Skin and Coat in Dogs

Key Points

- **The skin is particularly sensitive to alterations in its nutrient supply, but its response to dietary changes may take several months to appear.**
- **Nutritional deficiencies are rare but may occur if the diet is poorly formulated or stored, if the animal's intake is reduced or if it cannot absorb or utilize the nutrient as result of disease or genetic factors.**
- **Most nutrient deficiencies produce a similar range of clinical signs reflecting an impairment of keratinization, sebum production, and hair growth.**
- **Typical signs are those of seborrhea and include excessive scale, erythema, alopecia or poor hair growth, greasy skin, secondary pyoderma, and pruritus.**
- **Some nutrients may be administered in supraphysiologic doses to produce a therapeutic effect in certain nutrient-responsive dermatoses.**
- **Dietary sensitivity is rare but may present as a pruritic skin disorder or with other cutaneous signs.**
- **Protein malnutrition is most likely to occur following reduced intake, failure to compensate for transiently increased requirements, or increased protein losses in chronic disease.**
- **Essential fatty acid deficiency may result from feeding poor quality, low fat dry foods or inappropriate home-prepared diets, following oxidative damage to fatty acids in food, or as a sequel to fat malabsorption.**
- **Therapeutic supplementation with essential fatty acids may help in the management of certain skin disorders associated with abnormalities of fat metabolism (e.g., canine idiopathic seborrhea) or hypersensitivity (e.g., atopy, flea allergic dermatitis, dietary hypersensitivity). In the case of pruritic skin disease, essential fatty acids (particularly omega-3 fatty acids) may help to attenuate the inflammatory response through their effect on eicosanoid production.**
- **Zinc deficiency is now uncommon but has been associated with poor quality diets in which dietary interactions have reduced the availability of zinc.**

- **Two zinc-responsive dermatologic syndromes are recognized: *Syndrome I* is associated with an inherent defect of zinc absorption, especially in Siberian Huskies and Alaskan Malamutes, and may occur despite feeding a nutritionally adequate diet. Lifelong therapeutic supplementation may be required. *Syndrome II* may occur in large, rapidly growing dogs with high zinc requirements when fed a diet which is relatively zinc-deficient due to high phytate or calcium levels. These cases respond to dietary correction and oral zinc supplementation**
- **Vitamin A deficiency is rare in dogs, but a vitamin A-responsive dermatosis has been reported in Cocker Spaniels. Affected animals exhibit a generalized defect in keratinization.**
- **Vitamin E has been used therapeutically for its antiinflammatory effect in the management of canine discoid lupus erythematosus and primary acanthosis nigricans.**
- **Biotin deficiency may occur if the diet contains large amounts of avidin, contained in raw egg white. The vitamin has also been used as a supplement in the treatment of various dermatoses and disorders of keratinization.**
- **Deficiencies of other B-complex vitamins, including riboflavin, niacin, and pyridoxine, may also result in the development of skin lesions.**

The skin is the largest organ of the body and performs a multitude of functions. Its high level of metabolic activity creates a heavy physiologic demand for protein and other nutrients and makes it particularly sensitive to subtle changes in its own nutrient supply. Dietary factors can therefore have a significant effect on skin and coat condition in dogs and cats and may impact on the etiology and therapy of skin disease in three areas:

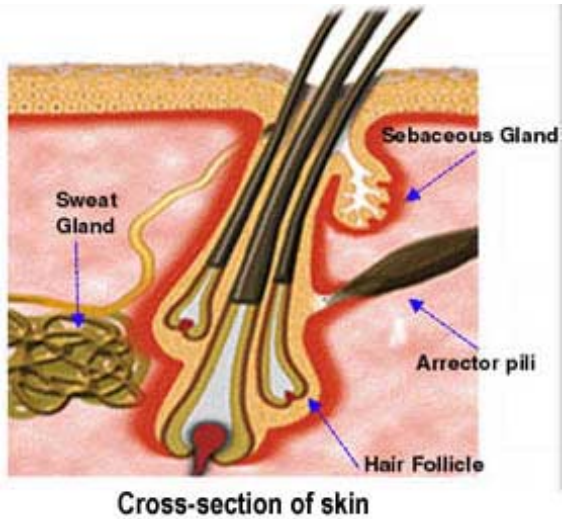
- **Nutrient deficiency or imbalance**
- **Nutritional supplementation for therapeutic effect**
- **Dietary sensitivity**

