**Diagnosing and Treating Canine Allergy**

**Canine Atopy** (environmental, pollen allergies)

**Features**

Canine atopy is a hypersensitivity reaction to inhaled (possibly a historic theory) or cutaneously absorbed environmental antigens (allergens) in genetically predisposed individuals. It is common in dogs, with age of onset ranging from 6 months to 6 years. However, in most atopic dogs, symptoms first appear at between 1 and 3 years of age.

Symptoms begin as skin erythema and pruritus (licking, chewing, scratching, rubbing), which may be seasonal or nonseasonal, depending on the offending allergen. The distribution of the pruritus usually involves the **feet**, flanks, groin, axillae, face, and ears. Self-trauma often results in secondary skin lesions, including salivary staining, alopecia, excoriations, scales, crusts, hyperpigmentation, and lichenification. Secondary pyoderma, *Malassezia* dermatitis, and otitis externa are common. Chronic acral lick dermatitis, recurrent pyotraumatic dermatitis, conjunctivitis, hyperhidrosis (sweating), and, rarely, allergic bronchitis or rhinitis may be seen.

**Top Differentials**

Differentials include food allergy, scabies, *Malassezia* dermatitis, bacterial pyoderma, as well as other hypersensitivities (flea bite, contact), parasites (cheyletiellosis, pediculosis), and folliculitis (dermatophyte, *Demodex*).

**Diagnosis**

**1.** Seasonal foot-licking is the most unique and typical symptom of atopy. If year-round allergens (house dust mites) are causing the allergy, the foot-licking may be nonseasonal.

**2.** Allergy testing (intradermal, serologic): allergy testing can be highly variable according to the method used. Positive reactions to grass, weed, tree, mold, insect, dander, or indoor environmental allergens are seen. False-negative and false-positive reactions may occur.

**3.** Dermatohistopathology (nondiagnostic): superficial perivascular dermatitis that may be spongiotic or hyperplastic. Inflammatory cells are predominantly lymphocytes and histocytes. Eosinophils are uncommon. Neutrophils or plasma cells suggest secondary infection.

**Treatment and Prognosis**

1. **Infection Prevention:**
	1. Any secondary pyoderma, otitis externa, and *Malassezia* dermatitis should be treated with appropriate therapies. Controlling and preventing secondary infection is an essential component of managing atopic dogs. Bathing every 3 – 7 days and treating the ears after every bath helps wash off pollens and disinfect the skin and ear canals, preventing the secondary infections from recurring.
2. **Symptomatic Therapy (itch control):**

**a** An integrated flea control program should be instituted to prevent flea bites from aggravating the pruritus.

 **b** Topical therapy with antimicrobial shampoos and anti-itch conditioners, and sprays (i.e., those containing oatmeal, pramoxine, antihistamines, or glucocorticoids) applied every 2 to 7 days or as needed may help reduce clinical symptoms.

**C** Systemic antihistamine therapy reduces clinical symptoms in many cases (Table 7-1). Antihistamines can be used alone or in combination with glucocorticoids or essential fatty acids for a synergistic effect. One- to two-week long therapeutic trials with different antihistamines may be required to determine which is most effective.

**D** Oral essential fatty acid supplements (180mg EPA/10 lb) help control pruritus in 20% to 50% of cases, but 8 to 12 weeks of therapy may be needed before beneficial effects are seen. Also, a synergistic effect is often noted when essential fatty acid supplements are administered in combination with glucocorticoids or antihistamines.

**E** Dextromethorphan, an opioid antagonist, may also be a useful adjunct in managing the licking, chewing, and biting behaviors associated with allergic dermatitis in dogs. Dextromethorphan 2mg/kg PO should be administered every 12 hours. A beneficial effect should be seen within 2 weeks.

F Systemic glucocorticoid therapy is often effective (75%) in controlling pruritus but almost always result in adverse effects ranging to mild (PU/PD) to severe (immuno-dysfunction, Demodicosis, and calcinosis cutis). It is a therapeutic option if the allergy season is very short but may result in unacceptable adverse effects, especially if used over the long term.

 1. Potent, long acting injectable steroids are contraindicated for the treatment of allergies due to their comparatively short anti-inflammatory benefits ( 3 weeks) relative to the prolonged metabolic and immuno-depressive effects (6-10 weeks).

