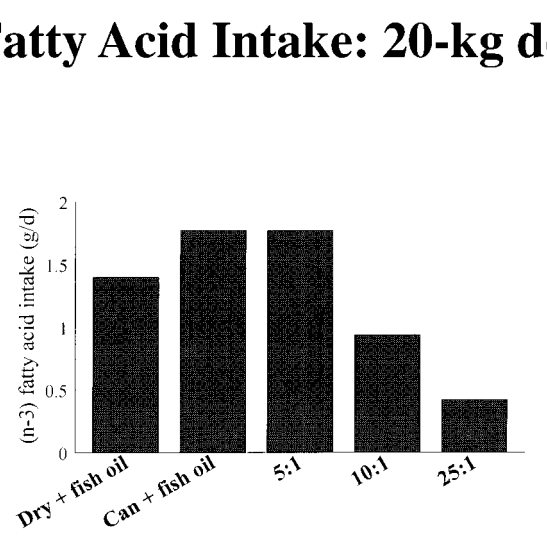


**FIGURE 5** Estimated (n-3) fatty acid (eicosapentaenoic plus docosahexaenoic acid) intakes for a 20-kg dog fed typical dry (Dry) and canned (Can) dog foods supplemented with marine fish oils at 1 mL/4.55 kg bodyweight (as per Logas and Kunkle 1995) These values are compared with theoretical intakes for the same size dog if fed the experimental diets with adjusted (n-6) to (n-3) fatty acid ratios used in the study of Vaughn et al. (1994).

food intolerance and food hypersensitivity because they are often impossible to differentiate on the basis of the observed clinical signs, and management protocols are identical for both.

Most cases of dietary sensitivity manifest as skin or gastrointestinal disorders; a number of cases will present with signs involving both systems (Paterson 1995). Pruritus is the most frequently observed presenting sign, which is accompanied by a gradation of clinical signs associated with self-inflicted trauma. Otitis externa may also be a feature of the condition, sometimes in the absence of other skin lesions, and dietary sensitivity has been implicated as a cause in some cases of feline miliary dermatitis and eosinophilic plaque (Wills and Halliwell 1994). The condition may mimic other allergic dermatoses, such as atopy or flea-allergic dermatitis, and the clinical picture may be further complicated by the presence of these and other factors that contribute to the development of skin disease.

The reported incidence of dietary sensitivity varies considerably but most authors agree that it is a rare cause of skin disease in dogs and cats. It has been estimated that dietary sensitivity accounts for ~1% of all canine and feline dermatoses (Walton 1967), 10% of canine allergic skin disease, excluding flea allergic dermatitis (Scott 1978), 10% of all nonseasonal dermatitis (Baker 1975), 11% of cases of feline miliary dermatitis (Scott 1987) and 10–20% of allergic dermatoses seen by referral dermatologists (Brown et al. 1995).

The true incidence of dietary sensitivity may be difficult to establish due to the inherent problems in reaching a definitive diagnosis. Rechallenge with the original diet is often omitted from the diagnostic protocol because the owner is unwilling to witness a recurrence of clinical signs. Furthermore, skin disease frequently has a multifactorial etiology, and successful management of one aspect of disease may be sufficient to render the animal asymptomatic. Investigation of other possible etiologies may therefore be discontinued at that point.

Specific tests for diagnosing food hypersensitivity using immunologic techniques are available; however, they are unreliable in companion animals. Intradermal testing with food extracts, radioallergosorbent testing (RAST), enzyme-linked immunosorbent assay (ELISA) and gastroscopic food sensitivity testing have all been used. Although they may help to rule out a specific diagnosis, their positive predictive value remains relatively low ( Brown et al. 1995, Wills and Halliwell 1994).

Although it does not identify the underlying mechanism of disease, the most useful and reliable method of diagnosing dietary sensitivity is to feed an elimination diet, based on previous dietary history, followed by dietary challenge with a test meal. Ideally, the elimination diet should contain no ingredients that have previously been consumed by the animal and should be formulated using single sources of protein and carbohydrate. The diet must be nutritionally complete and balanced because prolonged feeding may be necessary; this should be achieved using a minimum number of ancillary ingredients, which may also provoke sensitivity reactions.

Food intolerance may be associated with any dietary ingredient, including additives. Most basic food ingredients have the potential to induce an allergic response, but the majority of dietary hypersensitivity reactions are caused by proteins. The most commonly recognized causes of dietary sensitivity in dogs and cats include beef and dairy proteins, other meat proteins and eggs, lactose and gluten. Elimination diets that have been successfully employed in dogs and cats include lamb, chicken, rabbit, venison and various fish species; these are typically fed with rice or potatoes as a source of carbohydrate. There is no universally "hypoallergenic" diet, however, and the successful inclusion of these ingredients in elimination diets simply reflects the fact that traditionally, they have not been used widely in commercially prepared pet foods.

The elimination diet should be fed for a minimum of 3 wk, although a trial period of up to 60 d may be required for maximum improvement. Failure to respond within this time suggests that either dietary sensitivity is not involved, other factors may be contributing to the clinical disease or the animal is sensitive to the protein in the elimination diet. A small number of animals will react to commercially prepared elimination diets but not to home-prepared diets using the same ingredients and it may be preferable to use a home-prepared diet in the initial diagnostic stages.

A diagnosis of dietary sensitivity is confirmed by challenging with the original diet and demonstrating an exacerbation of clinical signs within 1–14 d. The animal may then be "rescued" with the elimination diet and it may be possible to introduce a commercially prepared diet with the same ingredients at this stage. Individual protein sources may be introduced sequentially and the response monitored in an attempt to identify specific allergens that should be avoided. Once a diagnosis has been established, it is usually possible to manage cases of dietary sensitivity by using commercial diets that have novel, restricted protein sources and are free of lactose and gluten. Alternatively, it may be possible to identify a range of standard products that the animal is able to tolerate.

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