ANATOMY

Skin

Adult skin is composed of three layers:

- The epidermis
- The dermis
- The hypodermis or subcutis



Cross-section of skin

Epidermis

The epidermis is the outermost layer of the skin. All epidermal cells are derived from the basal membrane *(stratum basale),* which is composed primarily of keratinocytes although other cells, including melanocytes, are also present. Keratinocytes produced by the basal membrane differentiate sequentially to form:

- The prickly cell layer *(stratum spinosum*)
- The granular layer *(stratum granulosum)*
- The clear layer *(stratum lucidum)* only in the footpads and, to a lesser extent, the nose
- The outermost horny layer *(stratum corneum),* which consists of nonnucleated, fully keratinized cells

Keratinocytes have many functions, including:

- Production of keratin, a fibrous, sulfur-containing protein
- Production of a lipid secretion which has an integral role in the regulation of the *stratum corneum* barrier function and desquamation

Dermis

The dermis supports the epidermis and consists of a matrix of collagen and reticular and elastic fibers in a ground substance of chondroitin sulfate and hyaluronic acid. Cells present in this layer are fibroblasts, mast cells, and histiocytes, although other cell types may be present in certain disease conditions.

The tensile strength and elasticity of the skin is largely attributable to the dermis, which is also responsible for the maintenance and repair of the skin and modifies the structure and function of the epidermis.

Hypodermis

The underlying hypodermis is made up of loose connective tissue, elastic fibers, and variable amounts of fat. This layer acts as an energy reserve, as an insulator, and as protective padding and maintains the body contours.

Hair

Three types of hair are present in dogs:

- The primary or guard hairs
- The fine secondary hairs
- The tactile or sinus hairs, including the whiskers, which are responsible for the perception of touch

Each hair is divided into a free part or shaft, and a proximal part or root. The hair shaft consists of:

- The inner medulla, forming an axial cord of cells
- The cortex, composed of tightly packed keratin and pigment
- The cuticle, a single layer of flat, cornified cells that overlap and are tightly pressed to the cortex

The hair is housed in an epithelial pit called a hair follicle and is attached, via the hair bulb, to the dermal papilla in the base of the follicle. It is here that mitotic activity occurs, which leads to the production of the hair matrix. Melanocytes, which produce the pigment melanin, are situated in the hair bulb.

Associated with the hair follicles are:

- Sebaceous glands, except in the footpads or on the nose, which produce sebum
- An erector pili muscle which elevates the hair and helps in the expression of sebum
- Apocrine sweat glands (except in the skin of the footpads and nose), which have a predominantly pheromonal, rather than a thermoregulatory, function
- Eccrine sweat glands (only in the skin of the footpads), which are activated under nervous control, particularly in stressed or excited dogs

FUNCTION

Major functions of the skin and coat include:

- Maintenance of an enclosing natural barrier between the animal's internal environment and the outside world
- Preservation of the animal's shape
- Protection against water loss
- Protection from physical, chemical, and microbial injury imposed by external agents
- Storage of nutrients
- Sensory perception
- Thermoregulation
- Vitamin D synthesis
- Important indicator of health status
- Significant role in communication (e.g., pilo erection, excretion of pheromones)

The protective function of the skin and coat is enhanced by the presence of an emulsion of sebum and sweat that permeates the cells of the *stratum corneum*. This emulsion also:

- Provides a physical barrier
- Maintains skin hydration to keep it soft and pliable
- Spreads over the hair coat to produce a glossy sheen
- Contains antimicrobial substances
- Is immunologically active

The presence of pigment, immunocytes, and the normal skin microflora also contribute to the skin's defense system.

Hair Growth

Hair growth in the dog is cyclic, and each cycle consists of:

- **Anagen** - the active growing phase
- **Catagen** - the transitional phase
- **Telogen** - the resting phase

In dogs, hair replacement occurs in a mosaic pattern so there is no synchronized single period of hair shedding. Neighboring follicles are in different phases of the hair cycle at any one time. Domestic dogs tend to molt continuously throughout the year, with peaks of activity during spring and autumn.