2. Injectable short acting steroids (dexamehtasone Sodium Phosphate, 0.5 – 1 mg/kg or prednisilone acetate 0.1mg – 1 mg/kg) are effective at providing relief and may last 2 to 3 weeks if there are no concurrent secondary infection. This treatment option allows the clinician to more closely control and monitor the patients steroids use compared to oral treatments that are administered by the owner.

3. Temaril-P (trimeprizine and prednisilone combination) is a unique drug that provides significant antipruritic effects at a relatively lower dose of the prednisilone. One tablet should be administered per 10 to 20 kg every 24 to 48 hours. The dosage should be tapered to the lowest possible dose and frequency.

4. Prednisone 0.25 to 1mg/kg (or methylprednisolone 0.2-0.8mg/kg) PO should be administered every 24 to 48 hours for 3 to 7 days. The dosage should be tapered to the lowest possible dose and frequency.

5. All dogs treated with long-term steroids (more than 3 months) should be frequently monitored for liver disease and UTI.

**8. Allergy Treatment (immune-modulation)**

**a.** Exposure to offending allergens should be reduced, if possible, by their removal from the environment. High-efficiency particulate (HEPA) air and charcoal filters should be used to reduce pollens, molds, and dust in the home. For house dust mite–sensitive dogs, household treatments for carpets, mattresses, and upholstery with the acaricide benzyl benzoate once a month for approximately 3 months, then every 3 months thereafter, may effectively eliminate house dust mites from the environment. Old dog beds should be discarded as these accumulate house dust mite antigens. Dehumidifying the house to below 40% relative humidity decreases house dust mite, mold, and flea antigen loads. To achieve this, high-efficiency dehumidifiers that are capable of pulling several liters of water from the air per day are required.

**b.** Cyclosporine (Atopica) helps control pruritus in 75% of atopic dogs. A dose of 5mg/kg PO should be administered every 24 hours until beneficial effects are seen (approximately 4‑6 weeks). Then, dosage frequency should be tapered down to every 48 to 72 hours. For long-term control, approximately 25% of dogs require daily dosing, 50% can be controlled with every-other-day dosing, and approximately 25% can be controlled with twice-weekly dosing. Glucocorticoids can be used initially to speed response. As of this writing, there are no statistically significant increases in tumor risk or severe infection resulting from the immune effects of cyclosporine.

**c.** With immunotherapy (allergy vaccine), 60% to 75% of atopic dogs show good (some medical therapy still needed) to excellent (no other therapy needed) response. Clinical improvement is usually noted within 3 to 5 months of initiation of immunotherapy, but it can take up to 1 year in some dogs.

11. The prognosis is good, although lifelong therapy for control is needed in most dogs. Relapses (pruritic flare-ups with/without secondary infections) are common, so individualized treatment adjustments to meet patient needs may be required periodically. In dogs that become poorly controlled, one should rule out secondary infection (e.g., that caused by bacteria or *Malassezia*); sarcoptic mange; demodicosis; concurrent food, flea bite, and recently acquired hypersensitivity to additional environmental allergens. Because a strong genetic component is present, the breeding of any male or female dog with clinical signs of atopic dermatitis should be discouraged.

Box

Author’s Note

Our profession has excelled at reducing the use of steroids for arthritis; however, we have failed to make similar achievements for allergic disease including atopy. Since both disease have many similarities, including chronicity and multimodal therapeutic options, our goal should be to minimize the use of steroids for allergic diseases through the use of alternative, safer treatment options. To achieve best medicine, the frequency of steroid use should be similar for patients with arthritis and allergy.

The use of long-acting, injectable steroids should be stopped due to the profound impact on the metabolic and immune systems as well as the growing concern of legal liability for the practitioner.

Author’s Note

The only real, long-term options for treating the allergic immune response to environmental allergens are avoidance, allergy vaccine, or cyclosporine (Atopica). Based on typical cgeneral practice demographics, every full time small animal veterinarian should have approximately 20-30 patients who are no longer controlled with symptomatic therapy and need more aggressive treatment (allergy vaccine or cyclosporine).

**Canine Food Hypersensitivity**

**Features**

Canine food hypersensitivity is an adverse reaction to a food or food additive. It can occur at any age, from recently weaned puppies to elderly dogs that have been eating the same dog food for years. Approximately 30% of dogs diagnosed with food allergy are younger than 1 year of age. It is common in dogs.

