Skin and Hair Disorders

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"Dermatoses affecting various species of animals are more commonly associated with malnutrition than they are with . . . a good state of nutrition."
F. Kral and B.J. Novak, Veterinary Dermatology, 1953

CLINICAL IMPORTANCE

Very little information is available concerning the demographics of canine and feline skin and hair disorders. Surveys and textbooks suggest that skin disorders are the most common reason for patient visits to the veterinarian's office (Scott et al, 2001). Surveys also indicate that 15 to 25% of all small animal practice activity is involved with the diagnosis and treatment of problems with the skin and coat (Scott et al, 1995).

The most commonly diagnosed canine skin disorders are: 1) allergy (flea-bite hypersensitivity, atopic dermatitis), 2) cutaneous neoplasms, 3) bacterial pyoderma, 4) seborrhea, 5) parasitic dermatoses, 6) adverse reactions to food (food hypersensitivity or food intolerance), 7) immune-mediated dermatoses and 8) endocrine dermatoses (Sischo et al, 1989; Scott and Paradis, 1990). The most common feline skin disorders are: 1) abscesses, 2) parasitic dermatoses, 3) allergy (flea-bite hypersensitivity, atopic dermatitis), 4) miliary dermatitis, 5) eosinophilic granuloma complex, 6) fungal infections, 7) adverse reactions to food, 8) psychogenic dermatoses, 9) seborrheic conditions, 10) neoplastic tumors and 11) immune-mediated dermatoses (Scott and Paradis, 1990; Nesbitt, 1982).

Clearly, skin and hair disorders are an important part of small animal practice; bacterial infections, ectoparasitism, allergies, fungal infections and neoplasia are common problems. Aside from adverse reactions to food, nutritional skin diseases in pets fed nutritionally adequate commercial pet food appear to be very uncommon. However, the skin and coat can be affected by many nutritional factors (**Table 32-1**), and many pet owners want to improve the quality and appearance of their pet's coat. The tactile and visual interactions between people and pets are among the greatest pleasures of the companion animal-human bond (Credille et al, 2000). This emphasizes the importance of understanding the nutritional factors that affect normal skin and hair and the nutritional factors that should be investigated in patients with skin disorders. This chapter discusses the nutritional factors that affect skin and hair, except for adverse reactions to food, which are specifically addressed in Chapter 31.

This chapter is divided into two sections: 1) nutrient-deficient dermatoses and 2) fatty acids for inflammatory skin disease. The first section covers dermatoses related to nutrient deficiencies. These deficiencies usually occur when pets in a nutritionally demanding lifestage are fed homemade foods, poor quality commercial foods, commercial foods that contain nutrient excesses or even high quality commercial foods that are inappropriately supplemented. Breed predilection can also be a factor. The second section focuses on the use of antiinflammatory fatty acids in the management of skin diseases that have an inflammatory or pruritic component. These dermatoses are responsive or partially responsive to antiinflammatory intervention. Many commonly diagnosed skin disorders have an inflammatory component.

Table 32-1. Key nutritional factors for foods and supplements for dogs and cats with nutrient-responsive dermatoses.

Factors	Associated conditions	Nutritional recommendations

Protein and fat	Keratinization abnormalities
	Loss of normal hair color
	Secondary bacterial or yeast infection
	Impaired wound healing
	Decubital ulcers
	Telogen defluxion
	Anagen defluxion
		Avoid protein and energy deficiency
		Adult maintenance
		    Dogs: Protein = 25 to 30% dry matter (DM)
		                    Fat = 10 to 15% DM
		    Cats: Protein = 30 to 45% DM
		                    Fat = 10 to 15% DM
		Growth/lactation
		    Dogs: Protein = 30 to 35% DM
		                    Fat = 15 to 30% DM
		    Cats: Protein = 35 to 50% DM
		                    Fat = 20 to 35% DM
		Phenylalanine + tyrosine >1.3% DM
		Use a food with DM digestibility >80%

Essential fatty acids (EFA)	Excessive scales (seborrhea sicca)
	Alopecia
	Dry, dull coat
	Lack of normal hair growth
	Erythroderma
	Interdigital exudation
		Avoid fatty acid deficiency
		    Dogs: Linoleic acid >1.0% DM
		    Cats: Linoleic acid >0.5% DM
		Some dogs and cats respond to levels in excess of those listed above
		Provide adequate levels and availability of zinc, B-complex
		    vitamins and vitamin E to ensure adequate use of EFA

Zinc	Alopecia
	Skin ulceration
	Dermatitis
	Paronychia
	Footpad disease
	Slow hair growth
	Buccal margin ulceration
	Hyperkeratotic plaques
	Secondary bacterial or yeast infection
		Avoid zinc deficiency
		    Dogs: 100 to 200 mg/kg food DM
		    Cats: 50 to 150 mg/kg food DM
		Avoid excess calcium
		    Higher levels of zinc are required in foods with calcium >1.5% DM
		Avoid excess copper (copper <200 mg/kg food DM)
		Avoid EFA deficiency (see above)
		Zinc supplementation (Do not give with food)
		    Zinc sulfate: 10 mg/kg body weight/day per os
		                           10 to 15 mg/kg body weight/week IV
		    Zinc methionine: 2 mg/kg body weight/day

Copper	Loss of normal color
	Dull or rough coat
	Reduced density of hair
	Alopecia
		Avoid copper deficiency
		    Dogs: >5 to 10 mg/kg food DM
		    Cats: >15 mg/kg food DM
		Avoid excess zinc (zinc <1,000 mg/kg food DM)
		Avoid ingredients that have low copper availability
		    Copper oxide
		    Liver from simple-stomached mammals
		Avoid excess calcium
		    Higher levels of copper are required in foods with
		        calcium >1.5% DM

Vitamin A	Seborrheic skin disease
	    (mainly cocker spaniel breed)
	Keratinization disorders
	Chin acne
	Nasodigital hyperkeratosis
	Ear margin seborrhea/dermatosis
	Callus
	Actinic keratosis
	Cutaneous neoplasms
	Schnauzer comedo syndrome
	Sebaceous adenitis
	Lamellar ichthyosis
		Treatment with retinoids (**Table 32-5**):
		Vitamin A alcohol
		    625 to 1,000 U/kg body weight, q24h, per os
		    10,000 U q24h, per os (cocker spaniel, miniature schnauzer)
		    50,000 U q24h, per os (Labrador retriever)
		Tretinoin
		    Apply topically q12 to 24h
		Isotretinoin
		    1 to 3 mg/kg body weight, q24h, per os
		Acitretin
		    0.75 to 1.0 mg/kg body weight, q24h, per os

Vitamin E	Discoid lupus erythematosus Systemic lupus erythematosus Pemphigus erythematosus Sterile panniculitis Acanthosis nigricans Dermatomyositis Ear margin vasculitis	Treatment with vitamin E: Dogs: 200 to 800 IU twice daily, per os

NUTRIENT-DEFICIENT DERMATOSES

Patient Assessment
History

The signalment (species, breed, age, gender, reproductive status, hair color) is an important part of the historical information that should be obtained for patients with dermatologic problems, especially those with possible nutritional disorders (**Table 32-2**). Both dogs and cats develop nutritionally related skin and hair disorders, although certain conditions such as zinc-responsive dermatoses are best characterized in dogs.

The age of the patient is important; most skin and coat changes due to nutritional deficiencies occur in young growing animals or adult females during gestation and lactation. The requirement for most nutrients is highest during growth and reproduction, which accounts for the increase in nutritionally related skin and hair problems seen during these lifestages. As an example, a biotin-deficient food will cause dermatitis, alopecia, dull fur and achromotrichia when fed to young growing kittens but will not cause similar clinical signs when fed to non-lactating adult cats (Pastoor et al, 1991). Many other examples of nutritional skin diseases exist that occur during periods of increased nutritional demand but do not occur during normal adult lifestages. This age-related phenomenon is complicated by the fact that congenital defects of the integument and certain parasitic, fungal and bacterial infections of the skin are also more common in dogs and cats younger than six months. Gender and reproductive status affect the prevalence of certain skin problems, but they are not usual risk factors in nutritional skin disorders, unless the increased nutritional demands of pregnancy or lactation are present.

The clinician should obtain a complete medical history in all cases. Specific details of the dermatologic history are found elsewhere (Scott et al, 2001). The nutritional history should focus on the adequacy of the specific food for the patient's lifestage, and types and dosages of nutritional supplements. The veterinarian or a veterinary nutritionist should evaluate homemade foods for nutritional adequacy (Chapter 10) because nutrient deficiencies or imbalances are more likely to occur in dogs or cats eating homemade vs. commercial foods. Excessive nutrient levels in food can cause skin disease due to direct toxicosis or interaction/interference with the use of other nutrients in the food.

Physical Examination

A comprehensive physical examination that evaluates all body systems should be performed on patients with skin or hair disease. Internal disease is often manifested as skin and coat disease, and this diagnostic possibility should not be overlooked by concentrating on the integumentary changes alone.

The skin can be affected by many nutritional factors, but usually responds in a limited number of ways. The cutaneous changes associated with nutritional abnormalities are often indistinguishable from those caused by other more common skin diseases. Changes that raise the suspicion for nutritional abnormalities include: 1) a sparse, dry, dull and brittle coat with hairs that epilate easily, 2) slow hair growth or regrowth from areas that have been clipped, 3) abnormal scale accumulation (seborrhea sicca), 4) loss of hair, erythema or crusting in areas of friction or stretch such as the distal extremities, 5) decubital ulcers and poor wound healing and 6) loss of normal hair color. Primary lesions such as papules and pustules rarely occur with nutritional abnormalities, but can occur with bacterial pyoderma secondary to nutritional, allergic or other underlying problems.

Laboratory and Other Clinical Information

Common laboratory evaluations including a complete blood count, serum biochemistry profile, urinalysis and thyroid panel

Table 32-2. Breed predilection for non-neoplastic skin diseases often managed by food changes or supplementation.*

Breed	Disease
Airedale terrier	Atopic dermatitis
Akita	Sebaceous adenitis
Basenji	Atopic dermatitis
Basset hound	Atopic dermatitis
Beagle	Atopic dermatitis
Boston terrier	Atopic dermatitis
Boxer	Adverse reactions to food
	Atopic dermatitis
Bull terrier	Acrodermatitis
	Atopic dermatitis
	Zinc-responsive dermatosis
Chesapeake Bay retriever	Atopic dermatitis
Dalmatian	Atopic dermatitis
English bulldog	Atopic dermatitis
German shepherd dog	Adverse reactions to food
	Atopic dermatitis
	Seborrhea, primary
Golden retriever	Atopic dermatitis
Gordon setter	Atopic dermatitis
Irish setter	Atopic dermatitis
	Seborrhea, primary
Labrador retriever	Adverse reactions to food
	Atopic dermatitis
	Seborrhea, primary
Lhasa apso	Atopic dermatitis
Malamute	Zinc-responsive dermatosis
Old English sheepdog	Atopic dermatitis
Poodle, standard	Sebaceous adenitis
Pug	Atopic dermatitis
Schnauzer, miniature	Atopic dermatitis
Shar Pei	Adverse reactions to food
	Atopic dermatitis
Shih Tzu	Atopic dermatitis
Siberian husky	Zinc-responsive dermatosis
Spaniels	Adverse reactions to food
	Atopic dermatitis (American cocker)
	Seborrhea, primary
Terriers	Atopic dermatitis
Vizsla	Sebaceous adenitis

*Atopic dermatitis is often managed with fatty acid supplementation, sebaceous adenitis and primary seborrhea with retinoid supplementation, zinc-responsive dermatosis with zinc supplementation and adverse reactions to food with dietary changes. Specific nutrient deficiencies are usually not breed-specific.

are rarely helpful in evaluating nutritional skin disease. However, these tests can be used to rule out internal or metabolic diseases as causes of cutaneous problems.

Routine laboratory procedures for patients with dermatologic problems include skin scrapings for parasites, hair examination, cytologic examination of tissue or fluids, fungal culture, bacterial culture and biopsy for dermatohistopathologic examination. Of these procedures, hair examination and dermatohistopathology are most helpful for evaluation of potential nutritional problems.

HAIR EXAMINATION

Plucking hairs from the skin and examining them microscopically is termed trichography. This procedure helps diagnose a number of conditions including nutritional diseases. Trichography is performed by grasping a small number of

Box 32-1. The Hair Cycle.

HAIR FOLLICLE STRUCTURE

Most omnivores and herbivores have "simple" hair follicles, which means that each infundibulum contains a single hair shaft. In contrast, dogs, cats and carnivores have "compound" hair follicles, where multiple follicles grow closely together and share a common infundibulum; multiple hairs exit through a common opening. By convention, the largest hairs in a compound follicle are called primary or guard hairs and the smaller hairs that make up the majority of the hair shafts in a compound follicle are called secondary or undercoat hairs. There is a distinct orientation of primary and secondary hairs. Primary hairs are always the most cranial (toward the head) with secondary hairs caudal (toward the rear) of the primary hairs. The secondary hairs that are closest to the primary hairs are the largest and become progressively smaller the more caudally they are positioned. In this way, hair follicles are designed so that hairs will lie down smoothly, with guard hairs on top of the fine undercoat. The ratio of secondary to primary hairs can be greater than 10 to 1.

THE HAIR CYCLE

The growth of hair is cyclical and each cycle consists of an active growth phase (anagen), a transitional involutionary phase between active and no growth (catagen) and a stage of senescence (telogen), during which hair is retained in the follicle. The length of time required to complete the hair cycle varies between different species and is different among dog breeds. In human scalp hair, anagen is the longest phase of the cycle; thus, hair grows almost constantly.

In most mammals, the hair cycle is telogen based; hairs grow to a predetermined length and then enter a long period of inactivity during which the telogen hair follicle firmly retains the hair shaft. Most dogs and cats appear to have telogen-dominant hair cycles in which the hair shafts are retained in telogen follicles for long periods of time. How long the follicle remains in telogen appears to be a breed-specific phenomenon. Some breeds of dogs, such as poodles and schnauzers, have anagen-dominant hair cycles with hair that grows almost constantly. Hair follicles have an intrinsic rhythm that can be altered by intrinsic (local growth factors, cytokines) and extrinsic (photoperiod, hormones, nutrition) factors.

One theory for why many canine breeds have such a long telogen phase is conservation of energy and protein required for active hair growth. For example, Nordic breeds appear to have a longer telogen phase than other canine breeds. They need a thick coat to provide insulation during cold weather and conserve the energy and protein needed to grow hair during the winter months when food may be more difficult to obtain. These breeds may benefit from maintaining hair in a state of "suspended animation." Preliminary studies have been conducted on the nutritional effects on canine hair follicles of various breeds. Further studies are needed to document how nutrition might influence hair follicle function, the hair cycle of various breeds and shedding.

The Bibliography for **Box 32-1** can be found at www.markmorris.org.

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hairs with the fingertips or hemostats, pulling them out completely, placing them on a microscope slide, adding mineral oil and examining them with the low-power objective of the microscope. One study evaluated the impact of anatomic location on trichogram analysis. Results of this study indicated that the shoulder was the site of choice for plucking hair (Diaz et al, 2004).

The hair bulbs are examined first. Hairs do not grow continuously but rather in cycles. Each cycle consists of a growing period (anagen), during which the follicle is actively growing hair, a transitional period (catagen) and a resting period (telogen), during which the hair is retained in the follicle as a dead or club hair that is subsequently lost (Scott et al, 2001). More details about hair follicles and normal hair-growth cycles can be found in **Box 32-1**. Anagen hair bulbs are rounded, smooth, shiny, glistening, often pigmented and soft, so the root may bend. In some cases, the end of the anagen bulb is tightly attached to the dermal papilla and when plucked the hair appears squared at the tip with a slight flair (i.e., like a "pant's leg"). Telogen bulbs are club- or spear-shaped, rough-surfaced, nonpigmented and generally straight. Normal adult dogs and cats have a mixture of anagen and telogen hairs, the ratio of which varies with the season and other factors. Estimation of the ratio of anagen to telogen hair bulbs can be useful. All the hair of normal patients should not be in telogen; this finding

suggests a diagnosis of telogen defluxion or follicular arrest. Inappropriate numbers of telogen hairs (e.g., mostly telogen hairs during the summer when the ratio should be about 50:50) suggest a diagnosis of nutritional, endocrine or metabolic disease (Scott et al, 2001). In people, the ratio of telogen to anagen hair increases with prolonged protein deficiency (Bradfield et al, 1967). Unfortunately, well-established normal trichogram values are not available in veterinary medicine, limiting their usefulness. The use of site, age, breed and climate matched controls, if possible, may increase the usefulness of this diagnostic procedure.

