

ACVN NUTRITION NOTES

Diets and the Dermis: Nutritional Considerations in Dermatology

Justin Shmalberg, DVM, DACVN, DACVSMR
University of Florida College of Veterinary Medicine

shutterstock.com/Christian Mueller

The American College of Veterinary Nutrition (acvn.org) and *Today's Veterinary Practice* are delighted to bring you the **Nutrition Notes** column, which provides the highest-quality, cutting-edge information on companion animal nutrition, written by the ACVN's foremost nutrition specialists.

The primary objectives of the ACVN are to:

- Advance the specialty area of veterinary nutrition
- Increase the competence of those practicing in this field
- Establish requirements for certification in veterinary nutrition
- Encourage continuing education for both specialists and general practitioners
- Promote evidence-based research
- Enhance dissemination of the latest veterinary nutrition knowledge

The ACVN achieves these objectives in many ways, including designating specialists in animal nutrition, providing continuing education through several media, supporting veterinary nutrition residency programs, and offering a wide array of resources related to veterinary nutrition, such as this column.



Dermatologic patients are often managed with topical and systemic pharmacologic therapies, but nutrition should be evaluated in all animals presenting with skin disease. Nutritional deficiencies and excesses are rarely the underlying cause of a patient's clinical signs, but nutritional modifications often reduce the severity of such signs.

NUTRIENTS

The influence of nutrition in dermatologic conditions is explained by critical nutrients that affect keratinization, cellular barriers, and turnover, as well as sebum production and composition.

Protein and amino acids provide substrates for keratinization, pigmentation, and hair growth. A substantial portion of daily protein requirements is used for skin and hair production.¹

- **Phenylalanine** and **tyrosine** are precursors to melanin, and relative deficiencies may induce reddening of black coats (**Figure 1**).²
- **Methionine** and **cysteine**, the primary sulfur-containing amino acids, are incorporated into hair in large amounts and may be the “limiting” amino



FIGURE 1. This middle-aged domestic shorthair cat displays reddening of the fur, which may prove responsive to increased dietary protein and/or supplementation with tyrosine or phenylalanine.

acids (ie, those which limit protein synthesis) in some pet diets. Deficiency of these amino acids may reduce hair growth and increase hair fragility.^{3,4}

- Proline is a nonessential amino acid critical to collagen formation in the skin. Protein deficiency would be expected to have a deleterious effect on skin strength.³

Polyunsaturated fatty acids provide substrate for the production of sebum, which protects the dermis and improves coat quality.^a

- **Linoleic acid**, an 18-carbon omega-6 fatty acid, appears to be most important for this effect.⁵⁻⁷
- **Omega-3 fatty acids**, such as eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), may reduce inflammation secondary to allergic disease in affected dogs and cats.⁸

Several vitamins play critical roles in skin health.

- **Vitamin A** is a group of fat-soluble retinoids critical for epidermal differentiation and normal sebum production. Deficiency is rare, but unlike dogs, cats cannot convert beta-carotene to retinol.³
- **Vitamin C** is synthesized in dogs and cats from glucose (unlike in humans) and is necessary for collagen synthesis and cross-linking.

^aFor more information on the roles of polyunsaturated fatty acids, see "Dietary Fatty Acids in Dogs & Cats" by Dr. Catherine Lenox in the September/October 2016 issue of *Today's Veterinary Practice* on tvjournal.com.

- **Vitamin E** is the primary antioxidant in the cell membrane and can refer to any of 4 tocopherols and 4 tocotrienols. Alpha-tocopherol is the form with greatest activity in cells, although others may be added to commercial diets as natural preservatives. Experimental deficiency causes alopecia, seborrhea, and increased cutaneous infections.³
- **B vitamins** participate in many biochemical reactions throughout the body, and experimental deficiencies have caused a variety of skin lesions. Vitamin B3 (niacin) and B5 (pantothenic acid) supplementation above normal recommended allowances has reduced transepidermal water loss in dogs and contributed to the integrity of the stratum corneum.⁹

Copper serves as a cofactor in enzymatic conversion of tyrosine to melanin. Deficiency can cause changes in pigmentation. **Zinc** is critical for the transition of nucleated epidermal cells to anucleate squamous cells in the stratum corneum, and deficiency induces parakeratosis. Zinc also participates in essential fatty acid conversion.³

Commercial diets conforming to Association of American Feed Control Officials recommendations should be replete in all of the nutrients above. However, several factors could increase the relative risk for deficiencies over time:

