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:
   a. 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 (dexamethasone 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. Temaril-P (trimeprazine 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 general
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

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

**Treatment and Prognosis**

1. **Infection Prevention:**
   a. 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 (dexamethasone 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. Anecdotal reports suggest that higher doses (10mg/kg) of cyclosporine (Atopica) may be 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.

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

**Features**

Flea allergy dermatitis is a common skin disease in dogs and cats sensitized to flea saliva proteins through repeated and intermittent flea bites. Symptoms are usually seasonal (warm weather months and in the fall) in temperate flea bites 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 lose 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.
      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 (dexamethasone 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. 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.
WHAT IS MAKING MY DOG SO ITCHY?

Evaluation Form
A thorough history can help us find the source of your dog’s itching more quickly. Please answer the following questions to help guide the diagnostic process.

Date ___________________ Pet owner name ___________________
Name of dog __________ Age ______ Breed ___________ Weight ___________

PHYSICAL EVALUATION
Please check any that describe your dog and circle problem areas on the drawing.

- Hair loss
- Foul odor
- Inflammation or redness
- Itching/Scratching
- Otitis (ear infections)
- Licking/Chewing
- Skin lesions (sores)
- Changes in skin (reddish brown stains, discolorations and/or areas that are thick and leathery)
- Other ________________

- Has your dog ever had ear problems? □ Yes □ No
- Does your dog have any chronic gastrointestinal signs like diarrhea or vomiting? □ Yes □ No

SEVERITY EVALUATION On a scale of 0 to 10 rank the severity of your dog’s symptoms.

SEVERITY OF CONDITION OVERALL

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SEVERITY OF SKIN LESIONS

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SEVERITY OF SCRATCHING/LICKING/CHewing

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ONSET AND SEASONALITY EVALUATION

- Is this the first time your dog has experienced these symptoms? □ Yes □ No
  - If no, at what age did the symptoms first occur? □ <1 yr □ 1-3 yrs □ 4-7 yrs □ 7+ yrs □ Yes □ No
  - If no, has it occurred around the same time of year each time? □ Yes □ No
  - If no, approximate time of year symptoms occur. ______________________
- How long have the current symptoms been going on? ______________________
- Did the itch start gradually and over time become worse? □ Yes □ No
- Did the itch come on suddenly without warning? □ Yes □ No
- Was there a “rash” first or itching first? Or simultaneous? □ Rash first □ Itch first □ Simultaneous

PARASITE CONTROL

- Is your dog on a flea/heartworm preventative? □ Yes □ No
  - If yes, what product(s)? ______________________
  - What months do you administer the preventative? ______________________
- When was the last time you administered the parasite control? ______________________
Infectious Disease Diagnosis and Management

Diagnostic Testing
The dermatologic diagnostic minimum database includes skin scrapes, otic swabs, and cutaneous cytology. The goal should be to identify all secondary infections (e.g., pyoderma, demodicosis, dermatophytosis, otitis, *Malassezia* dermatitis, infectious pododermatitis), then formulate a diagnostic plan for identifying and controlling the underlying/primary disease (i.e., allergies, endocrinopathies, keratinization defects, and autoimmune skin diseases).

Ask yourself, “What are the infections” for every dermatitis cases every time you evaluate the patient.

Using diarrhea and the microscopic fecal exam as a comparison works well since both skin cytology and fecal exams involve the use of a microscope, can easily identify the type of infection, and can be performed by trained technical staff. So why does your clinic perform fecal exams? When is a fecal exam performed (before the doctor’s examination)? Who performed the fecal? Does the clinic charge for the fecal exam? The answers to these questions should be the same for skin cytology (skin scrapings, impression smears, tape preps, and otic swabs).

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The practical solution and the best method to answer the question, “What are the infections?” is to implement a minimum data base “infection screening” procedure performed by the technician before the veterinarian examines the patient. Every dermatology patient should have an otic cytology, skin cytology (either an impression smear or tape prep), and a skin scrape performed every time the patient is examined (initially and at every recheck visit). This 3 Slide Technique™ can easily be performed and interpreted by a technician prior to the doctor’s evaluation; exactly how diarrhea cases and fecal exams are handled in most clinics.

Skin Scrapes (Slide #1 in the 3 Slide Technique)

Skin scrapes are the most common dermatologic diagnostic tests. These relatively simple and quick tests can be used to identify many types of parasitic infections. Although they are not always diagnostic, their relative ease and low cost make them essential tests in a dermatologic diagnostic minimum database.

Many practitioners reuse scalpel blades when performing skin scrapes; however, this practice should be stopped because of increased awareness of transmittable diseases (e.g., Bartonella, Rickettsia, feline leukemia virus [FeLV], feline immunodeficiency virus [FIV], herpes, papillomavirus).