Examination of the hair shaft follows bulbar examination. A normal hair shaft is uniform in diameter and tapers gently to the tip. The hairs may be straight or twisted depending on the coat type of the patient. All hairs should have a clearly discernible cuticle, and a sharply demarcated cortex and medulla. Hair pigmentation depends on coat color and breed. Hairs that are inappropriately curled, misshapen and malformed suggest an underlying nutritional or metabolic disease (Scott et al, 2001). When unusual pigmentation is observed, external sources (salivary staining, chemicals, topical medications), nutritional disorders, color dilution/color mutant disorders and endocrine disorders should be considered (Scott et al, 2001).

Hairs with a normal shaft that are suddenly and cleanly broken indicate external trauma from licking, scratching or groom-

ing. Breakage of hairs with abnormal shafts suggests nutritional disorders, dermatophytosis or congenitohereditary disorders such as color dilution alopecia. Morphologic changes in the hair bulb and hair diameter are sensitive indicators of overall protein status. Hair bulb atrophy, constriction and hair depigmentation may be seen in people after as little as two weeks of protein deprivation (Bradfield et al, 1967). Protein deprivation may not produce changes as rapidly in dog and cat hair because the hair in these species spends more time in telogen and less time in anagen.

BIOPSY AND DERMATOHISTOPATHOLOGY

The following are general guidelines for when a skin biopsy should be performed: 1) all obviously neoplastic or suspected neoplastic lesions, 2) all persistent ulcerations, 3) any case involving a major disease that is most readily diagnosed by biopsy (e.g., immune-mediated skin disease), 4) a dermatosis that is unresponsive to conventional therapy, 5) any unusual or serious dermatosis and 6) vesicular dermatitis. Some nutritional skin diseases, such as zinc-responsive dermatosis, have clearly delineated histopathologic lesions that are easily recognized during microscopic examination of a skin biopsy specimen. In general, the skin should be biopsied within three weeks for any dermatosis that does not respond to appropriate therapy. This includes those dermatoses that do not respond to initial management with a food change or supplementation.

CHEMICAL COMPOSITION OF HAIR

Some investigators and clinicians have advocated the use of chemical analysis of hair as a useful diagnostic technique. Hair is a complex tissue consisting of several morphologic components (epicuticle, exocuticle, endocuticle, medulla); each component has a different chemical composition. Genetic factors, nutrition, environmental effects and cosmetic treatment affect the chemical composition of hair (Robbins, 1988; Stafforst, 1982; Mundt and Stafforst, 1987). These complex factors and the expense of analysis make it unlikely that chemical composition of hair will be routinely useful as a diagnostic technique.

Hair, depending on its moisture content, consists of 65 to 95% protein. The remaining constituents are water, lipids, pigment and trace elements. The amount of moisture in hair plays a critical role in its physical and cosmetic properties. Moisture of hair often depends on relative humidity; as relative humidity increases from 29 to 70%, the approximate moisture content of hair increases from 6 to 14% (Robbins, 1988).

Hair consists of surface (external) lipid and internal lipid. In addition, part of the internal lipid is free lipid and part is structural lipid of the cell membrane complex. Skin surface lipids of cats and dogs contain more sterol esters, free cholesterol esters and diester waxes, but fewer triglycerides, monoglycerides, free fatty acids, monoester waxes and squalene than do skin surface lipids of people (Scott et al, 2001; Dunstan et al, 2000; Watson, 2003). It has been suggested that the skin surface lipids of cats and dogs are mainly of epidermal origin, whereas those of people are mainly of sebaceous gland origin (Scott et al, 2001).

Hair generally has very low mineral content (less than 1%),

and it is difficult to determine whether this inorganic matter is derived from extraneous sources or whether it arises during fiber synthesis. Hair length and pigmentation intensity have been reported to affect concentrations of zinc and other trace elements, as well as certain macro elements, in canine hair (Stafforst, 1982; Mundt and Stafforst, 1987). Zinc and copper concentrations in hair from normal cats have also been documented (van den Broek et al, 1992).

SKIN AND HAIR BIOPHYSICAL PARAMETERS

Several studies have evaluated typical skin and hair biophysical parameters in cats and dogs such as skin pH, thickness, hydration, elasticity, transepidermal water loss, coat thickness, hair regrowth and hair length. These parameters are used most often in research studies and are not yet available for routine clinical practice. The skin and hair biophysical parameters measured to date differ widely according to breed, gender, gonadal status and age of the animal, as well as, the season of the year, limiting their routine use (Young et al, 2003; Schroeder et al, 2003; Watson et al, 2001, 2002; Cline et al, 2003; Matousek and Campbell, 2002; Hester et al, 2003; Bourdeau et al, 2004, 2004a; Diaz et al, 2003). Various nutritional studies using these parameters have been published or reported and are described below.

Risk Factors for Nutritionally Related Skin Disease

Genotype, lifestage, food type and food supplementation are risk factors for nutritionally related skin disease. Breed predilection determines the prevalence of some skin disorders. Tables of common skin diseases categorized by breed are readily available (**Table 32-2**). In general, more than 30 canine breeds are at increased risk for skin diseases (Ihrke and Franti, 1985). The nutrient-sensitive skin diseases such as zinc-responsive dermatoses and retinoid-responsive dermatoses often occur in specific breeds. As an example, one form of zinc-responsive dermatosis is frequently seen in arctic-type breeds such as malamutes and Siberian husky dogs.

As mentioned before, nutrient deficiencies that cause skin disease are more likely to occur during growth, gestation, lactation and illness when nutritional requirements are highest.

Some dry, commercial, generic, private label brand and grocery pet foods have lower fat content, lower nutrient digestibility and higher mineral content than other grocery and specialty brands. Low amounts of fat and poor-quality fat are risk factors for essential fatty acid (EFA) deficiency; poor nutrient digestibility contributes to protein-energy malnutrition, especially during growth and lactation; and high levels of minerals such as calcium inhibit the absorption of nutrients such as zinc, which are essential for normal, healthy skin.

A pet that obtains most of its nutrients from homemade foods is at increased risk for several nutritional problems (Chapter 10). In general, homemade foods are more likely to lack adequate calcium, EFA, certain vitamins and other micronutrients (Roudebush and Cowell, 1992). Homemade foods should include: 1) a calcium source such as bone meal,

Box 32-2. Red Coat Syndrome.

The quantity and type of melanin pigments synthesized by follicular melanocytes and deposited in keratinocytes are the prime determinant of mammalian hair color. Hair or fur color is genetically controlled but can be affected by various extrinsic factors, including nutrition. The two melanin pigments, eumelanin and phaeomelanin, are synthesized from a common precursor, dopaquinone, which is a product of tyrosine oxidation.

Reports from breeders and pet owners have identified some cats and dogs that have had their coat change from black to a reddish-brown color or have had hair stripes become less noticeable. Controlled studies in cats and dogs suggest that dietary deficiency of the amino acid tyrosine or its precursor phenylalanine is a significant factor causing black hair to change to a reddish-brown color. When tyrosine is limiting, there is insufficient dopaquinone for full expression of eumelanin formation and the yellow to reddish-brown phaeomelanin is the predominant pigment observed.

Tyrosine has not been regarded as an essential amino acid because phenylalanine is metabolized to tyrosine in all mammals and can supply the total tyrosine needs. Tyrosine contributes to the total aromatic amino acid (phenylalanine plus tyrosine) requirement and can spare about half the phenylalanine requirement. This appears to be a unique situation in which the need for a dispensable amino acid to support maximal melanin synthesis in cats and dogs is much greater than that required for nitrogen balance or maximal growth. Dietary phenylalanine plus tyrosine levels greater than 2% dry matter or addition of L-tyrosine to the food should provide optimal amino acid levels for maximal melanin synthesis in cats and dogs.

The Bibliography for **Box 32-2** can be found at www.markmorris.org.

oyster shell or dicalcium phosphate, 2) a source of EFA such as corn oil, safflower oil or some other vegetable oil and 3) a multivitamin-trace mineral supplement. Also, homemade cat foods should be supplemented with taurine. The final risk factor for nutritionally related skin disease is oversupplementation using naturally occurring foods or commercial supplements. Vitamin A toxicosis is associated with excessive use of liver as a supplement. High levels of minerals such as calcium in commercial supplements can interfere with absorption of essential trace elements such as zinc.

The Skin as a Metabolic Organ

The skin is the largest organ of the body and the anatomic and physiologic barrier between the animal and its environment. The skin protects against water loss and physical, chemical and microbiologic injuries while its sensory components perceive heat, cold, pain, touch, pruritus and pressure (Scott et al, 2001). In addition, the skin is contiguous with several internal organs and may reflect internal pathologic processes. The subcutis, skin and hair of a newborn puppy represent 24% of its body weight, which decreases in some breeds to only 12 to 14% of mature body weight (Miller et al, 1964).

The skin and coat significantly influence nutrient requirements. The ability of an animal's coat to regulate body temperature and energy requirements in cold environments correlates closely with hair length, thickness and density and with the medullation of individual hair fibers. In general, coats composed of long, fine, poorly medullated fibers are the most efficient for thermal insulation at low environmental temperatures and thus help modulate energy requirements. The skin also influences water requirements by minimizing transepidermal moisture loss. Loss of this normal barrier function as a result of fatty acid deficiency can increase a patient's water requirement, which is clinically manifested as polydipsia (Burr and Burr, 1929, 1930; Basnayake and Sinclair, 1956).

The hair cycle, and thus the coat, is influenced by the general state of health, genetics, photoperiod, ambient temperature, hormones, nutrition and poorly understood intrinsic factors (Scott et al, 2001; Scott, 1990). **Box 32-1** provides more details.

Dog breeds can be classified as having high, moderate and low weights of hair. Longhaired breeds with relatively large body surface areas per body weight, such as Pomeranians, have the largest relative amount of hair. Estimates indicate that as much as 30% of protein in food is needed to maintain daily hair growth in small breeds with long coats (Stafforst, 1982; Mundt and Stafforst, 1987). On the other hand, larger dogs with short coats may use less than 10% of food protein to maintain daily hair growth (Stafforst, 1982; Mundt and Stafforst, 1987). Whether dogs of similar body surface areas have different requirements for protein and other nutrients based solely on their type of coat is unknown, but of possible clinical significance. For example, there may be differences in nutrient requirements during peak hair growth for a Pomeranian vs. a Chihuahua or an Old English sheepdog vs. a German shorthaired pointer based on coat type alone.

The epidermis has a renewable cell population. Keratinocytes migrate from the mitotically active pool in the basal layer of the epidermis, through the spinous layer and granular layer, and finally into the superficial stratum corneum, followed by normal exfoliation. The normal canine epidermis has a very slowly renewing cell population. Only 1.5% of epidermal basal cells undergo DNA replication at any point in time (Kwochka and Rademakers, 1989; Kwochka, 1990, 1991). In dogs, it takes approximately 22 days for cells to migrate from the basal layer to, but not through, the stratum corneum.

The upper external root sheath of the hair follicle and sebaceous gland have essentially the same cell kinetic growth characteristics as the surface epidermis (Kwochka and Rademakers, 1989; Kwochka, 1990, 1991). Conversely, the root matrix of anagen hairs is one of the most rapidly renewing cell populations of the body (Kwochka, 1990). In actively growing hair, up to 24% of cells are undergoing DNA replication.

Key Nutritional Factors

The key nutritional factors for foods and nutritional supplements and the recommended amounts and doses for dogs and cats with nutrient deficiency dermatologic problems are sum-

marized in **Table 32-1** and discussed in more detail below.

PROTEIN AND FAT

As mentioned previously, the integument is a metabolically active organ that is affected by the nutritional status of the animal. Protein and energy are required for the development of new hair and skin; fat is the most concentrated source of dietary energy. Developing hair requires sulfur-containing and other amino acids. Therefore, for normal skin and hair, it is important for the pet's food to provide optimal protein quantity, quality (appropriate levels of essential amino acids) and digestibility. Dogs and cats have increased protein and energy requirements during growth, gestation, lactation and some illnesses. Abnormal skin and hair will often be noted if nutritionally inadequate foods are fed during these stages (Ralston Purina Company, 1987; Huber et al, 1991). Key nutritional factor profiles for various lifestages of dogs and cats are listed in Chapters 13 through 24. Optimal levels of protein and fat, and minimum dry matter (DM) digestibility of foods for dogs and cats with skin and hair disorders are listed in **Table 32-1**.

Foods inadequate in protein and energy can cause keratinization abnormalities, depigmentation of hair and changes in epidermal and sebaceous lipids (**Box 32-2**). The skin loses its protective barrier function in patients with protein-energy malnutrition and becomes more susceptible to secondary bacterial or yeast infection. Impaired wound healing and decubital ulcers are also sequelae to protein-energy malnutrition. Protein-deficient dogs and cats have patchy alopecia and coats that are dry, dull and brittle.

Telogen defluxion is usually recognized as hair loss associated with a stressful event (e.g., pregnancy, severe illness, surgery) that causes the abrupt, premature cessation of growth of many anagen hair follicles and the synchronization of these hair follicles in catagen, then in telogen (Scott et al, 2001; Harvey, 1994). Short-term increased requirements of energy, protein and other nutrients during growth, gestation, lactation and illness may cause telogen defluxion if appropriate nutritional changes are not instituted. Bitches and queens in late gestation and lactation, and growing puppies and kittens are at risk unless they are fed nutritionally balanced, highly digestible foods that meet their increased nutritional requirements.

Anagen defluxion is a sudden loss of hair due to an unusual event (e.g., antimitotic drugs, infectious disease, metabolic disease) that interferes with anagen, resulting in abnormalities of hair follicles and shafts. Patients suffering from the stress of illness, injury and surgery often require increased amounts of energy, protein, specific amino acids and other nutrients. Patients with severe illness that do not receive adequate nutritional support are at risk for telogen defluxion, anagen defluxion or other coat abnormalities.

Dogs with severe primary seborrhea may have increased protein and other nutrient requirements. The calculated epidermal cell renewal time is approximately seven to eight days for dogs with primary seborrhea (Kwochka and Rademakers, 1990; Baker and Maibach, 1987). The hyperproliferative nature of the skin of dogs with primary seborrhea, with at least a threefold increase in epidermal cell renewal, may change the nutrient requirements of these dogs. However, no studies to date have evaluated the specific nutrient requirements of dogs with severe primary seborrhea vs. age- and breed-matched controls. Some authors suggest that primary seborrhea worsens greatly in dogs with nutritional inadequacies (Scott et al, 1995). Dogs with severe deep pyoderma secondary to generalized demodicosis or other underlying diseases may have increased nutrient requirements above those found in the normal adult animal.

Metabolic Epidermal Necrosis

Metabolic epidermal necrosis is a rare cutaneous disease that in most cases is a marker for a serious underlying metabolic disorder. In dogs, this syndrome has findings similar to those of necrolytic migratory erythema of people and has also been termed hepatocutaneous syndrome or superficial necrolytic dermatitis. Clinical features include crusting acral dermatopathy with erosions around the mouth, eyes, legs, feet and genitalia (Scott et al, 2001; Angarano, 1993). Hyperkeratosis, ulceration of footpads or both conditions is also prominent. The cutaneous syndrome is typically diagnosed in older dogs and is often associated with hepatic cirrhosis, other hepatopathies, diabetes mellitus, hyperadrenocorticism, and rarely, glucagon-secreting pancreatic tumors. Metabolic changes often include carbohydrate intolerance and marked hypoaminoacidemia (Outerbridge et al, 2002).

Specific treatment is aimed at correcting the underlying metabolic disease. Unfortunately, most cases are associated with irreversible chronic liver disease and hepatic cirrhosis (Chapter 68). Symptomatic treatment includes antimicrobials for secondary infections, insulin therapy as needed for diabetes mellitus, hydrotherapy to help remove crusts and lessen pruritus and glucocorticoids. Treatment of hypoaminoacidemia may reverse the skin lesions. Anecdotal reports suggest that foods for repletion/recovery containing moderate protein levels (Prescription Diet a/d Canine/Feline[a]), zinc, egg yolks or intravenous administration of crystalline amino acid solutions will reverse the skin lesions in some patients.[b,c] A review of 36 canine cases showed that dogs responded better to therapy with intravenous amino acid infusions rather than oral protein hyperalimentation (Outerbridge et al, 2002).

ESSENTIAL FATTY ACIDS
Functions in the Skin

EFA are polyunsaturated fatty acids (PUFA) derived from and including the parent EFA, cis-linoleic acid (LA) and α-linolenic acid (ALA). **Figure 32-1** summarizes the metabolic pathway of EFA. The skin of adult mice, guinea pigs and presumably other animals lacks Δ-6-desaturase and Δ-5-desaturase activity (Chapkin et al, 1987). Thus, the epidermis depends on food to supply EFA or the continuous formation of γ-linolenic acid (GLA), arachidonic acid (AA) and eicosapentaenoic acid (EPA) by the liver, with subsequent transportation to the skin by the blood (Chapkin et al, 1987; Horrobin, 1989; Campbell, 1990).