- Malabsorptive disorders, such as exocrine pancreatic insufficiency, inflammatory bowel disease, and small intestinal bacterial overgrowth
- Home-prepared diets without dietary analysis or supplementation
- Diets labeled for intermittent or supplemental feeding when fed for an extended time
- High dietary phytate concentration, which



FIGURE 2. Zinc-responsive dermatosis syndrome 1 in a young Siberian husky. Courtesy Dr. Dunbar Gram.

binds minerals such as zinc, or excesses of some minerals that adversely affect absorption of others

CLINICAL CONDITIONS

Several common dermatologic signs may prove nutritionally responsive (**Box 1**). The primary cause may not always be identified, but a skin biopsy is often prudent to better characterize the lesion and to decide how best to modify a patient's diet. Knowledge of characteristic or common clinical conditions informs the possible use of nutritional intervention, and a response to a particular nutritional therapy is often used to confirm a presumptive diagnosis.

Zinc-Related Dermatoses

In dogs, zinc-related dermatoses are characterized by parakeratotic hyperkeratosis of the face and feet, with resultant crusts and lesions (**Figure 2**).¹⁰ These dermatoses include lethal acrodermatitis in bull terriers and zinc-responsive dermatosis syndromes I and II. Characteristics of these conditions are presented in **Box 2**.

Serum and hair testing are unreliable measures of zinc status, so supplementation of dogs with characteristic lesions, especially in breeds

BOX 1. Dermatologic Signs That May Respond to Nutritional Management

- Pruritus
- Recurrent pyoderma
- Dull hair coat
- Epidermal scaling
- Urticaria
- Keratinization disorders
- Concurrent gastrointestinal signs
- Coat color change
- Chronic otitis
- Miliary dermatitis (in cats)
- Alopecia

TABLE 1 Concentration of Elemental Zinc in Different Oral Supplements*

ZINC SUPPLEMENT	MG ZN/100 MG POWDER
Zinc gluconate	13
Zinc methionine	23
Zinc sulfate	36

*A starting dose of 2 to 3 mg Zn/kg is often recommended. Many supplements are sold according to the amount of elemental zinc.

associated with zinc-responsive dermatosis syndrome I, is advised. Zinc gluconate is well tolerated and efficacious, but some clinicians prefer zinc methionine because of increased availability. The relative amount of elemental zinc for different supplements is found in **Table 1**. Vomiting is the most common, and generally dose-dependent, side effect of zinc supplementation, and several forms may need to be tried at varying doses to assess individual tolerance.

BOX 2. Characteristics of Zinc-Related Dermatoses

Lethal acrodermatitis of bull terriers¹¹

- Complete dysfunction of zinc metabolism
- Impaired growth, eating difficulty, crusting and scaling of the feet and pads, and splayed digits
- Unresponsiveness to zinc supplementation
- High mortality rate

Zinc-responsive dermatosis syndrome I^{3,10}

- Results from a genetic defect that reduces zinc absorption
- Is more prevalent in Northern breed dogs (huskies, malamutes)
- Requires lifetime oral zinc supplementation (2 to 10 mg elemental zinc per kg is empirically recommended)^{4,10}

Zinc-responsive dermatosis syndrome 2^{3,12}

- Associated with rapidly growing large-breed dogs or dogs receiving diets high in phytates and other zinc-binding compounds
- Resolves after a change to a diet with greater zinc concentrations and/or with reduced zinc-binding compounds

Vitamin A-Responsive Dermatitis

Dermatitis responsive to vitamin A is a rare, presumably heritable, condition of dogs. Lesions are characterized by abnormal cornification, hyperkeratotic plaques, abnormal sebum, epidermal scaling, alopecia, and secondary pyoderma (**Figure 3**). Biopsy specimens are consistent with orthokeratotic and follicular hyperkeratosis.¹³ Cocker spaniels are the most affected breed, but other breeds are also rarely affected.¹⁴ Oral retinol (vitamin A) is reported to ameliorate clinical signs, but the optimal dose is unknown (authors have suggested 10,000 IU/dog q24h or 1000 IU/kg q24h).^{4,13} Synthetic retinoids have been reported in the treatment of primary keratinization disorders but are unlikely to be necessary in cases of true vitamin A dermatosis; moreover, the side effect profiles of these drugs warrant their use only by those familiar with their administration.



FIGURE 3. Vitamin A-responsive dermatosis lesions in cocker spaniels. Courtesy UF Veterinary Dermatology.