Factors that affect hair growth in dogs include:

- Day length
- Ambient temperature
- Hormonal status
- Health status
- Nutrition

CLINICAL DISORDERS

Nutritional factors have a role in the etiology and management of skin disease in three broad areas:

- Nutritional deficiency or imbalance
- Nutritional supplementation for therapeutic effect
- Dietary sensitivity

Nutrient Deficiencies or Imbalances

Nutritional deficiencies are now rarely encountered in companion animals due to the widespread feeding of complete and balanced pet foods. Deficiencies may occasionally arise:

- When the animal's intake is reduced
- When the diet is poorly formulated or stored
- When the animal is unable to digest, absorb, or utilize the nutrient as a result of disease or genetic factors

Dietary interactions that reduce nutrient availability can result from:

- Errors in formulation
- Prolonged storage
- Oversupplementation of an otherwise balanced diet

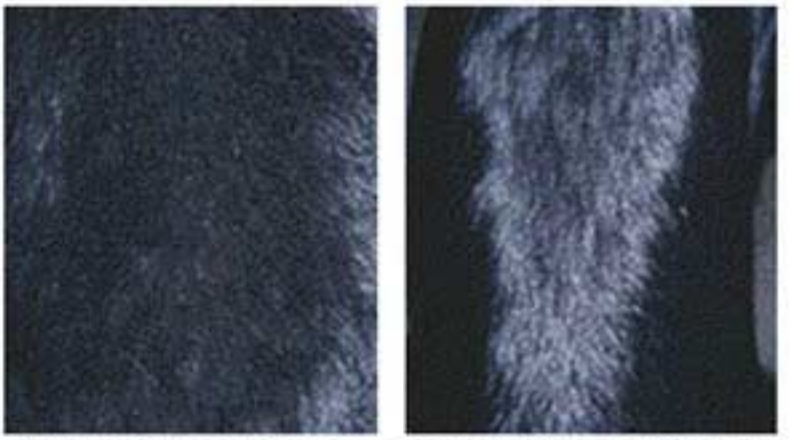
Where dietary errors are identified, it is usually simpler to feed a balanced commercial diet than to attempt to correct single deficiencies in a poorly formulated diet.

Many nutrient deficiencies may be associated with skin disorders, but most produce a range of similar clinical signs. In general, nutrient deficiencies produce seborrheic skin changes that reflect impairment of the fundamental metabolic processes of:

- Keratinization
- Sebum production
- Hair growth

Typical signs of a nutritional dermatosis include:

- Excessive scale
- Erythema
- Alopecia or poor hair growth
- Greasy skin, often accompanied by secondary bacterial infection and pruritus



Coat with dandruff Coat with healthy gloss

It is generally accepted that signs become evident only after deficient diets are fed for several weeks to months.

The following nutrients are crucial in maintaining optimal skin and coat condition:

- Protein
- Essential fatty acids
- Zinc
- Vitamin A
- Vitamin E
- B-complex vitamins

Nutritional Supplementation for Therapeutic Effect

Although nutritional supplements may be used to treat skin disorders arising from nutrient deficiency or imbalance, dietary correction is achieved more effectively by feeding a high quality commercial diet that is nutritionally complete and balanced.

Supplementation with specific nutrients may, however, be beneficial in the management of certain nutrient-responsive dermatoses. In such cases, supraphysiologic doses of the nutrient are required and its action is likely to be of a pharmacologic nature rather than merely correcting a deficiency.

Protein

Etiology

Protein deficiency is rare in clinical practice but is occasionally encountered following:

- Starvation
- Disease-induced inappetence and anorexia

- Prolonged feeding of a poorly formulated or inappropriate diet

In the short term, primary protein deficiencies are most likely to occur when requirements are transiently increased (e.g., as in young, growing animals and in pregnant or lactating females.

Alternatively, protein malnutrition may be associated with excessive protein loss, which may occur in certain chronic illnesses such as:

Protein-losing nephropathy
Protein-losing enteropathy
Excessive exudative disorders, such as pyothorax or burns

Protein malnutrition may also be seen in critically ill animals with increased protein requirements and decreased intake, as well as in patients with endocrine pancreatic insufficiency with protein malabsorption.