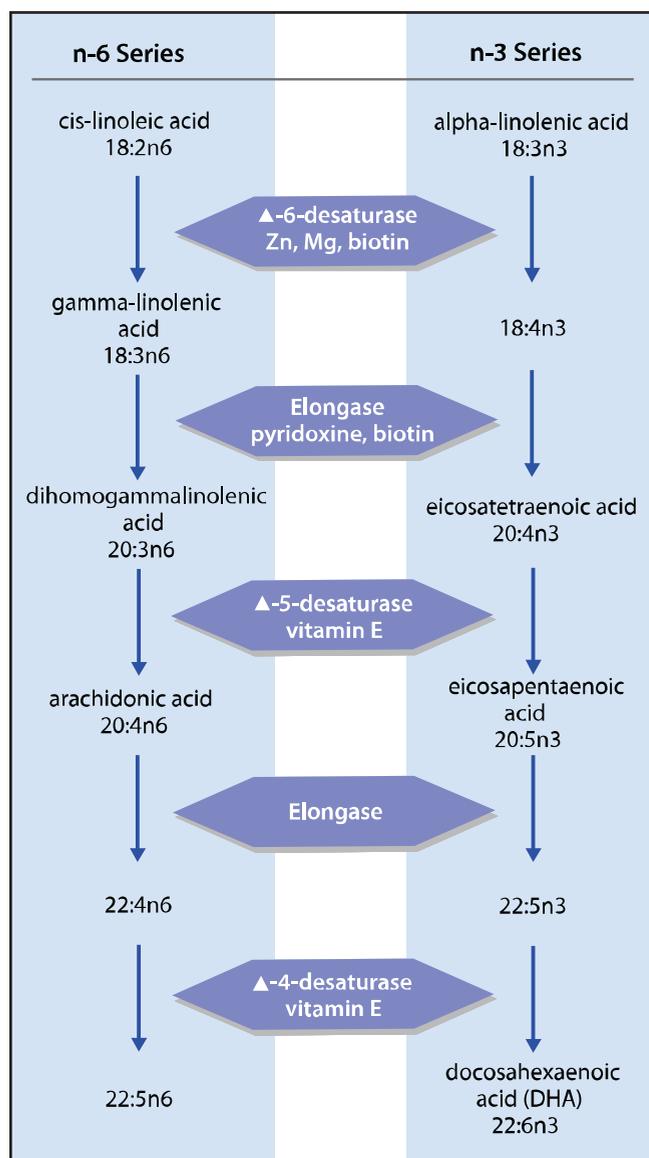In the skin, EFA are principally found in phospholipids, and

Figure 32-1. Diagram of metabolic pathways for essential fatty acids.

so have an accepted structural function in the lipoproteins of cell membranes. The high degree of unsaturation of EFA bestows fluidity to these structures at physiologic temperatures, allowing conformational changes to occur (Prottey, 1976). One of the most important skin-related functions of EFA is the incorporation of LA into the ceramides of the lipid portion of the epidermal cornified envelope. This envelope serves an essential barrier function to prevent loss of water and other nutrients. EFA are a source of energy for the skin and serve as precursors to a variety of potent, short-lived molecules including prostaglandins (PG), leukotrienes (LT) and their metabolites.

Essential Fatty Acid Deficiency

When mammals are deprived of fats, among other things, they develop characteristic signs of EFA deficiency. The exis-

tence of EFA was first recognized when rats deprived of fat had poor weight gain, increased water intake, necrosis of the tail and scaly skin (Burr and Burr, 1929, 1930). The skin scaliness was exacerbated by low ambient humidity or restricted access to water. The increased water intake was later linked to increased transepidermal water loss (Basnayake and Sinclair, 1956). When newly weaned rats were fed foods devoid of EFA, LA and AA levels in the skin rapidly declined (Basnayake and Sinclair, 1956). After five weeks, these acids were virtually absent from the skin, weight loss and increased water intake ensued and scaly skin developed. After 10 weeks, the rate of transepidermal water loss began to increase rapidly to values about 10 times those of normal rats. Growth stunting caused by EFA deficiency is predominantly due to the increase in thermogenesis required to counter heat loss from accelerated transepidermal water evaporation (Phinney et al, 1993).

Cutaneous changes have been described in fatty acid deficiency in dogs (Hansen and Weise, 1951) and cats (Frankel and Rivers, 1978). These cutaneous abnormalities include scaliness (seborrhea sicca), matting of hair, loss of skin elasticity, alopecia, a dry and dull coat, erythroderma, hyperkeratosis, epidermal peeling, interdigital exudation, otitis externa and lack of hair regrowth following plucking. These changes are associated with epidermal and dermal metabolic effects leading to: 1) increased transepidermal water loss, 2) increased epidermal cell turnover, 3) sebaceous gland hypertrophy, 4) increased sebum viscosity, 5) poor wound healing, 6) increased susceptibility to infection and 7) weakening of cutaneous capillaries.

Dogs with cutaneous abnormalities due to low-fat foods have lower levels of fatty acids in serum, skin, liver, kidneys and heart muscle than do animals with healthy skin (Hansen and Weise, 1951). Cats fed an EFA-deficient food developed moderate seborrhea sicca and mild hair loss after six months (MacDonald et al, 1984). Severe seborrhea sicca with large scales developed in EFA-deficient cats when the environmental relative humidity decreased from approximately 75 to 55% (MacDonald et al, 1984). Hair loss is extensive and stroking causes clumps of hair to epilate.

Deficiency of other nutrients, particularly zinc (Ohlen and Scott, 1986), vitamin E (Scott and Sheffy, 1987) and pyridoxine (Cunnane et al, 1984) can cause clinical signs similar to those caused by experimental EFA deficiency. EFA intake influences the requirement of these nutrients. In rodents, clinical signs of zinc deficiency can be largely prevented by EFA supplementation (Cunnane and Horrobin, 1980).

Fatty acid deficiency is rapidly reversible if EFA are introduced orally, parenterally or topically. Various abnormal cutaneous parameters of dogs (Hansen and Weise, 1951) and cats (Frankel and Rivers, 1978; MacDonald et al, 1984) are restored within a few days by LA supplementation.

Supplementation with EFA will increase fatty acid levels in serum of dogs (Campbell et al, 1992; Campbell and Roudebush, 1995) and cats (MacDonald et al, 1984), and in the skin of normal and seborrheic dogs (Campbell et al, 1992; Campbell and Roudebush, 1995; Rees et al, 2001; Marsh et al, 2000). Optimal food levels of EFA for normal dogs and cats

and those with skin and hair disorders are listed in **Table 32-1**.

Use of Fatty Acids for Seborrhea

Fats and fatty acids have been recommended for many years as supplements to improve the sheen and luster of hair. In the past, animal and vegetable sources of fat were recommended to improve coat quality.

Dogs with seborrhea have abnormally low cutaneous levels of LA and increased cutaneous levels of oleic acid (Campbell et al, 1992). These low cutaneous levels are found despite normal food and serum fatty acid concentrations. Following supplementation for 30 days with a vegetable oil high in LA (sunflower oil), the cutaneous fatty acid concentrations return to near normal and clinical signs of seborrhea improve. The clinical signs of seborrhea in dogs may be partly attributable to a localized deficiency of LA, elevated levels of AA in the skin or both (Campbell et al, 1992). However, one study noted no significant differences in the serum and skin fatty acid profiles of normal and a small number of seborrheic dogs (White, 1990).

Seborrhea sicca is also associated with increased transepidermal water loss, which can be reversed with cutaneous administration of vegetable oils rich in LA (Campbell and Kirkwood, 1993). Supplementing food with ALA can also decrease transepidermal water loss (Campbell and Roudebush, 1995). Dietary supplementation with EFA also appears to improve skin and coat condition in normal animals consuming otherwise complete and balanced commercial pet foods (Rees et al, 2001; Marsh et al, 2000).

Antiinflammatory Fatty Acids

See the Fatty Acids for Inflammatory Skin Disease section below.

MINERALS

Minerals in food interact with one another (**Figure 32-2**) and this interaction must be kept in mind when assessing integumentary problems that might be associated with certain homemade foods, commercial foods or nutritional supplements. Skin manifestations of mineral imbalances are seen most commonly with primary (nutritional inadequacy) or secondary (nutrient interaction) deficiencies of copper and zinc.

Copper

Copper is an essential trace element of all plant and animal cells (Brewer, 1987). Copper is involved in various biologic functions, primarily as a component of storage and transport proteins and cuproenzymes. One of the enzymes is lysyl oxidase, which is required for maturation of connective tissue and the cross-linking of aldehydes in collagen and elastin (Brewer, 1987). Copper-containing enzymes also catalyze the conversion of carotene to retinal, the formation of keratin from prekeratin and the biosynthesis of melanin from L-tyrosine.

Cutaneous manifestations of copper deficiency include achromotrichia or loss of normal hair coloration, reduced density or lack of hair and a dull or rough coat. Pigmented hair on the head and face loses its normal color, develops a "washed
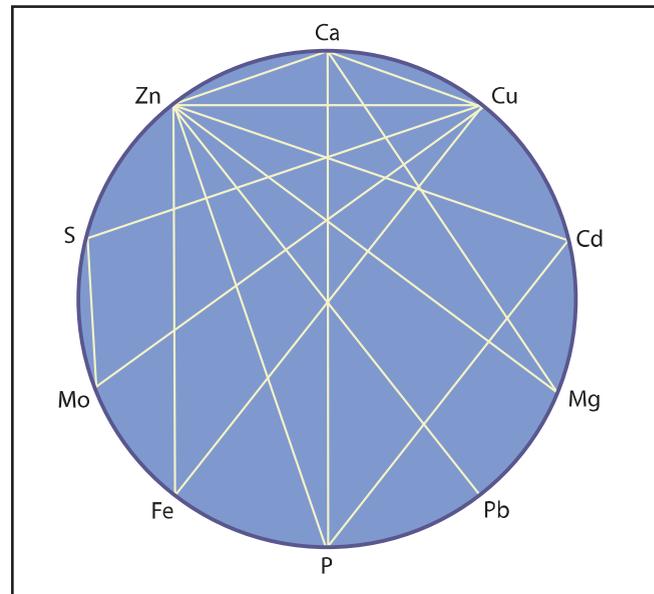

Figure 32-2. Diagram of clinically important mineral interactions in patients with cutaneous disease.

out" appearance and becomes gray (Zentek and Meyer, 1991; Morris and Rogers, 1995; van den Broek and Thoday, 1986). This change may extend over the entire body (Ralston Purina Company, 1987; Morris and Rogers, 1995). In dogs and cats with cutaneous manifestations of copper deficiency, copper concentrations are significantly reduced in plasma, hair, liver, kidney and heart muscle (Zentek and Meyer, 1991; Morris and Rogers, 1995; van den Broek and Thoday, 1986). Copper deficiency is seen most commonly in young puppies and kittens.

Dogs and cats can develop copper deficiency due to: 1) inadequate copper in food, 2) poor availability of copper in food or 3) an excess of competing minerals. Zinc, in particular, can adversely affect copper homeostasis. Zinc is thought to inhibit copper absorption by its action on intestinal metallothioneins, which sequester copper in the intestinal epithelial cells and make copper unavailable for use elsewhere in the body (Fosmire, 1990). The greater the intake of zinc and the lower the intake of copper (absolute or relative), the greater the potential for copper sequestration and ultimately, copper deficiency. Studies involving people and foals have shown increased nutritional copper requirements as the amount of zinc in the food increases (Fosmire, 1990; Bridges and Moffitt, 1990). Optimal levels of copper in foods for dogs and cats with skin and hair disorders are listed in **Table 32-1**.

Copper availability varies widely among feed ingredients (Aoyagi et al, 1993). Copper availability is relatively high in poultry by-product meal, avian liver (chicken and turkey) and ruminant liver (beef and sheep); copper from soybean meal and corn gluten meal is moderately available; copper from monogastric mammalian liver (pork and rat) and copper oxide is poorly available (Morris and Rogers, 1995; Aoyagi et al, 1993; Aoyagi and Baker, 1993; Czarnecki-Mauldin et al, 1993). Risk factors for copper deficiency in dogs and cats include: 1) rapid

Table 32-3. Classification scheme for zinc-related cutaneous disorders in dogs.

Previous classification schemes
Acrodermatitis of bull terriers
Dry juvenile pyoderma
Generic dog food syndrome
Syndrome I (Siberian husky, malamute, other breeds)
Syndrome II (growing dogs)
Proposed classification
Animal abnormalities
  Acrodermatitis of bull terriers
  Zinc malabsorption (Siberian husky, malamute)
Nutritional abnormalities
  Primary zinc deficiency
  Secondary zinc deficiency
  Essential fatty acid deficiency

Table 32-4. Risk factors for zinc-related skin disease in dogs.

Certain breeds
Siberian husky
Malamute
Bull terrier
Great Dane
Labrador retriever
Other rapidly growing large and giant breeds
Food
High mineral levels (calcium, phosphorus, magnesium)
High phytate levels (high levels of cereal ingredients)
Low essential fatty acid levels
Dietary supplements
Calcium and/or other mineral supplements
Cottage cheese or other dairy products
Small intestinal disease
Viral enteritis
Malassimilation (malabsorption, maldigestion)

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growth, 2) unsupplemented homemade foods, 3) commercial or homemade foods supplemented with copper oxide and 4) homemade or commercial foods supplemented with excessive levels of zinc, calcium or iron.

Zinc
Zinc Deficiency

Zinc is an important cofactor of numerous metalloenzymes and modulator of many critical biologic functions. Numerous reports have linked zinc deficiency to many dermatoses in dogs and cats.

Zinc deficiency in animals has been well documented through experiments in numerous species including dogs and cats. Investigators reported decreases in plasma zinc concentrations, a dull and rough coat and skin lesions on the abdomen and hind limbs after feeding a calcium-supplemented, zinc-deficient, corn-soy food to dogs (Robertson and Burns, 1963). Another study documented the progressive development of cutaneous lesions when young puppies were fed a corn-soy, zinc-deficient food (Sanecki et al, 1982). Puppies developed alopecia, skin ulceration, dermatitis, parakeratotic hyperkeratosis, follicular hyperkeratosis and generalized acanthosis similar

to lesions described in other animals with zinc deficiency (Sanecki et al, 1982). These changes were prominent in areas of contact and trauma (footpads), areas of stretch (skin over joints), areas of friction (axillae, groin), distal extremities and tail, mucocutaneous junctions and ear canals. The feet were severely affected with paronychia and fissured, cracked and focally eroded footpads. The cutaneous lesions were completely reversible within six weeks of adding zinc to the food.

Dogs fed a zinc-deficient food developed skin lesions, which improved dramatically within 72 hours of adding zinc to the food (Banta, 1989). A study in kittens fed a zinc-deficient food described poor coats characterized by hair thinning, slow hair growth, scaliness and buccal margin ulcerations (Kane et al, 1981).

Studies in rodents demonstrated the close linkage of zinc and EFA metabolism (Cunnane and Horrobin, 1980; Cunnane, 1982; Huang et al, 1982). Zinc deficiency accelerates development of clinical signs of EFA deficiency; conversely, supplementing with EFA can largely reverse clinical signs of zinc deficiency. Several of the manifestations of zinc deficiency are mediated by a relative state of EFA deficiency attributed, in part, to reduced Δ-6-desaturase enzyme activity (Cunnane and Horrobin, 1980; Cunnane, 1982; Huang et al, 1982). Zinc deficiency may impair the absorption of EFA and vice versa (Huang et al, 1982). Low zinc intake during pregnancy prevents the normal accumulation of long-chain fatty acids and differentially depletes maternal whole-body stores of LA and ALA (Cunnane et al, 1993). This finding suggests that low zinc intake during pregnancy and lactation may be a risk factor for fatty acid deficiency. It is unknown whether similar interactions of zinc and EFA also occur in dogs and cats.

Zinc-Related Dermatoses

A variety of cutaneous diseases in dogs have been described that are thought to be primary or secondary zinc deficiency, or that respond to zinc supplementation. The classification of these skin diseases is confusing and often overlaps (**Table 32-3**). A crusted dermatosis has been reported to occur in young shorthaired dog breeds termed dry juvenile pyoderma or juvenile hyperkeratosis (Baker, 1974; Anderson, 1977). Many cases were not caused by primary bacterial infection and often resolved spontaneously at sexual maturity. In retrospect, these case reports most likely represent the first clinical descriptions of cutaneous disease caused by zinc deficiency in young dogs. A classification scheme was proposed in 1980 for zinc-responsive dermatoses that included two syndromes (Kunkle, 1980). Syndrome I included Siberian husky and malamute dogs, which usually developed lesions in early adulthood and responded to zinc supplementation. Syndrome II included rapidly growing puppies that developed lesions due to zinc deficiency and responded to food change, zinc supplementation or both. Later, a generic dog food syndrome was described in adult dogs and rapidly growing puppies consuming a poor quality food (Sousa et al, 1988). These animals had lesions consistent with zinc deficiency and responded to a food change. Simultaneously, acrodermatitis was described in bull terriers

and linked to abnormal zinc absorption and metabolism (Jezyk et al, 1986; Mundell, 1988).