Hepatocutaneous Syndrome

This condition, also called *migratory necrolytic erythema* or *superficial necrolytic dermatitis*, is diagnosed infrequently in dogs and, very rarely, cats with liver dysfunction, chronic phenobarbital exposure, diabetes mellitus, or pancreatic tumors (such as glucagonomas).^{4,15,16} The syndrome has been reported in older dogs of many breeds, and evidence exists for a possible heritable predisposition in shih tzus.¹⁶

Elevated alkaline phosphatase and microcytosis are laboratory findings reported in case series, but liver values may be normal. Histologic lesions are unique with parakeratotic hyperkeratosis ("red" layer), pale and edematous keratinocytes in the stratum granulosum and stratum spinosum



FIGURE 4. Hepatocutaneous syndrome lesions of the nasal planum and feet with characteristic severe crusts and secondary inflammation. Courtesy UF Veterinary Dermatology.

(“white” layer), and stratum basale hyperplasia (“blue” layer). Distribution of lesions is similar to that in zinc dermatoses but of increased severity, and affected animals generally display nonspecific systemic signs (lethargy, inappetence) or risk factors described above (**Figure 4**). Plasma amino acid levels are often below reference intervals.¹⁷ Hepatic ultrasonography may reveal diffuse mottling or a reportedly characteristic “Swiss cheese” or “honeycomb” appearance. Median survival times with supportive treatments are less than 6 months.¹⁶

Reported treatments include¹⁶:

- Parenteral amino acid infusions (weekly, adjusted according to lesion response)
- Diets with moderate to high amounts of digestible dietary protein (>75 g/Mcal)
- Hepatic antioxidants (vitamin E, silybin, glutathione precursors)
- Oral zinc
- Essential fatty acids
- Anti-inflammatory glucocorticoids

Parenteral amino acid infusions appear to have the greatest effect on the condition, and one report suggested a reduced frequency of administration with concurrent parenteral lipid administration (2.4 g/kg amino acids + 1.4 g/kg lipid).¹⁸ The underlying cause, if identified, should be concurrently managed. High-protein diets should not be given to dogs with hepatic encephalopathy.

Vitamin E Deficiency in Cats

This condition is associated with diets high in polyunsaturated fats, such as canned seafood, that are deficient in vitamin E. Affected cats display reluctance to move and pain and are often febrile. Pansteatitis is evident grossly, and ceroid deposits within affected subcutaneous fat are characteristic of this condition (**Figure 5**).¹⁹

The vitamin E requirement is mildly increased as the polyunsaturated fat concentration of the diet increases (<5 IU per g of marine oil).²⁰ Improper food storage, heat processing, or inadequate vitamin E in high-fat diets all increase

the risk for the condition, as the degree of fatty acid oxidation is likely correlated to risk.²⁰

Diet-Related Dermatoses

These disorders are characterized by cutaneous manifestations of food intolerance or food allergy. Adverse reactions to food may also cause gastrointestinal, respiratory, or systemic signs, which are reviewed elsewhere.^{21,22} Owners commonly ascribe dermatologic symptoms to food allergy, but atopic dermatitis (AD; see **Nutritional Considerations in Diagnosing and Managing Atopic Dermatitis**) is more prevalent. Owners also often insist that dietary modification be made even when a dietary origin of clinical signs is unlikely.

Food intolerance could result from many individual predispositions, but by definition, it is nonimmunologic in origin.²² Gastrointestinal signs are most commonly ascribed to food intolerance.



FIGURE 5. Nodular pansteatitis of the mesenteric fat (top) and subcutaneous fat (bottom). Courtesy of Mike Schaer, DVM, DACVIM, DACVECC, University of Florida.

Dermatologic manifestations of food intolerance are presumptively related to dietary compounds that worsen the response to another disease, such as AD. Examples may include³⁸:

- Foods naturally high in histamine (or the amino acid precursor l-histidine), such as animal meats (eg, pork and beef—histidine), fish, tomatoes, mushrooms, spinach (histamine)
- Foods containing other amines, such as tyramine: cured or processed meats, fermented foods, dried or overripe fruits

Some foods may increase endogenous histamine release via activation of enzymatic pathways of histamine production.

Trials of diets with different primary, even if not novel, ingredients should be sufficient to determine whether food intolerance is responsible for cutaneous signs.