Pathophysiology and Clinical Signs

The protein requirement for normal growth of hair and keratinization of the skin may account for between 25-30% of the animal's daily protein requirement. Failure to meet this demand results in the cutaneous manifestations of protein malnutrition including:

- Brittle, depigmented hair that is easily shed and slow to regrow
- Excessive scaling
- Thin, inelastic, and hyperpigmented skin

Dietary Management

Dietary correction involves supplementation with high quality protein sources such as meat, eggs ,and milk, but the prognosis may be complicated by the presence of underlying disease.

Essential Fatty Acids

Etiology

Dietary deficiencies of essential fatty acids (EFA) are uncommon but may occasionally occur in association with:

- Poor quality, low fat dry foods or inappropriately formulated home-prepared diets
- Oxidative destruction of EFA in food due to prolonged storage or insufficient inclusion of antioxidants, such as vitamin E
- Fat malabsorption due to hepatic, pancreatic, or gastrointestinal disease, although this is rare

Pathophysiology

Fatty acids of the omega-6 and omega-3 series perform a range of functions, many of which are vital for maintaining normal skin structure and function. They:

- Have a structural role in cell membranes
- Are involved in the maintenance of the cutaneous water permeability barrier
- Act as precursors for eicosanoids, such as prostaglandins
- Regulate epidermal proliferation

Dogs are unable to synthesize linoleic acid and therefore require a dietary source from which other omega-6 fatty acids may be derived. There may also be a subtle dietary requirement for omega-3 fatty acids in some physiologic states, and it has been implicated that docosahexaenoic acid (DHA) plays an important role in the development of retinal and brain tissue in the developing animal.

Clinical Signs

Following the introduction of a deficient diet, cutaneous signs of EFA deficiency may appear within 2-3 months and are related to abnormalities of keratinization. Initially, there is a reduction in surface lipid production, but, subsequently, sebum production is increased.

Early signs include:

- Dull, dry coat
- Fine scale

If prolonged, EFA deficiency results in:

- Alopecia
- Greasy skin, particularly in the ears and between the toes
- Pruritus
- Secondary pyoderma

Severe cases may also present with systemic signs including:

- Poor wound healing
- Reduced immunocompetence
- Growth retardation
- Infertility

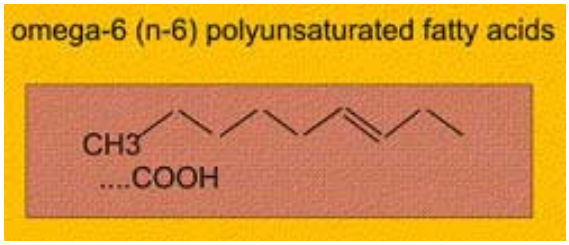
Dietary Management

Dietary correction of EFA deficiency may be achieved by:

- Changing to a higher fat, premium quality diet
- Supplementing the diet with oils

- Administration of proprietary fatty acid supplements

Where the deficiency is uncomplicated by other factors, a response is usually visible within 3-8 weeks.



Typically, omega-6 fatty acids are obtained from plant seed sources or terrestrial animal flesh, whereas omega-3 fatty acids are principally found in marine lipid and some terrestrial plant sources. Although vegetable oils, such as sunflower oil, are a rich source of linoleic acid, appreciable quantities of its derivative arachidonic acid are found only in animal fats

.A general recommendation for dietary supplementation with linoleic acid is based on supplying 10-15 ml of oil for a 25 kg dog.

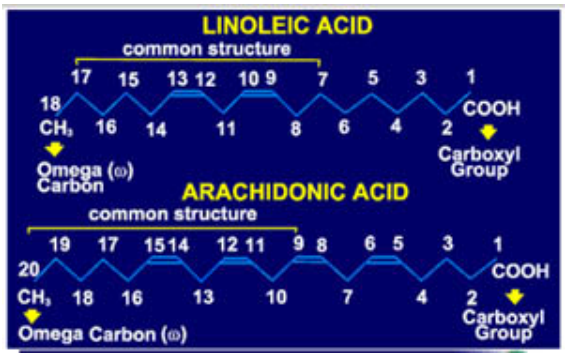
Increasing the dietary polyunsaturated fatty acid (PUFA) content simultaneously increases the requirement for vitamin E and may also increase the requirement for other vitamins and minerals involved in fatty acid utilization. In most cases it is preferable to feed a better quality prepared pet food or to provide a balanced veterinary supplement containing essential fatty acids, vitamin E, and zinc.