In all of these syndromes, the dermatoses were clearly associated with zinc deficiency and possibly a deficiency or abnormal metabolism of other nutrients. A more practical classification scheme for zinc-related cutaneous changes in dogs includes clinical syndromes due to nutritional abnormalities (primary or secondary zinc deficiency) or abnormalities of zinc metabolism (**Table 32-3**).

Zinc-Related Dermatoses Associated with Nutritional Abnormalities. Zinc-responsive cutaneous lesions have been frequently described in rapidly growing puppies and less frequently in adult dogs. Many breeds may be affected, but Great Danes, Doberman pinschers, German shepherd dogs, German shorthaired pointers, beagles, standard poodles, Rhodesian ridgebacks and Labrador retrievers are reportedly affected more often (Scott et al, 1995; Ohlen and Scott, 1986; van den Broek and Thoday, 1986; Kunkle, 1980; Sousa et al, 1988; Fadok, 1982; Wright, 1985; Wolf, 1987; Gross et al, 1992; Degryse et al, 1987). Lesions somewhat resemble those of experimental zinc deficiency in puppies and include erythroderma, alopecia and hyperkeratotic plaques (exudative crusts) on the face, head, distal extremities and mucocutaneous junctions (Scott et al, 1995; Gross et al, 1992). Thickened, fissured footpads are also frequently seen. Severely affected animals have systemic signs of lymphadomegaly, poor growth, fever, depression and anorexia. Microscopic examination of skin biopsy specimens shows hyperplastic superficial perivascular dermatitis with diffuse parakeratotic hyperkeratosis.

Risk factors for development of zinc-responsive cutaneous disease are listed in **Table 32-4**. Foods with high mineral levels (calcium, phosphorus, magnesium), poor digestibility, high levels of phytate and/or low levels of total fat and EFA are significant risk factors, especially when fed to puppies during the rapid growth phase. As shown in **Figure 32-2**, other minerals in the food influence zinc absorption. Foods high in calcium, phosphorus and magnesium adversely affect absorption of zinc. Excessive use of mineral supplements containing calcium in large- and giant-breed puppies is common and can inhibit zinc absorption.

Phytin, phytate and phytic acid are different forms of organic phosphorus, presumably inositol hexaphosphate, found in plant proteins. Foods high in cereal ingredients often have excessive levels of phytate that complex with and prevent normal absorption of zinc. Phytate and calcium also interact to affect zinc absorption (Forbes et al, 1984). The relative effect of phytate on zinc absorption increases with the calcium level in the food. Thus, foods high in phytate and calcium have an even greater negative impact on zinc absorption.

Low-cost commercial generic or private label brand dry pet foods are often low in total fat and EFA because fat is an expensive ingredient. Zinc and EFA metabolism interact and foods with marginal concentrations of zinc and EFA may be more likely to cause clinical disease.

Viral enteritis and prolonged diarrhea adversely affect zinc absorption in swine and similar changes may occur in other animals (Whiteneck et al, 1978).

Zinc Deficiency Associated with Metabolic Abnormalities.
Lesions attributed to zinc deficiency develop early in adulthood in Siberian huskies, malamutes and bull terriers and progress at a variable rate (Scott et al, 2001). Skin lesions develop in these breeds despite consumption of well-balanced commercial foods containing adequate levels of zinc. Lesions include erythroderma, alopecia, crusting, scaling and suppuration involving the head, extremities and mucocutaneous junctions. The footpads may become hyperkeratotic. Secondary Malassezia and bacterial infections are common. Some malamute and Siberian husky dogs appear to have a decreased capability for zinc absorption (Brown et al, 1978; Willemse, 1992). Bull terriers that are siblings of those with acrodermatitis may also be affected and probably have abnormal zinc absorption and metabolism (Scott et al, 1995). These patients will probably require zinc supplementation for life to maintain normal tissue zinc concentrations and to avert clinical disease.

Acrodermatitis is an inherited, autosomal recessive metabolic disease reported to occur in bull terriers in the United States, Canada and the United Kingdom (Jezyk et al, 1986; Mundell, 1988; Smits et al, 1991; McEwan, 1993). The syndrome develops shortly after birth and is associated with defects in zinc absorption and metabolism. The condition has been termed lethal acrodermatitis because homozygously affected dogs rarely live beyond 18 months of age.

Cutaneous and systemic clinical signs resemble those of severe zinc deficiency, including growth retardation, gastrointestinal disease, chronic bacterial infections and progressive, erythematous, exfoliative, papular to pustular dermatitis of the distal extremities and skin surrounding the mucocutaneous junctions (Jezyk et al, 1986; Mundell, 1988; Smits et al, 1991; McEwan, 1993). Surface crusts usually contain numerous bacteria and yeast organisms (Smits et al, 1991). Owners complain that their dogs have difficulty eating and are affected by skin disease, poor growth and large, splayed, painful feet (McEwan, 1993). Ulcerated and thick, crusted lesions are prominent on the muzzle and ears. Abnormal keratinization of the footpads, severe nail dystrophy and paronychia are also common. Histopathologic examination of skin reveals massive parakeratotic hyperkeratosis.

A study showed that two affected dogs had significantly lower plasma zinc concentrations, lower zinc and copper concentrations in the kidneys and liver and lower zinc absorption when compared with age-matched control dogs (Mundell, 1988). Serum zinc concentrations may also be normal (Smits et al, 1991; McEwan, 1993). Supplementation with oral or intravenous zinc fails to ameliorate clinical signs. Treatment with systemic antimicrobials, especially for secondary superficial yeast infections, may result in marked improvement, although systemic and cutaneous infections recur. Some apparently normal littermates may develop a zinc-responsive dermatitis (Scott et al, 2001).

Diagnosis and Management of Zinc-Responsive Skin Disease

Diagnosis of zinc-responsive cutaneous disease is based on the history, physical examination and results of skin biopsy evaluation. Hyperplastic superficial perivascular dermatitis with marked diffuse and follicular parakeratotic hyperkeratosis is suggestive of zinc deficiency (Gross et al, 1992a). In general, zinc concentrations in serum, leukocytes and hair are not good indicators of zinc status in dogs (van den Broek and Thoday, 1986; Wolf, 1987; van den Broek et al, 1988; Logas et al, 1993). Age, seasonal variation and many diseases affect serum zinc concentrations (Fisher, 1977; Keene et al, 1981). One study found no significant difference in serum zinc concentrations between normal dogs, dogs that were ill without skin disease, dogs with allergic skin disease and dogs with other dermatoses (Logas et al, 1993).

Treatment generally includes changing to a food that avoids excess minerals and contains adequate amounts of zinc and EFA. Optimal levels of zinc and EFA in foods for dogs and cats with skin and hair disorders are listed in **Table 32-1**. This type of change will usually result in rapid improvement in puppies and some adult dogs. Zinc supplementation will be necessary in those breeds in which decreased ability to absorb zinc is suspected. Oral supplementation with zinc sulfate (10 mg/kg body weight/day) or zinc methionine (2 mg/kg body weight/day) is adequate in most cases. Zinc absorption is maximal if supplements are given between, rather than with, meals. Supplemental zinc from zinc amino acid chelates may be more available to dogs than are inorganic zinc sources (Lowe et al, 1994). Some dogs, especially Siberian huskies, do not respond to oral zinc supplementation. Intravenous injection of sterile zinc sulfate solutions at dosages of 10 to 15 mg/kg body weight has been effective in these dogs (Willemse, 1992). Weekly injections for at least four weeks are necessary to resolve the lesions, and maintenance injections every one to six months may be necessary to prevent relapses.

Existing skin lesions can be improved by hydrating the crusts with wet dressings, applying petrolatum or petrolatum-based topical agents or whole-body warm water soakings. Dogs with evidence of superficial pyoderma or *Malassezia* infections should be treated with appropriate antimicrobials. Some authors also recommend low doses of oral, short-acting glucocorticoids (Kwochka, 1993).

VITAMINS
Vitamin A

Retinol, retinal and retinoic acid are three natural compounds that have vitamin A activity in mammals. Food sources include retinyl esters (vitamin A palmitate) in animal tissues and carotenoids (β-carotene) in vegetables. These sources are assimilated and ultimately stored as retinyl palmitate in the liver. Cats require preformed vitamin A because they lack the ability to effectively convert β-carotene to vitamin A (NRC, 2006).

The general functions of vitamin A include growth promotion, differentiation and maintenance of epithelial tissue and maintenance of normal reproductive and visual functions. Retinoic acid affects differentiation and proliferation of epithelial cells by binding to and activating a specific set of cell nuclear receptors (Wolf, 1990; Blumenberg et al, 1992). In particular, epithelial cells have a specific nuclear receptor for retinoic acid (Blumenberg et al, 1992). The mechanism of action of retinoic acid is similar to that of steroid hormones and thyroxine, and involves activation of specific genes. Retinoic acid and thyroid hormone control overlapping gene networks, regulating growth and differentiation through nuclear receptors that can modify rates of gene transcription.

Retinoic acid influences epidermal differentiation and directly affects keratinization by action of retinoic acid receptors on regulatory sites in keratin genes. Retinoic acid may also influence hair growth through activity at the hair bulb.

Vitamin A deficiency in dogs was among the first of the vitamin deficiencies to be studied experimentally (NRC, 2006). Skin lesions and focal atrophy of the skin have been reported with experimental vitamin A deficiency in dogs and cats, although it is seldom encountered clinically (NRC, 1986, 1985). Some of the earliest work with vitamin A showed that puppies had heavier, more lustrous coats when foods were supplemented with vitamin A (Bradfield and Smith, 1938). It is unlikely that vitamin A deficiency would occur in dogs and cats eating typical commercial pet foods because these foods contain several times the minimum daily requirement of vitamin A (Kallfelz and Dzanis, 1989). The minimum recommended allowance for vitamin A in foods for dogs is 5,050 IU/kg of food (DM) (growth/reproduction and maintenance requirements are the same) and 3,333 and 6,666 IU/kg of food (DM) for growth/maintenance and gestation/lactation for cats, respectively (NRC, 2006).

Retinoid-Responsive Dermatoses

The term "retinoids" refers to the entire group of naturally occurring and synthetic vitamin A derivatives. These therapeutic agents should be reserved for cases in which there are clinical and histopathologic abnormalities most consistent with primary keratinization disorders of the surface and/or follicular epithelium or abnormalities of the sebaceous glands (Power and Ihrke, 1990; Kwochka, 1993a). Other causes of clinical scaling (ectoparasitism, allergies, infections, endocrinopathies) should first be eliminated through other diagnostic testing.

A vitamin A-responsive dermatosis has been described primarily in cocker spaniels but it has also been recognized in a Labrador retriever and a miniature schnauzer (Scott et al, 2001; Kwochka, 1993a). The condition is characterized by adult-onset, medically refractory seborrheic skin disease with marked follicular plugging and hyperkeratotic plaques, primarily on the ventral and lateral thorax and abdomen (Scott et al, 2001; Kwochka, 1993a). A ceruminous otitis externa and unthrifty appearing coat are often present. The clinical lesions are characterized histologically by marked follicular orthokeratotic hyperkeratosis. Improvement is noted within three to four weeks of starting oral vitamin A alcohol (retinol) with complete remission by eight to 10 weeks (Scott et al, 2001; Kwochka,

1993a). It is important to remember that this syndrome represents only a small subset of seborrheic disease in cocker spaniels. However, it is logical to try a four- to eight-week course of vitamin A in dogs with ventral hyperkeratotic plaques that do not respond well to other therapy (Kwochka, 1993a).

Many synthetic retinoids have been developed to offer better therapeutic response and less toxicity than naturally occurring vitamin A compounds. The most commonly used synthetic retinoids include tretinoin, isotretinoin and etretinate.

Tretinoin is effective topically as therapy for localized follicular and epidermal keratinization disorders such as chin acne, nasodigital hyperkeratosis, calluses and ear margin seborrhea/dermatosis (Scott et al, 2001; Power et al, 1990; Kwochka, 1993a). Isotretinoin and etretinate are given orally, in combination with food, and may be useful to manage primary idiopathic seborrhea in cocker spaniels, keratinization disorders in other breeds, schnauzer comedo syndrome, sebaceous adenitis, lamellar ichthyosis, actinic keratosis (solar-induced precancerous lesions) and various cutaneous neoplastic disorders (squamous cell carcinoma, cutaneous T-cell lymphoma, multiple keratocanthomas). Etretinate is no longer available but has been replaced by acitretin, a metabolite of etretinate (Scott et al, 2001).

Retinoid dosages commonly recommended by veterinary dermatologists are outlined in **Table 32-5**. Side effects that occur commonly with retinoids include conjunctivitis, decreased tear production, vomiting, diarrhea, arthralgia/myalgia, moderate to marked elevations in serum triglyceride levels, elevations in liver enzyme activity and teratogenic effects.

Vitamin E

Eight isomeric forms of tocopherol represent vitamin E activity, with α-tocopherol being most important biologically. Vitamin E quenches free radicals in PUFA of membrane phospholipids. The nutritional requirement of vitamin E is closely related to the dietary intake of PUFA.

Naturally occurring vitamin E deficiency has only been reported to occur in cats. Steatitis occurs when sources of highly unsaturated fatty acids, such as red meat tuna, are fed to cats without adequate vitamin E. Clinical signs and laboratory findings include anorexia, fever, hyperesthesia, hemolytic anemia, leukocytosis and firm subcutaneous nodules. Diagnosis is confirmed by microscopic examination of biopsy specimens from adipose tissue. Typical lesions are firm, yellow to orange-brown fat with lobular panniculitis and ceroid within lipocytes, macrophages and giant cells. Treatment includes a change of food to a complete and balanced ration, supplemental vitamin E (25 to 75 mg/kg body weight/day), corticosteroids and supportive care.

Naturally occurring vitamin E deficiency has not been reported to occur in dogs, but experimentally induced vitamin E deficiency does produce skin lesions (Scott and Sheffy, 1987). Initial lesions consist of a keratinization defect (seborrhea sicca), followed by a greasy and inflammatory stage (erythroderma and seborrhea oleosa) and secondary bacterial pyoderma. The dermatosis rapidly responds to vitamin E supplemen-

tation. All lesions respond within eight to 10 weeks. It is unlikely that vitamin E deficiency would occur in dogs and cats that eat typical commercial pet foods because such foods contain three to five times the minimum daily requirement of vitamin E (Kallfelz and Dzanis, 1989).

Because of its role as a barrier, the skin is uniquely challenged by oxidants (i.e., free radicals). The skin is continuously exposed to an oxidative environment, including high oxygen tensions, air pollutants, ultraviolet (UV) radiation, parasites, aerobic microorganisms and oxidants released as a result of normal metabolism. UV radiation causes tissues to produce reactive oxygen species, eicosanoids and cytokines, which can result in acute adverse effects (e.g., sunburn, photosensitivity) as well as long-term sequelae (e.g., actinic keratosis, solar dermatitis, malignant skin tumors) (Scott et al, 2001; Nikula et al, 1992; Kimura and Doi, 1994). Because of the high lipid content of skin, lipophilic antioxidants such as α-tocopherol are expected to play a major role in scavenging reactive oxygen species during oxidative stress (Thiele et al, 2001). Vitamin E protects against UV-induced skin damage through a combination of antioxidant and UV-absorptive properties (Thiele et al, 2001). One study revealed that increasing vitamin E amounts in food significantly increased vitamin E concentrations in serum and skin of cats and dogs (Jewell et al, 2002). Previous studies have shown that increased vitamin E levels decrease serum levels of some biomarkers associated with oxidative stress (Jewell et al, 2000). This suggests that increases in dietary vitamin E concentrations are likely to be beneficial. However, the relationship between increases in serum and skin vitamin E concentrations and the prevention, development and treatment of specific skin diseases remains to be elucidated by intervention studies. The recom-

Table 32-5. Indications and dosages for retinoids in primary keratinization disorders.

Vitamin A alcohol (retinol)
Subset of seborrheic skin disease, primarily in cocker spaniels
Dosage: 625 to 1,000 IU/kg q24h per os
    10,000 IU q24h per os in cocker spaniels and miniature
    schnauzers
    50,000 IU q24h per os in Labrador retrievers
Tretinoin (all-trans retinoic acid)
Chin acne of dogs and cats
Nasodigital hyperkeratosis
Ear margin seborrhea/dermatosis
Dosage: Apply topically q12 to 24h to control; then decrease
    frequency for maintenance
Isotretinoin (13-cis retinoic acid)
Lamellar ichthyosis
Schnauzer comedo syndrome
Sebaceous adenitis
Dosage: 1 to 3 mg/kg q24h per os with food for control; then try
    to decrease to alternate-day therapy
Acitretin (analogue of retinoic acid ethyl ester)
Actinic keratosis
Idiopathic seborrhea, especially of cocker spaniels
Lamellar ichthyosis
Sebaceous adenitis
Dosage: 0.75 to 1.0 mg/kg q24h per os for control; then try to
    decrease to alternate-day therapy

mendation for vitamin E in foods is at least 400 IU/kg of food (DM) for dogs and at least 500 IU/kg of food (DM) for cats (Chapters 13 and 20). The minimum recommended allowances for foods for adult dogs and cats for maintenance is 30 IU/kg (DM) and 38 IU/kg (DM), respectively (NRC, 2006).