Food allergy is an inappropriate immunologic response to dietary antigens.²¹ Clinical signs may include erythema, epidermal scaling, patchy alopecia, miliary dermatitis (in cats), recurrent pyoderma, and urticaria (**Figure 6**). Many dogs with food allergy display perianal pruritus.²⁴ Concurrent gastrointestinal signs are variably present. Signs are often nonseasonal and unresponsive to immunosuppressive corticosteroids.

A physical examination is generally insufficient to distinguish atopic from food-associated

Nutritional Considerations in Diagnosing and Managing Atopic Dermatitis

Atopic dermatitis (AD) produces clinical signs and lesions often indistinguishable from those of food allergy (**Figure 7**). However, several nutritional modifications have been shown or suggested to influence disease severity in dogs or cats.

Dietary food trials should be performed in all atopic animals. Food allergy is a comorbidity in many atopic patients, and food allergens may worsen atopy.^{22,23} An 8-week trial is thought sufficient to identify 90% of cases.²⁴

Essential fatty acids—primarily **EPA** and **DHA**—reduce the severity of pruritus and other signs in AD.^{8,25} Clinical effects may not be documented for 4 to 12 weeks, as membrane incorporation of these dietary fats is required. EPA and DHA produce fewer inflammatory prostaglandins and leukotrienes by providing omega-3 substrate for cyclooxygenase and lipoxygenase (LOX) enzymes.⁸

Fish oil (menhaden, sardine, anchovy) is the primary commercial source of EPA and DHA. Krill oil represents a possible alternative but has not been studied in dogs or cats and is more expensive. Algal oil is not recommended because it contains primarily DHA.

A daily dose of 70 mg/kg combined EPA + DHA is recommended. This is equivalent to approximately 130 mg/kg^{0.75} or about 1.5 mg per calorie fed. One standard fish oil capsule (300 mg EPA + DHA) per 10 lb body weight provides this dose. A cyclosporine-sparing effect was reported at these doses.²⁵

Gamma-linolenic acid (GLA) is a plant-sourced omega-6 fatty acid that is converted to dihomo-

gamma-linolenic acid (DGLA).²⁶ Borage seed, evening primrose, and black currant seed oil are common sources. Unlike other omega-6 fatty acids, DGLA impairs leukotriene formation from LOX enzymes and reduces inflammation.

Doses of 10 to 50 mg GLA/kg are reported, and the high end of the dose range is well tolerated by most dogs.

- This dose is equivalent to 20 to 100 mg/kg^{0.75} or 0.2 to 1 mg GLA per calorie fed.
- The equivalent borage seed oil dose (20% to 25% GLA) is about 1 g per 10 lb body weight (high end).

Combination products of fish oil and GLA-containing oils appear efficacious in clinical studies.

Linoleic acid is a common plant-based omega-6 fatty acid. High concentrations are found in safflower and sunflower oils. Elevated doses are recommended in humans with skin disease because of positive effects on the epidermal barrier. Very high doses (1.4 g/kg) improved seborrhea in dogs,²⁷ but this dose would increase a patient's caloric intake by 25% and possibly unbalance a diet. Most commercial diets are replete in linoleic acid, so routine supplementation of such diets is not recommended.

Owners may use other oils based on anecdotal reports, but objective information is limited. No studies show positive effects of dietary coconut oil on skin health in dogs. Flax seed does not adequately increase EPA or DHA because of reduced conversion in dogs and cats, and it is not recommended for atopy.⁸ Flax seed did improve coat



FIGURE 6. Severe food allergy in a Labrador retriever with pruritus, bacterial pyoderma, and partial alopecia. Courtesy UF Veterinary Dermatology.

dermatopathy, and an estimated 30% of dogs and cats with food allergy have other dermatologic comorbidities.²¹ Pathophysiology of food allergy may be mediated by IgE, antibody-antigen complexes, or cell-mediated immunity.

Dogs and cats of all breeds are affected, and Siamese cats may be overrepresented.¹⁹ Some diets contain variable amounts of storage mites, which trigger allergic reactions in sensitized animals. Studies have not yet determined differences in response to variable diet types.²⁴

DIAGNOSIS AND TREATMENT

Diagnosis and treatment of food allergy require a diet change. Three strategies may be used for a food trial.

quality in dogs, but only transiently.⁵ Conjugated linoleic acid failed to improve canine atopy.²⁸

The following nutritional approaches can be considered, but these have less evidentiary support.