Therapeutic supplementation

Essential fatty acid supplements have also been used therapeutically in the management of a number of skin disorders, particularly those associated with hypersensitivity reactions or with abnormalities of fatty acid metabolism (Campbell 1993). In the dog, conditions that may respond to EFA supplementation include:

- Canine idiopathic seborrhea
- Canine atopy
- Flea-allergic dermatitis
- Dietary hypersensitivity

Supplementation with linoleic acid is indicated for dogs with keratinization defects, such as idiopathic seborrhea, because of its effect on the cutaneous permeability barrier and the ability of its metabolite arachidonic acid to regulate epidermal proliferation. A general recommendation for dietary supplementation with linoleic acid is based on supplying 10-15 ml of oil for a 25 kg dog.



Dietary PUFA supplementation has also been used in the management of certain inflammatory conditions, including pruritic skin disorders associated with hypersensitivity. In such cases,

dietary PUFA may help to control clinical signs and can reduce or eliminate the requirement for corticosteroid or other therapy (Campbell 1993, Harvey 1993). Their effect may be partly attributable to the role of PUFA in the production of eicosanoids, which are important mediators of inflammation in the dog.

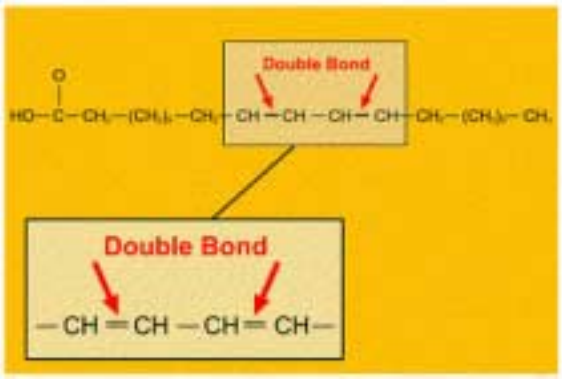


Diagram of a polyunsaturated fatty acid

It is thought that dogs with atopic dermatitis may have an impaired ability to convert linoleic acid to the longer chain omega-6 PUFA and their derivatives. Furthermore, manipulation of dietary PUFA may alter the balance of pro- and antiinflammatory eicosanoid production.

Supplementation with gamma-linolenic acid, in the form of evening primrose oil, and/or eicosapentaenoic acid, as marine fish oils, may help to attenuate the inflammatory process when administered at relatively high doses.

Zinc

Etiology

Absolute dietary deficiencies of zinc are considered rare in dogs, but a relative deficiency may occur when the availability of dietary zinc is reduced through nutrient interactions or where intestinal absorption of zinc is impaired through disease or genetic factors.

Intestinal absorption of zinc can be inhibited by:

- Excessive levels of dietary calcium, iron and copper, which compete with zinc for intestinal absorption sites
- High levels of dietary phytate, found in cereal-based diets, which chelates zinc
- Inherent defects of zinc absorption
- Prolonged enteritis or other malabsorption syndromes

Most cases of zinc-responsive dermatosis in dogs have been associated with the feeding of poor quality, cereal or soy-based dry foods, the effects of which may be exacerbated in some animals by other predisposing factors.

Lethal acrodermatitis is an inherited disease of English Bull Terriers in which a defect of zinc metabolism is thought to give rise to severe systemic, as well as cutaneous, signs that resemble experimental zinc deficiency. The condition is unresponsive to zinc supplementation and is usually fatal, with an average survival time of 7 months for affected puppies.

Pathophysiology

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

Mineral	Function
Zinc (Zn)	**An essential component to many enzyme systems, including those relating to protein and carbohydrate metabolism, and is essential for maintaining healthy coat and skin. Zinc is required by all animals, but the zinc requirements is particularly affected by other components of the diet. For example, a high dietary calcium content to a vegetable protein-based diet can dramatically increase the zinc requirement and this latter effect may be related to that reported for iron absorption.**

Clinically, two zinc-responsive dermatologic syndromes are recognized, although there is considerable overlap between the two:

- **Syndrome I** is associated with defective intestinal absorption of zinc and occurs predominantly in Alaskan Malamutes and Siberian Huskies, although other breeds may also be affected. The appearance of lesions frequently coincides with the onset of adulthood and during periods of stress and may be linked to higher metabolic requirements in these animals (Buffington 1987). Although dietary interactions may limit zinc absorption in some affected animals, the condition occurs in many cases despite feeding a nutritionally complete and balanced diet.
- **Syndrome II** is usually seen in rapidly growing puppies, particularly of the giant breeds, and may correspond with a high metabolic requirement for zinc in affected animals. The condition occurs when the diet is absolutely or relatively deficient in zinc, and is most likely to be seen when the diet is high in phytate or is oversupplemented with calcium.