Vitamin E-Responsive Dermatoses

A number of inflammatory dermatoses in animals have been treated with oral vitamin E, including discoid lupus erythematosus, systemic lupus erythematosus, pemphigus erythematosus, sterile panniculitis, acanthosis nigricans, dermatomyositis and ear margin vasculitis. Vitamin E is often used in conjunction with systemic glucocorticoids, topical steroids and other immunosuppressive agents. Large doses of vitamin E may stabilize cell and lysomal membranes against damage induced by free radicals and peroxides, modulate AA and PG metabolism, inhibit proteolytic enzymes, enhance phagocytic activity and enhance humoral and cellular immunity.

Vitamin E appears to have limited value as an antipruritic agent. One uncontrolled study in allergic dogs that received vitamin E failed to document a reduction in pruritus (Miller, 1989). A well-controlled study in adult people with atopic dermatitis also failed to demonstrate improvement with vitamin E or selenium supplementation (Farris et al, 1989).

The oral dosage of vitamin E for inflammatory dermatoses is 200 to 800 IU twice daily (Scott et al, 2001; Rosenkrantz, 1993). This dose is seven to 27 times higher than the lower end of the recommended daily amount (≥400 IU/kg food [DM]) for a 10-kg dog (Chapters 13 and 14). Anecdotal reports suggest that topical vitamin E may help resolve discoid lupus erythematosus lesions (Rosenkrantz, 1993). Vitamin E is one of the least toxic vitamins.

B-Complex Vitamins

Experimental deficiencies of biotin and riboflavin can cause cutaneous lesions in dogs and cats (NRC, 2006). The most common clinical signs include anorexia, weight loss, diarrhea, alopecia and dry, flaky seborrhea. Clinical lesions are more likely to occur in young, growing animals than in adults (Pastoor, et al, 1991).

Several B-complex vitamins act as cofactors in EFA metabolism. LA desaturation and GLA elongation may be impaired in pyridoxine deficiency (Cunnane et al, 1984). EFA may have a sparing effect on the cutaneous lesions caused by B-complex vitamin deficiency and vice versa (Cunnane et al, 1984).

It is unlikely that B-complex vitamin deficiency would occur in dogs and cats that eat commercial pet foods, because most foods contain several times the minimum daily requirement of these vitamins. However, supplementation of complete and balanced pet foods with biotin, pantothenic acid, inositol, choline, other B vitamins, zinc and fatty acids has been shown to alter skin function (reduce transepidermal water loss) and improve coat softness and appearance (Markwell et al, 2004; Watson and Marsh, 2001). Thus, for some patients with skin/coat problems, feeding foods with additional amounts of these nutrients may be beneficial. B-complex deficiency could occur in animals eating homemade foods inadequately supplemented with vitamins.

Feeding Plan
Assess and Select the Food

In general, a patient's food should be changed if one of the following skin or coat conditions develops:
- Loss of normal hair color, especially lightening, graying or reddish-brown discoloration of normally pigmented hair,
- Brittle and easily broken hair,
- Generalized scaling, crusting, alopecia or loss of normal hair sheen for which no underlying skin disorder can be identified through routine diagnostic evaluation,
- Poor wound healing or decubital ulcers,
- Severe, generalized inflammatory skin disease such as deep pyoderma or immune-mediated skin disease,
- Hyperproliferative skin disorder such as primary seborrhea,
- Abnormal hair growth or failure of hair to regrow where clipped or lost.

Table 32-1 summarizes the key nutritional factors for foods for patients with nutrient responsive-dermatoses. A food for patients with skin and hair problems should include the recommended levels of these nutrients, and the nutrients should be available to the patient. Digestibility and assimilation of nutrients are especially important during periods of increased nutrient demand such as growth, gestation and lactation. Use of maintenance-type foods (which are usually lower in protein, fat, minerals, vitamins and digestibility than growth/lactation or foods for repletion/recovery) may be a risk factor for nutritional skin disease during these lifestages. Levels of these nutrients can be found in food tables for normal dogs and cats (Chapters 13 and 20). Otherwise, they can be obtained by contacting the manufacturer or distributor of the food.

A detailed assessment of nutritional supplements is also important. Vitamin supplements are rarely indicated except in those nutrient-sensitive disorders that respond to high levels of vitamins A or E. Excessive use of mineral supplements can interfere with assimilation of zinc and copper. Besides recommended key nutritional factors for foods for patients with nutrient-deficiency dermatologic conditions, Table 32-1 also provides information about nutritional supplements for patients with skin and hair disorders. When appropriate, nutritional supplements can be used in conjunction with the food change or can be added to the original food. Changing to a food appropriate for the patient's lifestage will usually reverse cutaneous signs associated with a relative nutrient deficiency. Select a food whose nutritional adequacy was determined by animal tests (Chapter 9). This helps ensure that the nutrients in the food are available to the animal. Foods formulated to meet the nutrient profiles set forth by the Association of American Feed Control Officials might be nutritionally adequate but this form of establishing nutritional adequacy cannot ensure nutrient availability. Supplementation alone will not usually improve a poor-quality food. Supplementation with fatty acids, zinc, retinoids and vitamin E usually exceeds levels used to meet nutrient requirements. In these cases, nutrient supplements are being used as therapeutic agents.

Another criterion for selecting a food that may become increasingly important in the future is evidence-based clinical nutrition. Practitioners should know how to determine risks and benefits of nutritional regimens and counsel pet owners accordingly. Currently, veterinary medical education and continuing education are not always based on rigorous assessment of evidence for or against particular management options. Still, studies have been published to establish the nutritional benefits of certain pet foods. Chapter 2 describes evidence-based clinical nutrition in detail and applies its concepts to various veterinary therapeutic foods.

Assess and Determine the Feeding Method

The method of feeding is often not altered in the nutritional management of skin and hair disease. If a new food is fed, the amount to feed can be determined from the product label or other supporting materials. The food dosage may need to be changed if the caloric density of the new food differs from that of the previous food. The food dosage is usually divided into two or more meals per day. The food dosage and feeding method should be altered if the patient's body weight and condition are not optimal.

For clinical nutrition to be effective, there needs to be good compliance. Enabling compliance includes limiting access to other foods and knowing who feeds the animal. If the patient comes from a household with multiple pets, it should be determined whether the pet with skin disease has access to the other pets' food.

Reassessment

Cutaneous disease due to a nutrient deficiency will usually respond rapidly and dramatically to appropriate nutritional change or supplementation. Patients will usually improve within a few days to weeks. Nutrient-sensitive disorders usually respond to supplements more slowly, over several weeks to several months. After a nutritional change or supplementation has been started, the patient should be examined monthly for significant changes in skin lesions and hair quality. Trichograms can be repeated in those patients that have abnormal hair quality or hair growth.

FATTY ACIDS FOR INFLAMMATORY SKIN DISEASE

Clinical Importance

EFA exhibit multiple antiinflammatory and immunomodulating properties. They have the potential to affect allergic and other forms of skin inflammation through modulating cytokine production, inhibiting cellular activation and cytokine secretion and altering the composition and function of the epidermal lipid barrier (Olivry et al, 2001). Their mechanisms of action, therefore, are likely to be explained by a combination of effects.

The most commonly proposed mechanism of action of EFA in the treatment of inflammatory skin diseases is the modulation of cutaneous production of PG and LT (Olivry et al,

2001). AA is the major PUFA in cell membrane phospholipids (Stossel et al, 1974). The normal response of injured tissue is inflammation, a tissue protective mechanism. Under these circumstances, phospholipases are activated and act on phospholipids of cell membranes to release constituent fatty acids. AA, the fatty acid in greatest concentration, is released and converted into eicosanoids, which mediate inflammation.

Eicosanoids are also derived from GLA and EPA. They also include PG, thromboxanes, LT, hydroperoxyeicosatetraenoic acids (HPETE) and hydroxyeicosatetraenoic acids (HETE). Macrophages are the most significant sources of eicosanoids (Lokesh et al, 1988; Meydani et al, 1991; Hwang, 1989; Magrum and Johnston, 1983).

Four AA-derived LT and one PG play a central role in the inflammatory process. LTB_4 stimulates neutrophil and eosinophil chemotaxis and increases vascular permeability. LTC_4, LTD_4 and LTE_4 encourage smooth muscle contraction and increase vascular permeability. PGE_2 inhibits T and B lymphocyte proliferation, reduces cytokine production and limits natural killer cell activity. However, these proinflammatory eicosanoids can result in pathologic conditions when produced in excessive amounts and/or prolonged periods of time (Sigal, 1991; Lands, 1989). Increased production of LT and PGE_2 has been reported in many chronic inflammatory diseases (Goodwin and Ceuppens, 1983).

Modulating the PUFA content of cell membrane phospholipids by dietary means can alter eicosanoid production. Such modulation can affect the intensity and duration of inflammatory and immune responses (Lokesh et al, 1988; Meydani et al, 1991; Hwang, 1989; Magrum and Johnston, 1983). Generally, omega-3 fatty acids are thought to produce less inflammatory cytokines (Sigal, 1991; Lands, 1989; Lokesh et al, 1988; Lokesh and Kinsella, 1987; Broughton et al, 1991; Croft et al, 1987).

Consumption of flax or fish oils with omega-3 PUFA results in replacement of AA in the macrophage membrane with ALA, docosahexaenoic acid (DHA) or EPA. The result is production of fewer AA- (omega-6) derived eicosanoids and more ALA- (omega-3) derived eicosanoids, thereby reducing the inflammatory response (Meydani et al, 1991; Calder et al, 1990; Endres et al, 1989, 1993; Baldie et al, 1993; Lee et al, 1985). Studies using neutrophils from normal dogs have shown that enhanced levels of dietary omega-3 fatty acids inhibit leukotriene generation (Byrne et al, 2000; Vaughn et al, 1994). Studies have also shown alterations in inflammatory response and immune function in normal cats fed foods with enhanced levels of omega-3 fatty acids (Chew et al, 2000). Consequently, changing the type of eicosanoid production and the subsequent alteration in cytokine production can reduce inflammation by eicosanoid-mediated effects (Horrobin and Manku, 1990; Calder et al, 1990; Endres et al, 1989, 1993; Baldie et al, 1993; Lee et al, 1985; Watson et al, 1990). This premise is the basis for using omega-3 fatty acids for treatment of chronic inflammatory conditions.

A similar effect is proposed for the use of GLA, an omega-6 derivative of LA. GLA and DHA reduce histamine release and

alter cell mediator production in canine mast cells (Gueck et al, 2004). If similar changes occur in dermal mast cells from patients with atopic dermatitis, these results suggest that GLA and omega-3 fatty acid supplements or similarly enriched foods might be beneficial. Black currant oil, borage oil and evening primrose oil are sources of GLA (Meydani et al, 1991; Calder et al, 1990; Endres et al, 1989, 1993; Baldie et al, 1993; Lee et al, 1985).

Besides affecting LT and PG production, EFA exhibit numerous additional immunomodulating properties. They have been reported to decrease the synthesis of proinflammatory cytokines, decrease T-cell lymphocyte proliferation and activation, affect expression of cell adhesion molecules, influence signaling within cells of the immune system and regulate cytotoxic activity of phagocytes by modulating the production of reactive oxygen species (Olivry et al, 2001a). Preliminary investigations suggest that dogs with atopic dermatitis also may exhibit abnormal epidermal lipid levels and metabolism (Olivry and Hill, 2001a). EFA have the potential to modulate this abnormal skin lipid barrier function in animals with inflammatory skin disease.

For more information about fatty acid metabolism and fatty acid modulation of the inflammatory response see the Lipids section in Chapter 5.

Patient Assessment
History and Physical Examination
Numerous skin diseases have an inflammatory component. However, dietary fatty acid therapy has been used primarily in patients with allergic skin disease or patients with pruritus or papulocrustous dermatitis for which a specific cause has not been identified.

Pruritus is the most common historical feature of allergic skin disease in dogs and cats. Clinical signs reportedly first occur in most dogs and cats with atopic dermatitis between six months and three years of age (Scott et al, 2001; Griffin et al, 1993; Griffin and DeBoer, 2001). Lesions of canine atopic dermatitis usually involve the muzzle, periocular region, pinnae and external ear canals, paws, axillae, groin and abdomen. Although the face and paws are most commonly involved, many animals will have generalized pruritus by the time they are examined. Chronic licking, rubbing, chewing or scratching can result in alopecia, lichenification, hyperpigmentation, scaling and excoriation. Other common lesions in atopic dogs include papules and erythematous macules, secondary superficial pyoderma, secondary *Malassezia* dermatitis, chronic otitis externa and seborrhea.

Cats with atopic dermatitis most commonly exhibit symmetric alopecia, miliary dermatitis, eosinophilic plaques, indolent ulcer of the lip, pruritus of the head and neck with excoriations or generalized pruritus (Scott et al, 2001; Sousa, 1995). Atopic cats are pruritic, but many are secretive and groom or traumatize themselves without the owner's knowledge.

Cats with miliary dermatitis have numerous small erythematous papules with adherent brownish crusts and various degrees of alopecia and pruritus (Scott et al, 2001; Sousa, 1995). These lesions can usually be palpated over the dorsal lumbar and cervical regions long before they are visualized. Feline miliary dermatitis is most commonly a manifestation of flea allergy, but may occur with other ectoparasite infestations, dermatophytosis, bacterial folliculitis, adverse food reactions, atopic dermatitis, drug eruptions and immune-mediated skin disease.

Canine flea-bite hypersensitivity is characterized by a pruritic, papular dermatitis (Scott et al, 2001). Flea bites induce an initial papule that may then form a crust. Chronic pruritus may lead to alopecia, lichenification, severe crusting and hyperpigmentation. Lesions are typically confined to the dorsal lumbosacral area, caudomedial thighs, ventral abdomen and flanks. Pyotraumatic dermatitis ("hot spots"), secondary bacterial pyoderma and secondary seborrhea are common in chronic cases. The presence of otitis externa, severe pedal pruritus or facial pruritus strongly suggests concurrent atopic dermatitis or adverse food reaction.

Numerous insects besides fleas and arachnids in the normal dog and cat environment can stimulate hypersensitivity reactions. Blackfly, deerfly, horsefly, mosquito, red ant, black ant and tick bites may all contribute to allergic skin disease in dogs and cats (Griffin et al, 1993). The primary clinical sign is pruritus, although an erythematous maculopapular dermatitis may be present (Scott et al, 2001). Nodules and papules induced by mosquito bites are usually found on the bridge of the nose and pinnae of cats. Stable flies occasionally induce a granulomatous reaction, producing nodules or plaques and varying degrees of alopecia on the pinnae. Ticks may induce nodules due to granuloma formation at the site of attachment. Acute-onset nasal dermatitis has also been observed in dogs; pruritic papules and nodules are found on the bridge of the nose.

Adverse reactions to food mimic other allergic diseases. The clinical features and management of adverse food reactions are described in detail in Chapter 31.

Laboratory and Other Clinical Information
Skin biopsy and histopathology can be used to confirm the presence of inflammatory skin disease. Chronic hyperplastic dermatitis is a common histopathologic reaction pattern seen in dogs with chronic allergy (Gross et al, 1992; Olivry and Hill, 2001b). The predominant types of inflammatory cells may suggest the specific allergic disease. However, many chronic dermatoses have similar histopathologic features, making specific diagnosis difficult. The nature of epidermal and dermal inflammatory cell infiltrates in canine atopic dermatitis has recently been characterized using modern immunologic techniques and is described in other sources (Olivry and Hill, 2001b).

Two methods of allergy testing are available to practitioners. Intradermal testing has been performed for many years. More recently, in vitro tests for detection of allergen-specific IgE have become commercially available.

Intradermal testing is widely used by veterinary dermatologists for making a definitive diagnosis of canine atopic disease and for selecting allergens for hyposensitization (Scott et al, 2001; Hillier and DeBoer, 2001). Intradermal allergy tests detect the allergen-specific IgE fixed to the surface of mast cells

in the dermis, and assess the ability of IgE to fix allergen and cause mast cell degranulation and subsequent vasodilatation. In a well-controlled study using allergen mixes, 59% of dogs responded to hyposensitization that was formulated on the basis of intradermal testing results (Willemse et al, 1984).

Intradermal allergy testing has several disadvantages (Hillier and DeBoer, 2001). Negative intradermal results occur in some dogs strongly suspected to have atopic dermatitis. Anti-inflammatory and antihistamine drugs must be withdrawn before testing to prevent false-negative results. The test cannot be performed on dogs with generalized dermatitis. Shaving of the coat and sedation are usually required. Intradermal allergy testing is time-consuming and not cost-effective when performed infrequently. The usefulness of intradermal allergy testing is also limited by lack of standardized allergy extracts and no homogeneous criteria for the interpretation of results. Most intradermal testing is performed at dermatologic referral centers because of these disadvantages. Intradermal testing for food hypersensitivity is unreliable in animals with dermatologic disease (Chapter 31).