- Supplementation of nutrients affecting the epidermal barrier.
 - A diet containing increased fortification of vitamin B5 (6.5x), vitamin B3 (24x), vitamin B6 (8x), choline (1.4x), and inositol (2.8x) fed to pregnant Labrador retrievers and their puppies reduced the incidence of atopy.²⁹
 - *In vitro* effects of a combination of aloe vera, curcumin, vitamin C, and taurine decreased water diffusion across canine keratinocytes³⁰ and are incorporated into a commercial therapeutic diet for AD.
- Herbal extracts.
 - An extract containing the Chinese herbs *Glycyrrhiza uralensis*, *Paeonia lactiflora*, and *Rehmannia glutinosa* demonstrated a 25% reduction in pruritus in dogs compared with a 13% reduction in a placebo group, but this product is not available in the US market.³¹
 - Similar products are available but are not strongly regulated.³²
- Vitamin E (8 IU/kg) improved AD scoring in dogs compared with placebo.³³
- Probiotics have been suggested to have immunomodulatory properties in AD.

- *Lactobacillus* strains reduced AD symptoms in predisposed growing dogs and decreased clinical signs in adult dogs.^{34,35}
- Prebiotics and fecal microbiota transplantation have also been theorized to have similar benefits in animal patients.³⁶

Diets marketed for AD may produce differing clinical effects. Two of 3 tested diets marketed for canine AD produced significant improvement compared to a maintenance diet. Higher levels of EPA and DHA in these diets may explain the clinical effects.³⁷



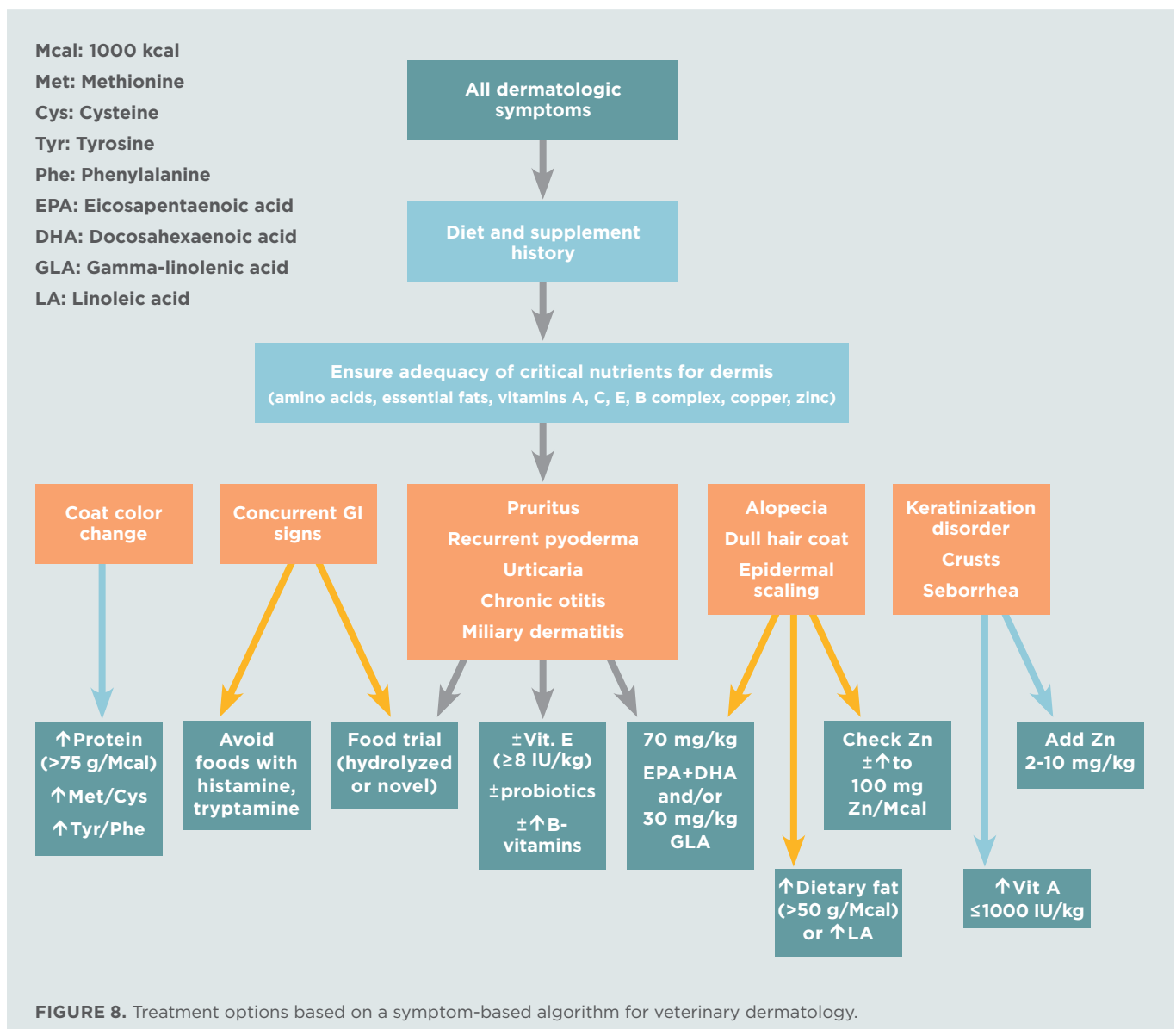
FIGURE 7. Atopic dermatitis in a young Boston terrier with erythema, urticaria, self-excoriation, pruritus, and conjunctivitis.