Canine food hypersensitivity is characterized by nonseasonal pruritus that may or may not respond to steroid therapy. The pruritus may be regional or generalized and usually involves the ears, feet, inguinal or axillary areas, face, neck, and **perineum**. Affected skin is often erythematous, and a papular rash may be present. Self-trauma–induced lesions include alopecia, excoriations, scales, crusts, hyperpigmentation, and lichenification. Secondary superficial pyoderma, *Malassezia* dermatitis, and otitis externa are common. Other symptoms that may be seen are acral lick dermatitis, chronic seborrhea, and recurring pyotraumatic dermatitis. Some dogs are minimally pruritic, with the only symptom being recurrent infection with pyoderma, *Malassezia* dermatitis, or otitis. In these cases, the pruritus is present only when secondary infections are left untreated. Occasionally, urticaria or angioedema may occur. Concurrent gastrointestinal signs (e.g., frequent bowel movements, vomiting, diarrhea, flatulence) are reported in 20%-30% of cases.

**Top Differentials**

Differentials include atopy, scabies, *Malassezia* dermatitis, bacterial pyoderma, as well as other hypersensitivities (flea bite, contact), parasites (cheyletiellosis, pediculosis), and folliculitis (dermatophyte, *Demodex*).

**Diagnosis**

**1.** Perianal dermatitis with or without recurrent otitis is the most common and unique feature of food allergy. However, food allergy can manifest in many patterns and should be suspected for atypical pruritic patient including cases of recurrent infections without pruritus.

**2.** Dermatohistopathology (nondiagnostic): varying degrees of superficial perivascular dermatitis. Mononuclear cells or neutrophils may predominate. Eosinophils may be more numerous than in atopy

**3.** Food allergy testing (intradermal, serologic)(nondiagnostic): not recommended because test results are unreliable. Some dogs will have positive reactions to storage mite antigens, which may be clinically relevant, or they may be caused by cross-reactivity with other insects. Storage mites are ubiquitous, and their clinical significance is currently unknown.

**4.** Response to hypoallergenic diet trial: symptoms improve within 10 to 12 weeks of initiation of a strict home-cooked or commercially prepared restricted diet (one protein and one carbohydrate source). The hypoallergenic diet should not contain food ingredients previously administered in dog food, treats, or table scraps, nor should flavored heartworm preventative, flavored medications, nutritional supplements, or chewable treats (i.e., pig ears, cow hooves, rawhide, dog biscuits, table food such as cheese or peanut butter to hide pills in) be administered during the hypoallergenic diet trial. Beef and dairy are the most common food allergens in dogs and avoiding these alone may result in clinical improvement. Other common food allergies include chicken, eggs, soy, corn, and wheat.

**5.** Provocative challenge: recurrence of symptoms within hours to days of reintroduction of suspect allergen into the diet.

**Treatment and Prognosis**

1. **Infection Prevention:**
	1. Any secondary pyoderma, otitis externa, and *Malassezia* dermatitis should be treated with appropriate therapies. Controlling and preventing secondary infection is an essential component of managing atopic dogs. Bathing every 3 – 7 days and treating the ears after every bath helps wash off pollens and disinfect the skin and ear canals, preventing the secondary infections from recurring.
2. **Symptomatic Therapy (itch control) is variably effective for food allergy:**

**a** An integrated flea control program should be instituted to prevent flea bites from aggravating the pruritus.

 **b** Topical therapy with antimicrobial shampoos and anti-itch conditioners, and sprays (i.e., those containing oatmeal, pramoxine, antihistamines, or glucocorticoids) applied every 2 to 7 days or as needed may help reduce clinical symptoms.

**C** Systemic antihistamine therapy reduces clinical symptoms in many cases (Table 7-1). One- to two-week long therapeutic trials with different antihistamines may be required to determine which is most effective.

**D** Oral essential fatty acid supplements (180mg EPA/10 lb) help control pruritus in 20% to 50% of cases, but 8 to 12 weeks of therapy may be needed before beneficial effects are seen. Also, a synergistic effect is often noted when essential fatty acid supplements are administered in combination with glucocorticoids or antihistamines.

**E** Dextromethorphan, an opioid antagonist, may also be a useful adjunct in managing the licking, chewing, and biting behaviors associated with allergic dermatitis in dogs. Dextromethorphan 2mg/kg PO should be administered every 12 hours. A beneficial effect should be seen within 2 weeks.