In vitro "allergy" tests measure serum concentrations of allergen-specific IgE and avoid many of the disadvantages of intradermal allergy tests (Codner and Griffin, 1996). In vitro tests require only a serum sample; so they are readily available to private practitioners and can be used on patients with generalized dermatitis. Laboratories use several different techniques to detect circulating IgE levels, including a radioallergosorbent test (RAST), enzyme-linked immunosorbent assay (ELISA) or liquid-phase enzyme immunoassay (EIA). Problems with in vitro testing include poor reproducibility and a high false-positive rate (Codner and Lessard, 1993). Results to date suggest that more than 60% of atopic dogs respond to hyposensitization formulated on the basis of in vitro results (Anderson, 1993; Sousa and Norton, 1990). In vitro testing is also available for confirming flea-allergic dermatitis (Cook et al, 1996) but is unreliable for diagnosing food hypersensitivity (Chapter 31).

Controversy continues over whether intradermal or in vitro testing is the better method for confirming a diagnosis of atopic dermatitis and for selecting allergens for hyposensitization (DeBoer and Hillier, 2001). Furthermore, long-term studies are needed to evaluate responses of allergic animals to hyposensitization based on both types of testing.

Risk Factors

Atopy (atopic state) is a genetically-predisposed tendency to develop IgE-mediated allergy to environmental allergens (Olivry et al, 2001c). Atopic disease is any clinical manifestation of atopy including most commonly atopic dermatitis, atopic conjunctivitis and/or atopic rhinitis (Olivry et al, 2001c). Atopic dermatitis is a genetically predisposed inflammatory and pruritic allergic skin disease with characteristic clinical features (Olivry et al, 2001c). It is associated most commonly with IgE antibodies to environmental allergens. Although the exact mode of inheritance is unknown, strong breed predilection and familial involvement in dogs indicate a genetically determined cause. Canine breeds reported to be predisposed to atopy in-

Table 32-6. Key nutritional factors for foods and supplements for dogs and cats with inflammatory dermatoses.

Factors	Nutritional recommendations
Omega-3 fatty acids (ALA, EPA and/or DHA)	Supplements or foods should initially provide 50 to 300 mg total omega-3 fatty acids/kg body weight/day Foods should contain between 0.35 to 1.8% dry matter

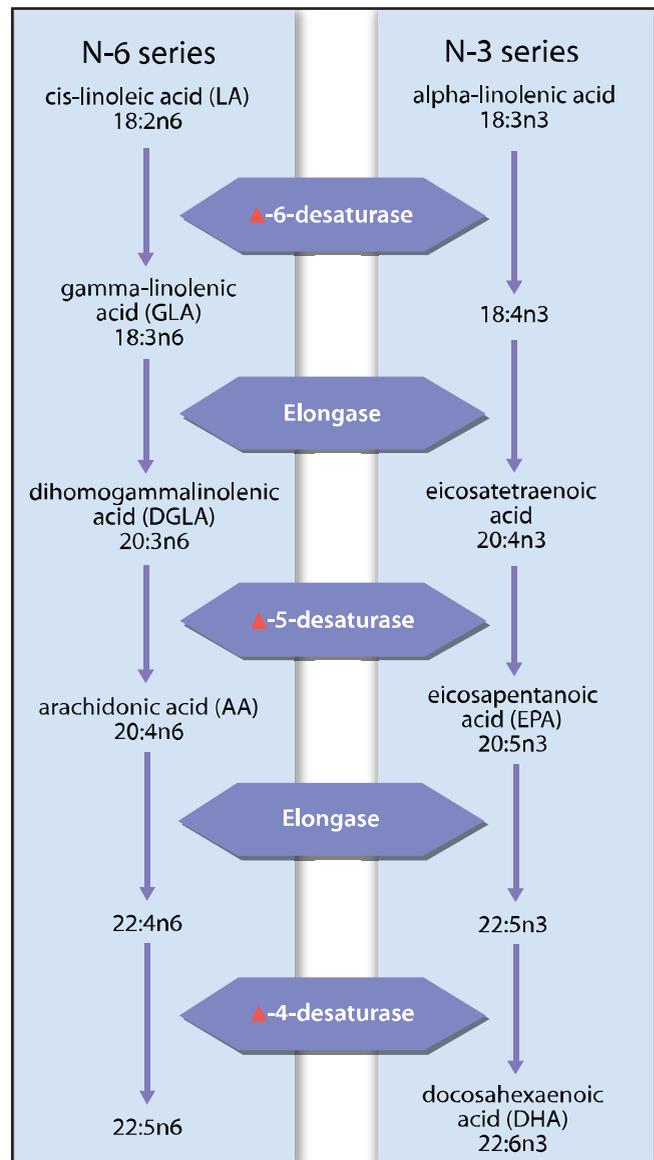Key: ALA = α-linolenic acid, EPA = eicosapentaenoic acid, DHA = docosahexaenoic acid.

Figure 32-3. Metabolic transformation of two major unsaturated fatty acid families by desaturation and elongation.

clude Cairn terriers, West Highland white terriers, Scottish terriers, wire-haired fox terriers, Boston terriers, Sealyham terriers, Lhasa apsos, Dalmatians, pugs, Irish setters, English setters,

Table 32-7. Fatty acids found in pet food ingredients and supplements.

Fatty acids	Ingredients/supplements
Linoleic acid (omega-6)	Vegetable oils (soy oil, corn oil, safflower oil, canola oil, etc.) Grains (corn, soybeans)
γ-linolenic acid (GLA, omega-6)	Black currant oil Borage oil Evening primrose oil
α-linolenic acid (ALA, omega-3)	Flax Flax (linseed) oil
Eicosapentaenoic acid (EPA, omega-3)	Fish meal Cold water marine oils
Docosahexaenoic acid (DHA, omega-3)	Fish meal Cold water marine oils

golden retrievers, Labrador retrievers, boxers, miniature schnauzers, English bulldogs, Bichon Frise, Chinese Shar-Pei, Shih Tzu, German shepherd dogs, Belgian Tervuren, beauceron and cocker spaniels (Scott et al, 2001; Griffin et al, 1993; Anderson, 1993). However, canine atopic dermatitis may be seen in any breed, including mixed breeds. Breed predisposition has not been reported for atopic cats (Scott et al, 2001).

Hypersensitivity requires environmental exposure to flea, other biting insect or arachnid allergens. Depending on the offending allergen, these cases may be seasonal in temperate climates; worse clinical signs occur during warm weather. The onset of clinical signs may be historically correlated with an increase in insect or arachnid numbers in the environment.

Key Nutritional Factors

The key nutritional factors for foods and dietary supplements for omega-3 fatty acid-responsive skin diseases are summarized in **Table 32-6** and are discussed in more detail below.

OMEGA-3 FATTY ACIDS

The use of omega-3 fatty acids as antipruritic agents in dogs and cats has been the subject of numerous studies and considerable debate. The inflammation and dermatitis associated with allergic skin disease may be partially caused by abnormal EFA metabolism and inappropriate eicosanoid synthesis (White, 1993). A unique feature of skin is that it lacks Δ-6- and Δ-5-desaturase enzyme activity, and thus is incapable of making AA from LA or EPA from ALA (**Figure 32-3**) (Campbell, 1990). Skin can elongate GLA to dihomogammalinolenic acid (DGLA) and EPA to DHA. Normal dogs metabolize dietary sources of ALA to EPA and DHA elsewhere in the body. These fatty acids are then incorporated into the skin (Campbell and Roudebush, 1995).

DGLA, EPA and DHA in cutaneous cellular membranes may decrease inflammation through competition with AA for metabolic enzymes or because of the antiinflammatory nature of the eicosanoids produced (White, 1993). The rationale for specifically administering products high in GLA (an omega-6 [n-6] fatty acid) is that GLA can be incorporated into the skin, where it is rapidly elongated to DGLA. Because skin lacks

desaturase enzymes, DGLA is not further metabolized to AA. As a result, DGLA competes with AA for metabolic enzymes. Thus there is a decrease in AA-derived eicosanoids and an increase in the antiinflammatory eicosanoids PGE_1 via the cyclooxygenase cascade and 15-HETE via the lipoxygenase pathway. Specific recommendations for food amounts for GLA have not yet been determined. Thus, GLA is not listed as a key nutritional factor at this time. However, evening primrose, borage or black currant oils are used to increase dietary GLA intake.

ALA is an omega-3 (n-3) PUFA that is metabolized to EPA and DHA, and incorporated into the skin of normal dogs (Manku et al, 1982). Findings suggest that atopic dermatitis in human beings is associated with a deficiency of Δ-6-desaturase activity, which prevents the rapid conversion of ALA to EPA and DHA in atopic individuals (Manku et al, 1982, 1984). Comparable studies using atopic dogs and cats have not been published. However, one study suggested that subsets of atopic dogs exist with different fatty acid metabolic capabilities (Scott et al, 1997). Other studies suggest that ALA can modulate inflammatory and immune responses in normal cats (Chew et al, 2000). Flax, flax oil or linseed oil is typically used to provide ALA for supplements or foods.

The use of fatty acids for treating atopic dermatitis and chronic pruritus has been extensively studied in dogs (Scott et al, 1992, 1997; Scott and Buerger, 1988; Miller et al, 1989, 1992; Lloyd and Thomsett, 1989; Lloyd, 1989; Scott and Miller, 1990; Scarff et al, 1990; Paradis et al, 1991; Scarff and Lloyd, 1992; Bond and Lloyd, 1992, 1992a, 1993; White, 1992; Logas and Kunkle, 1994; Schick et al, 1995). Unfortunately, most of these studies have been uncontrolled, masked clinical trials using low doses of fatty acids for short periods. In these studies, 0 to more than 75% of pruritic patients had degrees of clinical improvement. Clinical studies using randomization, placebos and high doses of fatty acids for six weeks or more showed decreased pruritus in 0 to more than 50% of patients (Scarff and Lloyd, 1992; White, 1992; Logas and Kunkle, 1994). Dogs that did not have decreased pruritus still showed improvement in other clinical signs, including less erythroderma and skin edema. The benefit of fatty acid supplementation is maximized in dogs if other contributing diseases such as adverse reactions to food, flea hypersensitivity, bacterial pyoderma and *Malassezia* dermatitis are controlled. Overall, it is probably safe to inform clients that up to 50% of dogs with allergic pruritus will improve with modification in fatty acid intake, if secondary bacterial and yeast infections are controlled. Synergistic effects have been documented between fatty acids and other antipruritic agents such as antihistamines and glucocorticoids (Scott et al, 2001; Scott and Miller, 1990; Paradis et al, 1991). Fatty acid supplementation may also allow lower doses of antihistamines and glucocorticoids to be used to control clinical signs (Sævik et al, 2004; Bond and Lloyd, 1994; Paterson, 1995; Scott et al, 2001).

The use of fatty acids for management of allergic skin disease and papulocrustous (miliary) dermatitis in cats has been reported (Harvey, 1991, 1993, 1993a; Miller et al, 1993). More than

Table 32-8. The total essential fatty acid intake for a 10-kg adult dog eating 600 kcal (2,510 kJ) per day of selected commercial foods or being given one of the selected supplements.*

Dry foods	Food consumed (g)	Total omega-6 consumed (mg)	Total omega-3 consumed (mg)**
Hill's Prescription Diet b/d Canine	165	4,884	1,548
Hill's Prescription Diet d/d Potato & Duck Formula Canine	161	4,854	1,164
Hill's Prescription Diet d/d Potato & Salmon Formula Canine	162	4,206	2,100
Hill's Prescription Diet d/d Potato & Venison Formula Canine	161	4,932	1,146
Hill's Prescription Diet d/d Rice & Egg Formula Canine	154	4,692	990
Hill's Prescription Diet j/d Canine	176	4,032	5,688
Hill's Prescription Diet z/d Canine Low Allergen	163	4,812	618
Hill's Prescription Diet z/d ULTRA Allergen Free Canine	161	6,222	804
Hill's Science Diet Canine Active Adult	130	5,976	678
Hill's Science Diet Canine Adult Original	162	5,310	726
Hill's Science Diet Canine Lamb Meal & Rice Recipe Adult	162	4,815	1,002
Hill's Science Diet Canine Light Adult	200	5,988	618
Hill's Science Diet Canine Senior 7+ Original	163	4,590	1,710
Hill's Science Diet Sensitive Skin Dog	158	7,392	2,166
Iams Eukanuba Adult Maintenance Formula	139	4,800	600
Iams Eukanuba Reduced Fat Adult Formula	155	3,600	600
Iams Eukanuba Senior Maintenance Formula	142	3,600	600
Iams Veterinary Formulas Joint/Articulation	142	4,200	600
Iams Veterinary Formulas Response FP	147	1,600	400
Nutro Ultra Adult	166	7,998	1,290
Nutro Ultra Senior	168	7,392	1,380
Purina Veterinary Diets DRM Dermatologic Management Canine Formula	151	1,680	1,680
Royal Canin IVD Limited Ingredient Diets Potato & Duck Canine Formula	175	2,940	1,020
Royal Canin IVD Limited Ingredient Diets Potato & Rabbit Canine Formula	177	3,120	1,380
Royal Canin Veterinary Diet Hypoallergenic HP19	143	7,158	1,158
Royal Canin Veterinary Diet Sensitivity RC21	168	3,354	1,512
Royal Canin Veterinary Diet Skin Support SS21	153	4,884	1,758
Moist foods	**Food consumed (g)**	**Total omega-6 consumed (mg)**	**Total omega-3 consumed (mg)****
Hill's Prescription Diet a/d Canine/Feline	521	6,882	3,126
Hill's Prescription Diet d/d Duck Formula Canine	624	4,932	1,248
Hill's Prescription Diet d/d Lamb Formula Canine	451	3,972	1,488
Hill's Prescription Diet d/d Salmon Formula Canine	613	5,148	4,350
Hill's Prescription Diet d/d Venison Formula Canine	550	4,950	1,098
Hill's Prescription Diet j/d Canine	446	4,104	6,066
Hill's Prescription Diet n/d Canine	380	2,772	8,088
Hill's Prescription Diet z/d ULTRA Allergen Free Canine	617	6,102	738
Iams Veterinary Formulas Response FP	475	9,600	1,200
Royal Canin IVD Limited Ingredient Diets Duck Canine Formula	536	5,340	720
Royal Canin IVD Limited Ingredient Diets Whitefish Canine Formula	522	6,600	3,300
Supplements			
3V Caps for Large & Giant Breeds	1 capsule	0	417
3V Caps for Medium & Large Breeds	1 capsule	0	300
3V Caps for Small & Medium Breeds	1 capsule	0	171
3V Caps Liquid	0.75 ml	0	187
3V Caps Liquid HR	1 ml	0	450
DermCaps 100 lb	1 capsule	402	252
DermCaps ES	1 capsule	368	123
DermCaps ES Liquid	1 ml	375	130
DermCaps Liquid	1 ml	621	65
DermCaps Regular	1 capsule	402	108
Nutrived O.F.A. Granules	1 scoop	539	129
EicosaDerm	1 pump	0	600
Welactin	1 pump	0	330-364
Omegaderm – Small Dogs & Cats	1 packet (4 ml)	1,488	300
Nordic Naturals Omega-3	1 capsule	0	350
Nordic Naturals Arctic Cod Liver Oil	1 capsule	0	280
Nordic Naturals Ultimate Omega	1 capsule	0	700

*Adapted from Roudebush P. Consumption of essential fatty acids in selected commercial dog foods compared to dietary supplementation: An update. In: Proceedings. Annual Members Meeting AAVD & ACVD, Norfolk, VA, 2001: 53-54.
**Laboratory and clinical studies in a number of species have established a daily dosage for total omega-3 fatty acids that seems to be a reasonable starting point in patients with inflammatory disease. An initial dose of 50 to 300 mg of total omega-3 fatty acids/kg body weight/day seems to be effective in a large number of studies.

Table 32-9. The total essential fatty acid intake for a 4.5-kg cat eating 260 kcal (1,088 kJ) per day of selected commercial foods or being given one of the selected supplements.