Clinical Signs

Signs of zinc deficiency are confined mainly to the skin, but may be accompanied by:

- Growth and other abnormalities in young animals
- Depressed appetite due to a diminished sense of taste and smell
- Weight loss, impaired wound healing, conjunctivitis, and keratitis
- Generalized lymphadenopathy, particularly in young animals

Cutaneous signs are characterized by:

- Focal areas of erythema, alopecia, scale, and crust with underlying suppuration, which develop symmetrically particularly around the face, extremities, mucocutaneous junctions, and pressure points of the limbs
- Hyperkeratotic footpads with deep fissures
- Dull and harsh hair coat
- Secondary pyoderma

Dietary Management

Syndrome I

Oral zinc supplementation, together with dietary correction, where appropriate, brings rapid resolution of signs in most cases.

Supplementation with zinc sulfate (10 mg/kg/day) or zinc methionate (1.7 mg/kg/day) is usually adequate, but lifelong therapy is normally required and the dosage may be adjusted for long term maintenance (Muller *et al*. 1989).

Some cases, especially Siberian Huskies, do not respond to oral supplementation and may require the intravenous administration of zinc sulfate (10-15 mg/kg) at weekly intervals for 4 weeks, followed by maintenance injections every 1-6 months to prevent a relapse.

Syndrome II

Lesions resolve within 2-6 weeks following dietary correction, but the response time can be hastened by oral supplementation with zinc. In these cases, supplementation may be discontinued once the clinical signs are in remission.

Vitamin A

Etiology

Both deficiency and excess of vitamin A can give rise to a similar range of cutaneous signs. Vitamin A deficiency is rare in companion animals, and a toxicity state, with its accompanying skeletal changes, is more likely to occur. Although rare in dogs, hypervitaminosis A may occasionally be seen when large amounts of liver are fed or following prolonged oversupplementation of the diet with vitamin A or cod liver oil.

Pathophysiology and Clinical Signs

Vitamin A (retinol and its derivatives) has many physiologic functions and is involved in the regulation of cellular growth and differentiation. It is essential to maintain the integrity of epithelial tissues and is particularly important for the keratinization process. Both deficiency and excess of vitamin A can give rise to cutaneous lesions of:

- Hyperkeratinization and scaling

- Alopecia
- Poor hair coat
- Increased susceptibility to microbial infections

Hyperkeratinization of the sebaceous glands can result in occlusion of their ducts and the formation of firm, papular eruptions.

Dietary Management

Dietary supplementation may be required to correct vitamin A deficiency, but care should be exercised to avoid the risk of toxicity. In true deficiency syndromes, vitamin A therapy should not exceed 400 IU/kg/day orally for 10 days or a single injection of 6000 IU/kg which need not repeated for over 2 months.

Therapeutic supplementation

Vitamin A-responsive dermatosis is a rare condition which is seen almost exclusively in Cocker Spaniels, even when fed an apparently nutritionally adequate diet. Affected animals exhibit a generalized defect in keratinization with

- Scaling
- Greasy skin
- Alopecia
- Pruritus and secondary pyoderma
- Characteristic hyperkeratotic plaques that project above the skin surface

The condition is refractory to other antiseborrheic treatment but responds slowly to oral supplementation with vitamin A (retinol) at 10,000 IU/day (Harvey 1993). Clinical improvement is observed within 5-8 weeks, although lifelong maintenance therapy is usually required. This dose is in excess of the normal dietary requirement for vitamin A in dogs, and it is important therefore that other causes of seborrhea are eliminated before therapy is initiated.

Vitamin E

Etiology and Pathophysiology

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

Clinical Signs

Although experimentally induced vitamin E deficiency may produce cutaneous signs, there is no record of naturally occurring vitamin E deficiency in dogs.