1. Hydrolyzed Diets

Dietary proteins are responsible for the inappropriate antigenic response in food allergies. Enzymatic hydrolysis reduces the size of these food antigens, thereby reducing IgE cross-linking and subsequent histamine response, as well as reducing epitopes for other immune responses.^{22,39} Diets vary in their degree of hydrolysis, and no product, except for prohibitively expensive pure amino acid diets, can guarantee complete reduction of antigenic stimulation.³⁹

Lower-molecular-weight diets may be best for therapeutic trials, and higher-molecular-weight hydrolyzed diets could be tolerated in some dogs that respond to the initial trial. Dogs with a known allergy to a protein should likely avoid that protein, even when hydrolyzed, to avoid issues of incomplete hydrolysis.³⁹

A diagnostic trial and improvement in clinical signs may require 8 to 12 weeks of feeding.²⁴ Other foods and flavorants should be avoided during this period. Hydrolyzed diets are expensive and often lower in protein than other diets. These factors should be considered in the nutritional plan.



2. Novel Protein Diets

These diets provide only proteins to which the specific patient has never been exposed.²² Appropriate use of such diets requires a careful and complete diet history. Over-the-counter diets containing previously rare proteins, such as alligator, kangaroo, pheasant, and bison, are available; therefore, clinicians should not assume that exotic proteins are novel for all patients. Contamination of diets during manufacturing may be more common than previously realized, and manufacturer quality control would contribute to response.^{40,41}

3. Home-Prepared Diets

These diets are popular among some owners and can be used for novel protein trials. They may be subject to less unintentional contamination with other dietary proteins, and owners may elect such diets to feel like participants in the treatment plan.

Patients with extensive diet histories or concurrent nutritional comorbidities (eg, pancreatitis) may require a custom protein or diet strategy. Unbalanced diets have historically been recommended to reduce ingredient numbers and possible antigenicity.⁴² Extended feeding (>4 weeks) of such diets might cause suboptimal levels of nutrients with effects on the epidermal barrier or turnover and immune status, but this has not been demonstrated in clinical studies.

Extrusion, canning, baking, boiling, and feeding raw could have differential effects on antigenic structure. Therefore, anecdotal responses to such diets could be related to these different methods of preparation.⁴³

Given the advantages and disadvantages for each strategy, two sequential strict food trials, using different diets, may be required to best eliminate food-responsive dermatopathies as differential diagnoses. Partial improvements should prompt clinicians to compare nutrient profiles in the event of intolerance or elevations of critical nutrients in the test diet (zinc, essential fatty acids, protein, B vitamins). Partial improvement may also suggest concurrent atopic or contact dermatitis

and should prompt additional screening for these conditions at the completion of the trial.

SUMMARY

Nutritional modification clearly affects epidermal turnover, defensive barriers, inflammatory mediators, hair growth, and important features of normal and pathologic skin. Nutritional interventions can initially be made symptomatically and further refined once a definitive diagnosis is identified. A diagnostic flowchart of nutritional considerations for common dermatologic signs is provided in **Figure 8**. All interventions should, however, be predicated on a careful diet history and thorough assessment of current feeding strategies. **TVP**