Dry foods	Food consumed (g)	Total omega-6 consumed (mg)	Total omega-3 consumed (mg)[*]
Hill's Prescription Diet d/d Duck & Green Pea Formula Feline	68	2,254	473
Hill's Prescription Diet d/d Rabbit & Green Pea Formula Feline	69	2,304	460
Hill's Prescription Diet d/d Venison & Green Pea Formula Feline	67	2,142	458
Hill's Prescription Diet z/d Low Allergen Feline	69	3,630	419
Hill's Science Diet Adult Original Cat Food	64	2,301	140
Hill's Science Diet Mature Adult 7+ Original Cat Food	66	2,114	146
Hill's Science Diet Sensitive Skin Adult Cat Food	67	3,123	294
Iams Eukanuba Chicken & Rice Formula Cat Food	55	2,158	302
Iams Eukanuba Mature Care Formula for Cats	61	2,049	411
Royal Canin Adult Fit 32 Cat Food	68	2,462	322
Royal Canin Indoor 27 Cat Food	70	2,395	408
Royal Canin IVD Limited Ingredient Diets Green Pea & Venison Feline Formula	73	1,794	624
Royal Canin Persian 30 Cat Food	60	2,889	481
Royal Canin Skin Care 30 Cat Food	63	2,951	499
Royal Canin Veterinary Diet Feline Hypoallergenic HP 23	63	3,003	486
Royal Canin Veterinary Diet Feline Sensitivity RD 30	67	2,140	213
Moist foods	**Food consumed (g)**	**Total omega-6 consumed (mg)**	**Total omega-3 consumed (mg)[*]**
Hill's Prescription Diet a/d Canine/Feline	226	3,344	1,422
Hill's Prescription Diet d/d Duck Formula Feline	215	3,354	666
Hill's Prescription Diet d/d Rabbit Formula Feline	233	3,403	699
Hill's Prescription Diet d/d Venison Formula Feline	206	4,178	988
Hill's Prescription Diet z/d ULTRA Allergen Free Feline	241	2,574	289
Hill's Science Diet Savory Salmon Entrée Adult Cat Food	250	2,072	1,147
Iams Veterinary Formulas Response LB/Feline	199	2,600	520
Supplements			
3V Caps for Small & Medium Breeds	1 capsule	0	171
3V Caps Liquid HR	1 ml	0	450
DermCaps ES Liquid	1 ml	375	130
DermCaps Liquid	1 ml	621	65
DermCaps Regular	1 capsule	402	108
Nutrived O.F.A. Granules	1 scoop	539	129
EicosaDerm	1/2 pump	0	300
Welactin	1 pump	0	330-364
Nordic Naturals Omega-3	1 capsule	0	350

[*]Laboratory and clinical studies in a number of species have established a daily dosage for total omega-3 fatty acids that seems to be a reasonable starting point in patients with inflammatory disease. An initial dose of 50 to 300 mg of total omega-3 fatty acids/kg body weight/day seems to be effective in a large number of studies.

50% of allergic cats may improve, based on the results of uncontrolled, masked clinical trials published to date. Better clinical studies using randomization, placebos and masked protocols are needed in cats with allergic and other forms of dermatitis.

Laboratory and clinical studies in a number of species have established a daily dosage for total omega-3 fatty acids that seems to be a reasonable starting point in patients with inflammatory disease. An initial dose of 50 to 300 mg of total omega-3 fatty acids (ALA, EPA and/or DHA)/kg body weight/day seemed to be effective in a large number of studies (Endres et al, 1989; Lee et al, 1985; Logas and Kunkle, 1994; Kremer et al, 1987, 1995; Geusens et al, 1994; Hawthorne et al, 1992; Vaughn and Reinhart, 1994; Lorenz et al, 1989; Stenson et al, 1992).[d,e] As a food amount, this dose range translates to approximately 0.35 to 1.8% total omega-3 fatty acids (DM). Also, this total dose can be supplied through a combination of appropriate foods and supplements.

Dietary omega-3 fatty acid supplements are usually derived from cold-water marine fish oils. Food ingredient sources of EPA and DHA are usually fish meal or fish oil. Most commercial pet foods already exceed the omega-6 EFA requirement for LA by using vegetable oil and/or vegetable ingredients in their formulas.

Feeding Plan
Assess and Select the Food and/or Supplement
Patients with dermatitis having an inflammatory component may benefit from changes in dietary fatty acid intake. The most common modification is to increase omega-3 fatty acid intake and/or increase intake of GLA, an omega-6 fatty acid. **Table 32-7** lists typical pet food ingredients and supplements with their associated fatty acids.

Changing the food, adding a supplement or both can modify fatty acid levels in the overall diet. Initially, the EFA levels in the current food should be assessed. Unfortunately, information about fatty acid concentrations in commercial pet foods is sometimes difficult to obtain. This information is not typically found in guaranteed or typical analysis statements on pet food

Table 32-10. Summary of randomized, masked clinical studies using fatty acid supplements or fatty acid-enhanced foods in dogs with dermatologic disease.

Reference*	Dogs (no.)	Type of trial	Duration of therapy (weeks)	Control of pruritus (%)**	Control of clinical signs***
Scott et al, 1992	20	R,DB	2	25	–
Scarff and Lloyd, 1992	35	R,DB,PC	9	0	+
Bond and Lloyd, 1992a	21	R,DB,PC	8	76	+
Bond and Lloyd, 1992b	37	R,SB	16	64	+
Bond and Lloyd, 1993	28	R,DB	16	67	+
White, 1992	10	R,DB,PC	8	0	+
Logas and Kunkle, 1994	16	R,DB,X	6	56	+
Harvey, 1999	18	R,DB,PC	8	50	+
Paterson, 1995	32	R,SB,PC	12	50-75	+
Sævik et al, 2004	60	R,DB,PC	12	57	–
Taugbøl et al, 2004	24	R,DB,PC	10	53	–
Noli and Banni, 2004	24	R,DB,PC	8	50	–
Mueller et al, 2003	29	R,DB,PC	NR	9-15	–
Mueller et al, 2005	30	R,DB,PC	10	40-50	+
Nesbitt et al, 2003	58	R,DB	8	50	+
Sture and Lloyd, 1995	25	R,DB,PC,X	9	40	+

Key: NR = not reported, DB = double blind, PC = placebo controlled, SB = single blind, R = randomized, X = cross-over.
*The references for **Table 32-10** are available at www.markmorris.org.
**Percentage of dogs in which good to excellent pruritus control was reported.
***A + symbol indicates that improvement in clinical signs other than pruritus was noted (e.g., less erythroderma, less edema, less scale).

Table 32-11. Summary of clinical studies using fatty acid supplements in cats with dermatologic disease.

Reference*	Cats (no.)	Type of trial	Duration of therapy (weeks)	Control of pruritus (%)**	Control of clinical signs***
Harvey, 1991	8	Open	6	75	+
Harvey, 1993	11	Open	12	100	+
Harvey, 1993a	14	Open	12	78	+
Miller et al, 1993	28	Open	6	57	–

Key: Open = nonblinded.
*The references for **Table 32-11** are available at www.markmorris.org.
**Percentage of cats in which good to excellent pruritus control was reported.
***A + symbol indicates that improvement in clinical signs other than pruritus was noted (e.g., less erythroderma, less edema, less scale).

labels and is often not published by the manufacturer. In those cases, the manufacturer should be contacted directly to obtain information about fatty acid concentrations in specific products. **Tables 32-8** and **32-9** contain information about fatty acid concentrations in selected commercial dog and cat foods, respectively. These tables compare the fatty acid intake of dogs and cats eating specific foods and supplements. If the patient is given a supplement, the fatty acid concentrations in the supplement should also be determined. Most supplements marketed to improve skin and coat list the fatty acid concentrations on the product label or in published technical information. **Tables 32-8** and **32-9** also contain information about fatty acid concentrations in selected commercial fatty acid supplements.

In many cases, fatty acid supplements contain much lower concentrations of fatty acids than concentrations already found in the food being consumed by the patient (**Tables 32-8** and **32-9**). Thus, it may be more appropriate and convenient to change the patient's food to one with higher concentrations of appropriate fatty acids rather than adding a fatty acid supplement to the current food. In some clinical cases, changing the food and simultaneously adding a fatty acid supplement may be appropriate.

The optimal concentrations and ratios of fatty acids have not been established for normal dogs and cats or patients with clinical disease. Trial and error with various food and supplement combinations may be needed in an individual patient to achieve the best clinical response.

The risks and side effects of high levels of dietary fatty acids are few. Soft feces, overt diarrhea, flatulence and oral malodor ("fishy breath") are most commonly noted at levels of fatty acid supplementation used in most patients. These risks and side effects are outweighed by the possibility that fatty acid supplements will allow practitioners to reduce or discontinue corticosteroid therapy for pruritic dogs and cats. Other nutrients such as zinc, magnesium, biotin, pyridoxine, vitamin E and vitamin C are important cofactors in fatty acid metabolic pathways. Most commercial pet foods have adequate levels of these nutrients; routine supplementation would not be expected to improve clinical response. Many fatty acid supplements contain additional amounts of these cofactor nutrients.

Another criterion for selecting a food that may become increasingly important in the future is evidence-based clinical

nutrition. Practitioners should know how to determine risks and benefits of nutritional regimens and counsel pet owners accordingly. Currently, veterinary medical education and continuing education are not always based on rigorous assessment of evidence for or against particular management options. Still, studies have been published to establish the nutritional benefits of certain pet foods. Chapter 2 describes evidence-based clinical nutrition in detail and applies its concepts to various veterinary therapeutic foods.

Assess and Determine the Feeding Method
Other than supplementation, the method of feeding is often not altered in the nutritional management of allergic dermatitis. If a new food and/or a supplement is fed, the amount to feed can be determined from the product label or other supporting materials. The food dosage may need to be changed if the caloric density of the new food differs from that of the previous food. The food dosage is usually divided into two or more meals per day. The food dosage and feeding method should be altered if the animal's body weight and body condition are not optimal.

For clinical nutrition to be effective, there needs to be good client compliance. Enabling compliance includes limiting the patient's access to other foods and knowing who is responsible for feeding the food. If the patient comes from a multiple-pet household, it should be determined whether the pet with dermatitis has access to the other pets' food.

Reassessment
Allergic dermatitis patients receiving appropriate omega-3 fatty acid dietary intervention will usually respond over several weeks to several months (**Tables 32-10** and **32-11**). After a dietary change or supplement has been started, the patient should be examined every four weeks for significant improvement in pruritus or skin erythema. Some patients may not respond for several months or may need concurrent therapy with antihistamines, topical agents (medicated shampoo) or corticosteroids.

ACKNOWLEDGMENTS
The authors and editors thank Drs. Candace A. Sousa, Dawn E. Logas and William S. Swecker for their contribution to this chapter in the previous edition.

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CASE 32-1

Seborrheic Dermatitis in a Cocker Spaniel
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Patient Assessment
A four-year-old spayed female cocker spaniel had a two-year history of seborrhea. The dog had previously been treated with antibiotics, steroids and topical antiseborrheic shampoos with minimal improvement. The dog weighed 10 kg and had a body condition score of 3/5.

The only abnormalities noted on physical examination were an odoriferous generalized dermatosis and bilateral otitis externa. The dermatosis was characterized by erythematous and hyperpigmented hyperkeratotic plaques in which the hairs were coated with keratinaceous casts that formed "fronds" (**Figure 1**). Multiple papules and pustules were noted on the ventrum and dorsum. Both ear canals were mildly erythematous and swollen with a thick, yellow waxy discharge.

Skin scrapings for parasites and fungal culture for dermatophytes were negative. Tape preparations of the skin revealed many cocci. Ear cytology revealed numerous yeast organisms. A culture specimen from a pustule grew moderate numbers of *Staphylococcus intermedius* colonies that were sensitive to all antibiotics except penicillin, amoxicillin and tetracycline. Histopathologically, the hyperkeratotic plaques were characterized by marked follicular hyperkeratosis with distended follicular ostia, orthokeratotic hyperkeratosis of the epidermis and irregular epidermal hyperplasia (**Figure 2**).

Assess the Food and Feeding Method

For the three years before presentation, the dog had eaten a commercial specialty brand dry dog food supplemented with table foods.

Therapy Including Feeding Plan

The tentative diagnosis was vitamin A-responsive dermatosis with superficial pyoderma and yeast otitis. Treatment was initiated with 10,000 IU of vitamin A orally along with the dog's original food. The patient was also given an appropriate antibiotic for the pyoderma and a topical antifungal for the yeast otitis.

Questions

1. Why is vitamin A essential for normal epidermal function?
2. Why is this condition referred to as vitamin A-responsive dermatosis?
3. What are possible mechanisms by which vitamin A might correct the keratinization defect of this dermatosis?
4. How long must vitamin A be given to this dog and what potential side effects of vitamin A therapy might be expected?

Answers and Discussion

1. Vitamin A appears essential in the control of epidermal differentiation from basal cells to corneocytes. This is best illustrated by comparing the dermatologic signs of vitamin A deficiency with the signs associated with vitamin A excess. Mucous membrane epithelium is normally composed of nonkeratinizing cells. In vitamin A deficiency, nonkeratinizing mucous membrane cells are replaced by keratinizing cells and cells that normally keratinize in the skin become hyperkeratotic. The opposite response occurs when vitamin A is given in excess; mucous or ciliated squamous cells replace cells that normally keratinize.

2. Serum vitamin A levels have been normal in all of the reported cases of vitamin A-responsive dermatosis. This finding suggests that systemic vitamin A deficiency is an unlikely cause of the dermatosis. These cases also fail to show other clinical signs associated with vitamin A deficiency such as retinal degeneration, hind leg weakness and keratinization of mucous membranes. Improvement is noted within three to four weeks of starting oral vitamin A alcohol (retinol) supplementation, with complete remission by eight to 10 weeks. The specific cause of the dermatosis is unknown but may represent a local or functional deficiency of vitamin A.

3. Vitamin A may be able to correct this dermatosis via anti-keratinization effects. Vitamin A normalizes the proliferation of keratinocytes and decreases the epidermal hyperproliferation. Vitamin A also alters the expression of certain structural genes that are important in epidermal differentiation and cornification. Examples include the suppression of transglutaminase, which is important in cell envelope formation, and the alteration of keratins to K19 and K13, which are normally not found in adult skin but are in fetal skin. Finally, vitamin A induces growth factors and the expression of growth factor receptors that also suppress epidermal differentiation.

4. These dogs usually must be given vitamin A for life. Discontinuing vitamin A supplementation usually results in recrudescence of dermatologic signs. Dogs generally tolerate vitamin A therapy quite well with minor side effects. Vitamin A should be used with caution in breeding animals because it may be teratogenic.

Figure 1. Hyperpigmented, hyperkeratotic plaques with fronding on the ventrum of a four-year-old cocker spaniel.

Figure 2. Skin biopsy specimen from a seborrheic cocker spaniel. The epidermis is mildly hyperplastic and hyperkeratotic. There is severe follicular hyperkeratosis and dilatation. (Magnification 10X.)

Progress Notes

The dog has done very well as long as vitamin A supplementation has been maintained. Cutaneous lesions reappeared when vitamin A supplementation was discontinued.

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CASE 32-2

Recurrent Pyoderma in a Chesapeake Bay Retriever

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Patient Assessment

A two-year-old intact male Chesapeake Bay retriever was presented with a 12-month history of recurrent bacterial pyoderma and seborrhea sicca. The dog had previously been treated with two- to three-week courses of various antibiotics at appropriate doses. Response to therapy was partial; papules and pustules would resolve but epidermal flakes and dry brittle hair persisted. All dermatologic signs including papules and pustules would return within weeks after antibiotic therapy was discontinued. Historically, the dog had no other problems.

The dog was slightly underweight (body condition score 2/5), normothermic, alert and well hydrated. Mucous membranes were pink and capillary refill time was 1.5 seconds. Lymph node size, chest auscultation and abdominal palpation were all within normal limits. No abnormalities were noted on ocular or musculoskeletal examination. Both ears had a slight accumulation of yellow waxy exudate but were not inflamed. The coat was thin, dry and lacked sheen. The dermatosis was generalized, sparing only the head and feet. It consisted of large white flakes, papules, pustules, epidermal collarettes and crusts. The dog was only mildly to moderately pruritic; the pruritus historically abated with antibiotic therapy.

Skin scrapings for parasites and fungal culture for dermatophytes were negative. Tape preparation of the skin revealed moderate numbers of cocci bacteria. *Staphylococcus intermedius* was cultured from a pustule. Thyroid profile results were within normal limits.

Assess the Food and Feeding Method

The dog was fed a grocery store brand dry puppy food until 10 months of age at which time the client switched to a generic dry adult dog food. The owner would often purchase whatever generic dog food was on sale.

Therapy Including Feeding Plan

The dog was diagnosed as having a recurrent pyoderma possibly secondary to malnutrition associated with consumption of generic dog food. The pyoderma was treated for six consecutive weeks with an appropriate antibiotic. The food was changed to a grocery brand dry adult dog food supplemented with one tablespoon of corn oil for 12 weeks.

Questions

1. Which major nutrients are essential for normal epidermal function and how might dermatoses due to malnutrition occur in patients eating generic pet foods?
2. How might consumption of generic food have contributed to the dog's recurrent pyoderma?
3. Could nutritional deficiencies account for the dermatologic signs (other than the pyoderma) observed in this dog?