F Systemic glucocorticoid therapy is only variably effective (unpredictable minimal to good response) in controlling pruritus cause by the food allergy; but almost always result in adverse effects ranging to mild (PU/PD) to severe (immuno-dysfunction, Demodicosis, and calcinosis cutis). (see Atopy section)

 1. Potent, long acting injectable steroids are contraindicated for the treatment of allergies due to their comparatively short anti-inflammatory benefits ( 3 weeks) relative to the prolonged metabolic and immuno-depressive effects (6-10 weeks).

2. Injectable short acting steroids (dexamehtasone Sodium Phosphate, 0.5 – 1 mg/kg or prednisilone acetate 0.1mg – 1 mg/kg) are effective at providing relief and may last 2 to 3 weeks if there are no concurrent secondary infection. This treatment option allows the clinician to more closely control and monitor the patient’s steroids use compared to oral treatments that are administered by the owner.

3. All dogs treated with long-term steroids (more than 3 months) should be frequently monitored for liver disease and UTI.

**8. Food Allergy Treatment**

**a.** Offending dietary allergen(s) should be avoided. A balanced home-cooked diet or a commercial hypoallergenic diet should be provided.

**b.** To identify offending substances to be avoided (challenge phase after food allergy has been confirmed with the dietary trial) one new food item should be added to the hypoallergenic diet every 2 to 4 weeks. If the item is allergenic, clinical symptoms will recur within 7 to 10 days. *Note*: Some dogs (approximately 20%) should be fed home-cooked diets to remain symptom-free. For these dogs, commercial hypoallergenic diets are ineffective, presumably because their hypersensitivity relates to a food preservative or dye.

**c.** Ancedotal reports suggest that higher doses (10mg/kg) of cyclosporine (Atopica) may ne beneficial in reducing the allergic immune response and symptoms of food allergy.

**8.** The prognosis is good. In dogs that are poorly controlled, owner noncompliance should be ruled out, along with development of hypersensitivity to an ingredient in the hypoallergenic diet, secondary infection (caused by bacteria, *Malassezia*, dermatophyte), scabies, demodicosis, atopy, flea allergy dermatitis, and contact hypersensitivity.

Author’s Note

Due to recent food industry changes, there has been an explosion of products available through prescription or over-the-counts and the listing is beyond the scope of this text.

Many of the over-the-counter diets are sufficiently restricted and of high enough quality to produce clinical benefit when a food allergic patient restricted to one of the nonBeef and nondairy products.

Food allergy is responsible for most of the very unusual clinical symptom patterns in dogs with recurrent infections (with or without pruritus).

Poor owner compliance should be expected making the long-term management of food allergic patients difficult and frustrating; repeated lapses in diet result in flare-ups in the pruritus and secondary infections.

Author’s Note

The use of long-acting, injectable steroids should be stopped due to the profound impact on the metabolic and immune systems as well as the growing concern of legal liability for the practitioner.

**Flea Allergy Dermatitis** (flea bite hypersensitivity)

**Features**

Flea allergy dermatitis is a common skin disease in dogs and cats sensitized to flea salivia proteins through repeated and intermittent flea bites. Symptoms are usually seasonal (warm weather months and in the fall) in temperate zones and often nonseasonal in subtropical and tropical areas. Fall is often the most severe season relating to when the first cold snap occurs.

**Dogs**

The distribution typically involves the caudodorsal lumbosacral area, dorsal tail head, caudomedial thighs, abdomen, and flanks. Lesions include pruritic, papular, crusting eruptions with secondary erythema, seborrhea, alopecia, excoriations, pyoderma, hyperpigmentation, and lichenification.

**Cats**

Cats do not have a pattern unique to flea allergy dermatitis. Patients commonly present with pruritic miliary dermatitis with secondary excoriations, crusting, and alopecia of the neck, dorsal lumbosacral area, caudomedial thighs, and ventral abdomen. Other symptoms include symmetrical alopecia secondary to excessive grooming and eosinophilic granuloma complex lesions.

**Top Differentials**

Differentials include atopy, food hypersensitivity, other ectoparasites (scabies, cheyletiellosis, pediculosis, demodicosis), superficial pyoderma, dermatophytosis, demodicosis, and *Malassezia* dermatitis.

**Diagnosis**

**1.** Lumbar dermatitis in the dog is the most consistent and unique feature of flea allergy dermatitis. In cats, flea allergy should be highly suspected in any cat with skin disease.