References

- Hendriks WH, Tarttelin MF, Moughan PJ. Seasonal hair growth in the adult domestic cat (*Felis catus*). *Compar Biochem Physiol Part A Physiol* 1997; 116:29-35.
- Anderson PJB, Rogers QR, Morris JG. Cats require more dietary phenylalanine or tyrosine for melanin deposition in hair than for maximal growth. *J Nutr* 2002; 132:2037-2042.
- National Research Council. *Nutrient Requirements of Dogs and Cats*. Washington DC: National Academies Press, 2006.
- Prelaud P, Harvey R. Nutritional dermatoses and the contribution of dietetics in dermatology. In Pibot P, Biourge V, Elliott D (eds). *Encyclopedia of Canine Clinical Nutrition*. Aimargues, France: Aniwa SAS, 2006; pp 58-91.
- Rees CA, Bauer JE, Burkholder WJ, et al. Effects of dietary flax seed and sunflower seed supplementation on normal canine serum polyunsaturated fatty acids and skin and hair coat condition scores. *Vet Dermatol* 2001; 12:111-117.
- Marsh KA, Ruedisueli FL, Coe SL, et al. Effects of zinc and linoleic acid supplementation on the skin and coat quality of dogs receiving a complete and balanced diet. *Vet Dermatol* 2000; 11:277-284.
- Kirby NA, Hester SL, Rees CA, et al. Skin surface lipids and skin and hair coat condition in dogs fed increased total fat diets containing polyunsaturated fatty acids. *J Anim Physiol Anim Nutr* 2009; 93:505-511.
- Bauer JE. Therapeutic use of fish oils in companion animals. *JAVMA* 2011; 239:1441-1451.
- Watson AL, Fray TR, Bailey J, et al. Dietary constituents are able to play a beneficial role in canine epidermal barrier function. *Exp Dermatol* 2006; 15:74-81.
- White SD, Bourdeau P, Rosychuk RAW, et al. Zinc-responsive dermatosis in dogs: 41 cases and literature review. *Vet Dermatol* 2001; 12:101-109.
- McEwan NA, McNeil PE, Thompson H, et al. Diagnostic features, confirmation and disease progression in 28 cases of lethal acrodermatitis of bull terriers. *J Sm Anim Pract* 2000; 41:501-507.
- Van den Broek AHM, Thoday KL. Skin disease in dogs associated with zinc deficiency: a report of five cases. *J Sm Anim Pract* 1986; 27:313-323.
- Hensel P. Nutrition and skin diseases in veterinary medicine. *Clin Dermatol* 2010; 28:686-693.
- Ihrke PJ, Goldschmidt MH. Vitamin A-responsive dermatosis in the dog. *JAVMA* 1983; 182(7):687-690.
- Byrne KP. Metabolic epidermal necrosis-hepatocutaneous syndrome. *Vet Clin North Am Sm Anim Pract* 1999; 29(6):1337-1355.
- Hall-Fonte DL, Center SA, McDonough SP, et al. Hepatocutaneous syndrome in Shih Tzus: 31 cases (1996-2014). *JAVMA* 2016; 248:802-813.

Justin Shmalberg

Justin Shmalberg, DVM, DACVN, DACVSMR, is a clinical associate professor of integrative medicine at University of Florida College of Veterinary Medicine. His service specializes in the incorporation of nutrition, rehabilitation, hyperbaric oxygen therapy, and acupuncture with conventional care; he holds certifications in acupuncture and herbal medicine. Dr. Shmalberg's research interests include nutritional oncology, sports and rehabilitative nutrition, evaluations of new small animal dietary trends, and the safety and efficacy of Chinese herbal products. He received his DVM from University of Wisconsin-Madison. Dr. Shmalberg completed an internship in veterinary acupuncture at University of Florida along with a residency in small animal clinical nutrition.