Answers and Discussion

1. Nutrients of special concern for maintaining normal skin and hair include protein, essential fatty acids (EFA), copper, zinc and certain vitamins. A previous report summarized the dermatologic signs that occurred in 13 dogs fed generic pet foods. Generic pet foods are marketed based on low daily cost of feeding. The low cost of the food is usually achieved by using ingredients that often have low total digestibility, low nutrient availability, high mineral content and low quantities of fat and EFA. Foods with high mineral levels (calcium, phosphorus, magnesium), poor digestibility, high levels of phytate and/or low levels of total fat and EFA are a significant risk factor for zinc-responsive cutaneous disease. See the section of this chapter that discusses the dermatologic signs associated with fatty acid and zinc deficiency.

2. Malnutrition resulting from consumption of generic dog food may have contributed to the recurrent pyoderma in this dog. Decreases in zinc and EFA can lead to changes in the microenvironment of the stratum corneum, which allow pathogenic bacteria to colonize the surface of skin. Once colonized, the skin may also be less able to control the infection because decreases in zinc, protein and EFA may diminish normal humoral and cellular immunity. Zinc deficiency impairs macrophage phagocytosis, diminishes chemotaxis and leads to lymphopenia. Decreased levels of EFA, which are normally converted to potent inflammatory mediators called eicosanoids, lead to decreases in chemotaxis, margination and killing ability of leukocytes, particularly neutrophils. Inadequate protein intake, particularly of essential amino acids, can alter the immune response.

3. EFA and zinc deficiency could account for the dry dull coat, fine scale and hyperkeratotic crusts. EFA are necessary for the formation of lamellar granules, which contain much of the epidermal lipids in dogs. These lipids are essential for the formation of the transepidermal water barrier. Without these lipids, the stratum corneum water loss increases and fine scales are formed. EFA are also necessary for the formation of normal sebaceous gland lipids. The sebaceous lipids are important for coating hairs and giving them their sheen.

   Zinc is necessary for normal keratinization. Although the exact mechanisms of zinc's effect on keratinization are unknown, they may be related to the many zinc metalloenzymes, which are found in the epidermis and are essential for epidermal cell differentiation. Therefore, zinc deficiency could lead to the hyperkeratotic crusts noted in this case.

Progress Notes

After six weeks of therapy there was no evidence of bacterial pyoderma or dry flakes. The patient's coat was much softer, shinier and fuller. Eight months after discontinuing antibiotic therapy, the dog's coat and skin remained normal with no recurrence of the pyoderma.

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CASE 32-3

Crusting Dermatitis in a Bull Terrier

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Patient Assessment

A four-year-old spayed female bull terrier was examined for severe skin crusting. The crusting had been present since the dog was five months old and had progressively worsened. Previous diagnostic tests included multiple skin scrapings for parasites (negative), a fungal culture (negative) and failure of a clinical response to several empirical therapies (antibiotics, shampoos).

Physical examination demonstrated a mature bitch that was well fleshed but smaller than breed standards (11.7 kg, body condition score 3/5). There were no abnormal physical findings other than those related to the integument. The coat was dull and brittle. Cream-colored patches of thick, tightly-adherent crusts were present above each eye, within the inner pinnae (**Figure 1**) and overlying the elbows and hocks. All of the footpads were thickened and cracked with "feathers" of adherent keratin extruding from the edges (**Figure 2**).

Results of a complete blood count, serum biochemistry profile and urinalysis were normal. Histopathology of skin biopsy specimens revealed marked parakeratotic hyperkeratosis. Neutrophils infiltrated the superficial dermis with some exocytosis, spongiosus and scattered individual dyskeratotic keratinocytes in the epidermis.

Assess the Food and Feeding Method
The dog had been fed a dry commercial food formulated for puppies for the first 10 months of life and several different dry dog foods formulated for adult maintenance for the next three years.

Therapy Including Feeding Plan
Based on the age at onset, breed, diagnostic results and a lack of response to various therapies, a tentative diagnosis of acrodermatitis of bull terriers was made.

Questions
1. What nutritional therapy should be recommended for this dog?
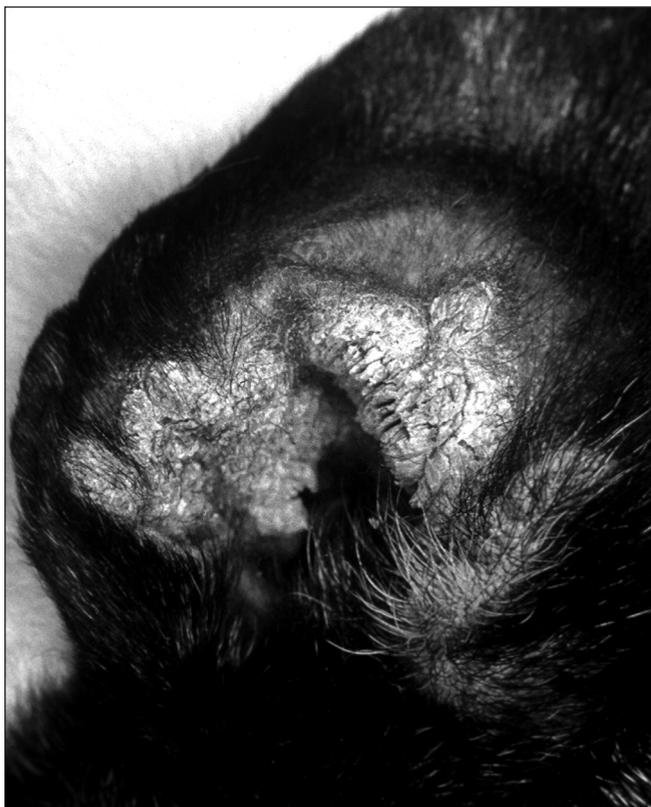2. What other information should the owner be given regarding this disease and the prognosis for the dog?

Figure 1. A four-year-old bull terrier with thick crusts in the inner pinna at the ear canal entrance.

Figure 2. The same dog with hyperkeratosis of the footpads.

Answers and Discussion
1. Acrodermatitis develops in bull terriers shortly after birth and is associated with defects in zinc absorption and metabolism. Cutaneous and systemic clinical signs resemble severe zinc deficiency with growth retardation, gastrointestinal disease, chronic bacterial infections and progressive, erythematous, exfoliative, papular to pustular dermatitis of the distal extremities and skin surrounding the mucocutaneous junctions. Surface crusts usually contain numerous bacteria and yeast organisms.

   Supplementation of the food with oral or intravenous zinc usually fails to ameliorate clinical signs. Treatment with systemic antimicrobials, especially for secondary superficial yeast infections, may result in marked improvement, although systemic and cutaneous infections recur. This dog was treated with a zinc methionine supplement (50 mg once daily) and oral cephalexin (250 mg b.i.d.) for secondary pyoderma.

2. Acrodermatitis is an inherited, autosomal recessive metabolic disease reported to occur in bull terriers in the United States, Canada and the United Kingdom. This bitch had already been spayed but further breeding of this dog's parents should be discouraged. The condition has been termed lethal acrodermatitis because homozygously affected dogs rarely live beyond 18 months of age. Some of the apparently normal littermates may develop zinc-responsive dermatitis. Owners of affected dogs usually complain that their pets have skin disease, stunting, difficulty with eating and large, splayed, painful feet. Ulcerated and thick, crusted lesions are prominent on the muzzle and ears. Abnormal keratinization of the footpads, severe nail dystrophy and paronychia are also common. Prognosis is guarded to poor for severely affected dogs.

Progress Notes
After eight weeks of therapy, the crusting shown in the pictures had decreased about 30%. At that point, the dog was lost to further evaluation.

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CASE 32-4

Crusting Dermatosis in a Siberian Husky Crossbred Dog

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Patient Assessment

A four-month-old male Siberian husky crossbred dog weighing 18 kg was presented for evaluation of an eight-week history of variable but persistent crusting. The lesions were first noticed above the eyes and around the mouth, but now extended to the chin and neck. A fungal culture was negative for dermatophytes. Therapy with topical agents, cephalexin and griseofulvin resulted in no clinical improvement. No history was available for either the parents or related dogs.

Physical examination revealed a bright, active and alert puppy with a body condition score of 3/5 (ideal). The only abnormalities noted were limited to the skin. Thick, tightly adherent white crusts were noted above both eyes. The outer ear pinnae were alopecic and crusty. Scattered, white, tightly adherent crusts 1 to 3 cm in diameter were found around the lip margins (**Figure 1**) and ventral neck.

Histopathology of multiple skin biopsy specimens demonstrated severe irregular acanthosis accompanied by prominent parakeratosis and marked serocellular crusting. The parakeratosis extended into the superficial hair follicles. A mixed inflammatory infiltrate, which included lymphocytes, macrophages, neutrophils, plasma cells and scattered eosinophils, was found beneath the acanthotic and multifocally spongiotic epidermis.

Assess the Food and Feeding Method

The owners fed the dog a combination of commercial moist and dry foods formulated for puppies after they obtained it from a private home at nine weeks of age.

Questions

1. Given the signalment and clinical signs, what diseases should be considered?
2. What are the risk factors for development of zinc-responsive cutaneous disease in dogs?
3. What are the best methods to diagnose zinc-related cutaneous disease in animals?
4. Outline an appropriate feeding plan for this puppy.

Answers and Discussion

1. The list of differential diagnoses for this dog should include demodicosis, dermatophytosis, bacterial pyoderma, primary keratinization defect (e.g., ichthyosis), nutritional dermatosis (vitamin A-responsive or zinc-responsive dermatosis) and autoimmune skin disease (e.g., pemphigus foliaceus, pemphigus erythematosus and lupus erythematosus). The histopathologic changes were most compatible with a zinc-responsive dermatosis and secondary pyoderma.

2. Risk factors for zinc-responsive skin disease in dogs include breed, high mineral or phytate levels in the food, low essential fatty acid levels, supplementation with calcium or other minerals and small intestinal disease resulting in malabsorption or maldigestion. Breeds in which zinc-responsive disease has been reported to occur include Siberian huskies, malamutes, bull terriers, Great Danes, Labrador retrievers and other rapidly growing large- and giant-breed dogs.

3. Diagnosis of zinc-responsive cutaneous disease is based on the history, physical examination and skin biopsy results.
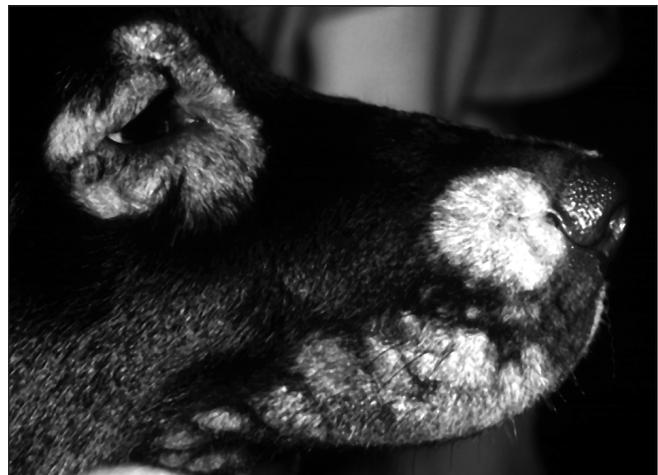

Figure 1. A four-month-old male Siberian husky cross with marked alopecia, lichenification and crusting of the periocular and perioral skin.

Hyperplastic superficial perivascular dermatitis with marked diffuse and follicular parakeratotic hyperkeratosis is suggestive of zinc deficiency. In general, concentrations of zinc in serum, leukocytes and hair are not good indicators of zinc status in dogs.

4. The tentative diagnosis was zinc-responsive dermatosis, which is often seen in Siberian husky dogs. Treatment generally includes changing to a food that avoids excess minerals and contains adequate amounts of zinc and essential fatty acids. Zinc supplementation will be necessary in those breeds in which decreased ability to absorb zinc is suspected. Siberian huskies are one such breed.

Progress Notes
Initial therapy consisted of feeding the moist food supplemented with 50 mg of zinc given orally once daily. Within two months, the lesions disappeared and the dog's coat had returned to normal. When the dog was 10 months old, the owner discontinued the zinc supplement and lesions began to return. Based on this finding, the dog will probably need zinc supplementation for the remainder of its life. Some Siberian huskies do not respond to oral zinc supplementation; however, intravenous injections of zinc sulfate solutions may be effective in these animals.

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CASE 32-5

Pruritus and Seborrhea in a Wire-Haired Fox Terrier
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Patient Assessment
A four-and-one-half-year-old, castrated male wirehaired fox terrier was examined for a two-year history of pruritus, oily coat and red skin bumps. Initially, the problems had been seasonal (occurring in the summer), but this year they did not clear up during the winter months. The pruritus had been responsive to oral prednisone. Physical examination revealed diffuse mild seborrhea oleosa with moderate erythema and scaling. These lesions were worse on the dorsum. Hypotrichosis with pustules and crusts were also found on the dorsum and in the axillae. Interdigital erythema was also present. The dog weighed 14 kg and had a body condition score of 4/5.

The initial evaluation of these problems included skin scrapings (negative) and interdigital skin cytology (no abnormal findings). Diagnosis was superficial bacterial pyoderma with seborrheic dermatitis and possible underlying allergic disease. Treatment was initiated with an oral antibiotic (cephalexin, 250 mg, t.i.d.), an antiseborrheic/antibacterial shampoo (twice weekly baths), and a six-week dietary elimination trial using a combination of commercial moist and dry therapeutic foods containing venison and potato.[a]

Six weeks later the dog weighed 13.5 kg and the owner reported a 50% improvement in the pruritus. Examination revealed no visible signs of bacterial pyoderma but erythema persisted in the axillary and interdigital areas.

Assess the Food and Feeding Method
The dog was normally fed a mixture of various dry and moist commercial grocery brand foods supplemented with occasional table foods (rice, potatoes, pasta) and various commercial biscuit snacks. The commercial venison and potato veterinary therapeutic foods were used for six weeks as part of a dietary elimination trial. The owners were instructed not to feed any other commercial foods, table foods or snacks during this trial.

Questions
1. What additional diagnostic tests would be helpful in this patient?
2. What dietary changes may help manage the pruritus and dermatitis in this patient?

Answers and Discussion

1. Underlying allergic disease due to atopy, flea allergy or food allergy could cause the pruritus, dermatitis and seborrhea seen in this patient and predispose the dog to secondary pyoderma. Atopy can be ruled out with intradermal and in vitro allergy testing. There was no evidence of flea infestation. The clinical improvement was probably due to elimination of the superficial bacterial pyoderma.

2. Supplementing the current food with fatty acids or changing to another food with higher fatty acid levels may benefit this patient. Fatty acid therapy alone is rarely successful in controlling moderate to severe pruritus in most patients with skin disease, but may be effective when used concurrently with other therapies. A synergistic effect between fatty acid and antihistamine administration has been documented in some clinical trials involving allergic dogs.

Progress Notes

Intradermal skin testing was positive for a few weed antigens. Blood was drawn for in vitro (ELISA) allergy testing. Positive reactions were found to house dust mites, several trees, several grasses and several weeds, including ragweed. The probable diagnosis was atopy with secondary superficial pyoderma and seborrhea. Treatment was initiated with an antihistamine (hydroxyzine, 25 mg, t.i.d.) and a fatty acid supplement[b] (1 capsule twice daily with food) that delivered 500 mg of eicosapentaenoic acid (EPA) daily in addition to the fatty acids in the food. Bathing with the shampoo was continued.

Eight weeks later the owners reported some improvement in the level of pruritus, but evidence of self-induced alopecia persisted (barbered hairs on the dorsal lumbar region). The skin was also erythematous; salivary staining was evident in these areas. The bathing, fatty acid supplementation and hydroxyzine administration were continued. Hyposensitization injections were started using allergens identified by the ELISA performed two months earlier. The veterinary therapeutic foods were fed until they were gone (about two weeks). At that time, the owner began feeding the dry and moist grocery store brand foods fed previously.

The owner reported significant improvement eight weeks later. There were no areas of visible erythema on the skin, but salivary staining persisted on all four feet. There was mild oiliness and scale accumulation on the dorsum. Bathing, hydroxyzine administration, hyposensitization injections and fatty acid supplementation were continued.

Further Discussion

The optimal dose of omega-3 fatty acids and γ-linolenic acid for control of inflammation and pruritus has not been established. The levels of these fatty acids in the grocery brand foods the patient was eating were unknown. The venison and potato veterinary therapeutic foods provided approximately 50 mg of omega-3 fatty acids per day to the patient. The supplement provided an additional 500 mg of EPA, which markedly increased total omega-3 intake. This dosing level may have been enough additional omega-3 fatty acids (36 to 40 mg EPA/kg body weight) to benefit this patient. The clinical improvement was probably attributable to the combination of all therapies used in this dog.

Endnotes

a. IVD Limited Ingredient Diets. Nature's Recipe Pet Foods, Corona, CA, USA.
b. 3V Caps Skin Formula. DVM Pharmaceuticals, Miami, FL, USA.

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