Therapeutic Supplementation

Supraphysiologic doses of vitamin E have been used in the treatment of canine discoid lupus erythematosus and primary acanthosis nigricans. Vitamin E may exert an antiinflammatory effect that may be related to stabilization of membranes against immune-mediated and toxic free radical damage.

The suggested therapeutic dosage is 200-400 mg vitamin E given twice daily. A clinical response may not be evident for 30-60 days after the initiation of treatment.

B-Complex Vitamins

Etiology and Pathophysiology

The B-complex vitamins are involved as cofactors in many metabolic functions, especially energy metabolism and synthetic pathways. Being water soluble, they are not stored in the body, but the animal's daily requirements can normally be met from a combination of dietary sources and intestinal microbial biosynthesis. However, deficiencies may occur following prolonged oral antibiosis or anorexia or when water loss is increased as in polyuric conditions or enteritis.

Occasionally, deficiencies of individual B-complex vitamins arise as a result of interaction with other dietary components.

Clinical Signs

In general, skin lesions associated with deficiencies of B-complex vitamins include dry, flaky seborrhea and alopecia.

Biotin deficiency produces a characteristic alopecia around the face and eyes with crusting in severe cases. This condition may occur after feeding large amounts of raw egg whites which contain avidin, a protein that binds biotin and prevents its gastrointestinal absorption.

Riboflavin deficiency produces cheilosis in addition to seborrhea but will not occur if the diet contains meat or dairy products.

Niacin is synthesized from tryptophan and a deficiency is only possible when the diet is low in animal protein and high in corn or other cereals which are a poor source of tryptophan. A deficiency results in pellagra (humans) or “black tongue” (dogs), with ulceration of mucous membranes, diarrhea, and emaciation and, occasionally, a pruritic dermatitis of the hind legs and ventral abdomen.

Hypersalivation due to niacin deficiency

Pyridoxine deficiency may cause a dull, waxy, unkempt coat with fine scales and patchy alopecia but has only been reproduced in experimental studies.

Dietary Management

Treatment of B-vitamin deficiency involves dietary correction, where appropriate, and supplementation with the entire vitamin B group to compensate for reduced intake or increased losses. Oral supplementation with brewer's yeast and/or parenteral administration of B-vitamins is usually effective.

Biotin supplementation has also been used with limited success in the treatment of various dermatoses and disorders of keratinization.

SUMMARY

The skin has a high physiologic demand for protein and other nutrients, making it particularly sensitive to alterations in its own nutrient supply. Nutritional factors have a significant role in the maintenance of skin and coat condition in dogs and may contribute to the etiology and management of skin disease in the areas of nutrient deficiency or imbalance, therapeutic supplementation and dietary sensitivity.

Nutritional deficiencies are rare when a complete and balanced diet is fed, but may occur when the diet is poorly formulated or stored, when the animal's intake is reduced, or when the animal is unable to digest, absorb, or utilize the nutrient as a result of disease or genetic factors. Cutaneous signs are not usually apparent until after several months of feeding the deficient diet and are generally characterized by the development of seborrheic skin changes. Typical signs include excessive scale, erythema, alopecia, or poor hair growth and greasy skin, which is often accompanied by secondary bacterial infection and pruritus.

Deficiencies of protein, essential fatty acids, zinc, and vitamin A appear to be the most important causes of nutritional dermatoses in the dog, but others include vitamin E as well as biotin and other B-complex vitamins. Secondary deficiencies of certain nutrients, such as zinc, vitamin E, fatty acids, and biotin, may occur due to their interaction with other nutrients in the diet or as a result of impaired nutrient absorption or metabolism by the individual animal. It is usually more

effective to feed a high quality, balanced commercial diet than to try to improve a poorly formulated diet that may be deficient in many aspects.

Supplementation with specific nutrients in supraphysiologic doses may be beneficial in the management of certain nutrient-responsive dermatoses. In such cases, their action may be considered pharmacologic rather than a correction of a simple deficiency. Examples of nutrients used in dogs for their therapeutic effect include essential fatty acids, zinc, vitamin A, vitamin E and biotin.

Adverse reactions to food are rare in the dog, but may also result in cutaneous disease. In most cases, dietary sensitivity manifests as a pruritic skin disorder accompanied by varying degrees of associated self-trauma. Diagnosis and management of the condition involve identification of the offending ingredient(s) and its removal from the animal's diet.

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