17. Outerbridge CA, Marks S, Rogers QR. Plasma amino acid concentrations in 36 dogs with histologically confirmed superficial necrolytic dermatitis. *Vet Dermatol* 2002; 13:177-186.
18. Bach JF, Glasser SA. A case of necrolytic migratory erythema managed for 24 months with intravenous amino acid and lipid infusions. *Can Vet J* 2013; 54:873-875.
19. Mueller RS, Dethioux F. Nutritional dermatoses and the contribution of dietetics in dermatology. In Pibot P, Biourge V, Elliott D (eds). *Encyclopedia of Feline Clinical Nutrition*. Aimargues, France: Aniwa SAS, 2008; pp 51-75.
20. Hendriks WH, Wu YB, Shields RG, et al. Vitamin E requirement of adult cats increases slightly with high dietary intake of polyunsaturated fatty acids. *J Nutr* 2002; 132:1613S-1615S.
21. Verlinden A, Hesta M, Millet S, et al. Food allergy in dogs and cats: a review. *Crit Rev Food Sci Nutr* 2006; 46:259-273.
22. Gaschen FP, Merchant SR. Adverse food reactions in dogs and cats. *Vet Clin North Am Sm Anim Pract* 2011; 41:361-379.
23. Pucheu-Hastoin CM, Bizikova P, Eisenschenk MNC, et al. Review: The role of antibodies, autoantigens and food allergens in canine atopic dermatitis. *Vet Dermatol* 2015; 26:115-130.
24. Olivry T, DeBoer DJ, Favrot C, et al. Treatment of canine atopic dermatitis: 2015 updated guidelines from the International Committee on Allergic Diseases of Animals (ICADA). *BMC Vet Res* 2015; 11:210.
25. Müller MR, Linek M, Löwenstein C, et al. Evaluation of cyclosporine-sparing effects of polyunsaturated fatty acids in the treatment of canine atopic dermatitis. *Vet J* 2016; 210:77-81.
26. Sævik BK, Bergvall K, Holm BR, et al. A randomized, controlled study to evaluate the steroid sparing effect of essential fatty acid supplementation in the treatment of canine atopic dermatitis. *Vet Dermatol* 2004; 15:137-145.
27. Campbell KL, Uhlend CF, Dorn GP. Effects of oral sunflower oil on serum and cutaneous fatty acid concentration profiles in seborrheic dogs. *Vet Dermatol* 1992; 3:29-35.
28. Noli C, Carta G, Cordeddu L, et al. Conjugated linoleic acid and black currant seed oil in the treatment of canine atopic dermatitis: a preliminary report. *Vet J* 2007; 173:413-421.
29. van Beeck FL, Watson A, Bos M, et al. The effect of long-term feeding of skin barrier-fortified diets on the owner-assessed incidence of atopic dermatitis symptoms in Labrador retrievers. *J Nutr Sci* 2015; 4:e5.
30. Fray TR, Watson AL, Croft JM, et al. A combination of aloe vera, curcumin, vitamin C, and taurine increases canine fibroblast migration and decreases tritiated water diffusion across canine keratinocytes in vitro. *J Nutr* 2004; 134:2117S-2119S.
31. Nagle TM, Torres SM, Horne KL, et al. A randomized, double-blind, placebo-controlled trial to investigate the efficacy and safety of a Chinese herbal product (P07P) for the treatment of canine atopic dermatitis. *Vet Dermatol* 2001; 12:265-274.
32. Shmalberg J. Surveying supplements: current trends, research, and recommendations. *Today's Veterinary Practice* 2014; 4(3):79-84.
33. Kapun AP, Salobir J, Levart A, et al. Vitamin E supplementation in canine atopic dermatitis: improvement of clinical signs and effects on oxidative stress markers. *Vet Rec* 2014; 175:560.
34. Marsella R, Santoro D, Ahrens K. Early exposure to probiotics in a canine model of atopic dermatitis has long-term clinical and immunological effects. *Vet Immunol Immunopathol* 2012; 146:185-189.
35. Kim H, Rather IA, Kim H, et al. A double-blind, placebo controlled-trial of a probiotic strain *Lactobacillus sakei* Probio-65 for the prevention of canine atopic dermatitis. *J Microbiol Biotechnol* 2015; 25:1966-1969.
36. Craig JM. Atopic dermatitis and the intestinal microbiota in humans and dogs. *Vet Med Sci* 2016. doi: 10.1002/vms3.24
37. Glos K, Linek M, Loewenstein C, et al. The efficacy of commercially available veterinary diets recommended for dogs with atopic dermatitis. *Vet Dermatol* 2008; 19:280-287.
38. Maintz L, Novak N. Histamine and histamine intolerance. *Am J Clin Nutr* 2007; 85:1185-1196.
39. Olivry T, Bizikova P. A systematic review of the evidence of reduced allergenicity and clinical benefit of food hydrolysates in dogs with cutaneous adverse food reactions. *Vet Dermatol* 2010; 21:32-41.
40. Willis-Mahn C, Remillard R, Tater K. ELISA testing for soy antigens in dry dog foods used in dietary elimination trials. *JAHA* 2014; 50:383-389.
41. Raditic DM, Remillard RL, Tater KC. ELISA testing for common food antigens in four dry dog foods used in dietary elimination trials. *J Anim Physiol Anim Nutr* 2011; 95:90-97.
42. Harvey RG. Food allergy and dietary intolerance in dogs: a report of 25 cases. *J Small Anim Pract* 1993; 34:175-179.
43. Davis PJ, Smales CM, James DC. How can thermal processing modify the antigenicity of proteins? *Allergy* 2001; 56:56-60.