**2.** Visualization of fleas or flea excreta on body: may be difficult on flea-allergic animals as flea-allergic animals are very effective at removing fleas through grooming

**3.** Allergy testing (intradermal, serologic): positive skin test reaction to flea antigen or positive serum immunoglobulin (Ig)E antiflea antibody titer is highly suggestive, but false-negative results are possible

**4.** Dermatohistopathology (nondiagnostic): varying degrees of superficial or deep perivascular to interstitial dermatitis, with eosinophils often predominating

**5.** Response to aggressive flea control (nitenpyram administered every other day for 1 month): symptoms resolve

**Treatment and Prognosis**

**1.** Integrated flea management program (Insect growth regulator combined with an adulticide combined with environmental treatments) is essential due to the progressive tolerance of the flea to available adulticides. With time, specific active ingredients typically loose efficacy due to the chronic exposure and genetic drift of the flea.

**2.** Topical or systemic insect growth regulators (lufenuron, piriproxyfen, methoprene) may be effective alone or used in combination with adulticidal therapy.

3. Affected and all in-contact dogs and cats should be treated with adulticidal flea sprays, spot-on solutions, orals, or dips every 7 to 30 days, as instructed on the label. Products that contain spinosid , imidocloprid, selamectin, or fipronil are especially effective when used topically every 2 to 4 weeks. In heavily flea-infested environments, fleas may continue to be found on animals for several months in spite of topical flea control. In these cases, affected animals should also be administered nitenpyram at a minimum dose of 1mg/kg PO every 24 to 48 hours for 2 to 4 weeks, or until fleas are no longer seen. The environment should be treated (see number 5 below).

**4.** Flea-allergic animals should be treated prophylactically with nitenpyram, minimum dose 1mg/kg PO, on any day that an encounter is planned with other potentially flea-infested animals (e.g., a visit to the groomer, veterinary hospital, park, another household with animals). No more than one treatment with nitenpyram should be administered per day.

**5.** In heavily flea-infested environments, areas where pets spend the most time should be treated. Indoor premises should be treated with an insecticide and an insect growth regulator (e.g., methoprene, piriproxyfen). The outdoor environment should be treated with insecticidal or biologic products designed for such use.

**6.** Flea control therapy should be continued from spring until first snowfall in temperate areas and year-round in warm climates. Year-round flea infestations can be perpetuated indoors and on wildlife despite extreme cold outdoors.

**7. Symptomatic Therapy (itch control):**

**a** Topical therapy with antimicrobial shampoos and anti-itch conditioners, and sprays (i.e., those containing oatmeal, pramoxine, antihistamines, or glucocorticoids) applied every 2 to 7 days or as needed may help reduce clinical symptoms.

**b** Systemic antihistamine therapy reduces clinical symptoms in many cases (Table 7-1).

c Systemic glucocorticoid therapy is often effective (75%) in controlling pruritus but almost always result in adverse effects ranging to mild (PU/PD) to severe (immuno-dysfunction, Demodicosis, and calcinosis cutis). It is a therapeutic option if the allergy season is very short but may result in unacceptable adverse effects, especially if used over the long term.

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3. Temaril-P (trimeprizine and prednisilone combination) is a unique drug that provides significant antipruritic effects at a relatively lower dose of the prednisilone. One tablet should be administered per 10 to 20 kg every 24 to 48 hours. The dosage should be tapered to the lowest possible dose and frequency.

4. Prednisone 0.25 to 1mg/kg (or methylprednisolone 0.2-0.8mg/kg) PO should be administered every 24 to 48 hours for 3 to 7 days. The dosage should be tapered to the lowest possible dose and frequency.

5. All dogs treated with long-term steroids (more than 3 months) should be frequently monitored for liver disease and UTI.

**8.** The prognosis is good if strict flea control is practiced. Fleas may infest other in-contact animals and humans. They may carry blood-borne diseases in a manner similar to ticks.

Author’s note

The use of long-acting, injectable steroids should be stopped due to the profound impact on the metabolic and immune systems as well as the growing concern of legal liability for the practitioner.

Any dog with lumbar dermatitis or any cat with skin disease should be highly suspected of having flea allergy dermatitis even if the patient has been treated with seemingly good flea control therapies.

A nitenpyram trial (every other day for 1 month) is the most efficient and cost effective way to convince the owner and yourself of the role of flea allergy in a pruritic patient.