Procedure

Deep Skin Scrapes (for Demodex spp except D. gatoi). A dulled scalpel blade is held perpendicular to the skin and is used with moderate pressure to scrape in the direction of hair growth. If the area is covered with hair (usually, alopecic areas caused by folliculitis are selected), it may be necessary to clip a small window to access the skin. After several scrapes, the skin should become pink, with the capillaries becoming visible and oozing blood. This ensures that the material collected comes from deep enough within the skin to allow the collection of follicular Demodex mites. Most people also squeeze (pinch) the skin to express the mites from deep within the follicles into a more superficial area, so that they may be more easily collected. If the scraping fails to provide a small amount of blood, then the mites may have been left in the follicle, resulting in a false-negative finding. In some situations (with Shar peis or deep inflammation with scarring), it may be impossible to scrape deeply enough to harvest Demodex mites. These cases are few in number but require biopsy for identification of the mites within the hair follicles. Hair-plucks from an area of lesional skin may be used to help find mites, but the accuracy of this technique compared with skin scrapes is unknown.

Regardless of the collection technique used, the entire slide should be searched for mites with the use of low power (usually a 10X objective). A search of the entire slide ensures that if only one or two mites are present (as is typical of scabies infection), the user will likely find them. It may be helpful to lower the microscope condenser; this provides greater contrast to the mites, thereby enhancing their visibility. (One must be sure to raise the condenser before looking for cells or bacteria on stained slides.)
There is no excuse for mistreating a patient who has demodicosis. Lesions caused by demodicosis can look identical to folliculitis lesion caused by bacterial pyoderma and dermatophytosis. Clinical appearance is not an acceptable method to rule-in or rule-out demodicosis. By having the technicians perform a skin scrape as part of the infection screen, 3 Slide Technique™, demodicosis can easily and accurately be identified and treated.

Cutaneous Cytology (Slide #2 in the 3 Slide Technique)

Cutaneous cytology is the second most frequently employed dermatologic diagnostic technique. Its purpose is to help the practitioner to identify bacterial or fungal organisms (yeast) and assess the infiltrating cell types, neoplastic cells, or acantholytic cells (typical of pemphigus complex).

The infections are always secondary to a primary disease; however, all too often, the patient is not evaluated or treated for the primary disease. This is due to 3 predominant factors: treating only the secondary infections over and over, the confusing nature of allergy, and access to cheap steroids which have delayed repercussions.

Superficial Pyoderma (superficial bacterial folliculitis)

Features

Superficial pyoderma is a superficial bacterial infection involving hair follicles and the adjacent epidermis. The infection is almost always secondary to an underlying cause; allergies and endocrine disease are the most common causes (Box 3-3). Superficial pyoderma is one of the most common skin diseases in dogs but rare in cats.

Superficial pyoderma is characterized by focal, multifocal, or generalized areas of papules, pustules, crusts, and scales, epidermal collarettes, or circumscribed areas of erythema and alopecia that may have hyperpigmented centers. Short-coated dogs often present with a “moth-eaten” patchy alopecia, small tufts of hair that stand up, or reddish brown discoloration of white hairs. In long-coated dogs, symptoms can be insidious and may include a dull, lusterless hair coat, scales, and excessive shedding. In both short- and long-coated breeds, primary skin lesions are often obscured by remaining hairs but can be readily appreciated if an affected area is clipped. Pruritus is variable, ranging from none to intense levels. Bacterial infections secondary to endocrine disease may cause pruritus, thereby mimicking allergic skin disease.

*Staphylococcus pseudintermedius* (previously *Staphylococcus intermedius*) is the most common bacterium isolated from canine pyoderma and is usually limited to dogs. *Staphylococcus schleiferi* is a bacterial species in dogs and humans that is emerging as a common canine isolate in patients with chronic infections and previous antibiotic exposure. Both *Staphylococcus pseudintermedius* and *Staphylococcus schleiferi* may develop methicillin-resistance especially if subtherapeutic doses of antibiotics or fluoroquinolone antibiotics have been previously used in the patient. Additionally, methicillin-resistant *Staphylococcus aureus* (human MRSA) is becoming more common among veterinary species. All three *Staphylococcus* may be zoonotic, moving from humans to canines or from canine to human; immunosuppressed individuals are at most risk.

Dr. Keith A Hnilica, DVM, MS, MBA, DACVD
itchnot.com
Causes of Secondary Superficial and Deep Pyoderma

- Demodicosis, scabies, Pelodera
- Hypersensitivity (e.g., atopy, food, flea bite)
- Endocrinopathy (e.g., hypothyroidism, hyperadrenocorticism, sex hormone imbalance, alopecia X)
- Immunosuppressive therapy (e.g., glucocorticoids, progestational compounds, cytotoxic drugs)
- Autoimmune and immune-mediated disorders
- Trauma or bite wound
- Foreign body
- Poor nutrition

Treatment and Prognosis

1. The underlying cause must be identified and controlled.
2. Systemic antibiotics (minimum 3-4 weeks) should be administered and continued 1 week beyond complete clinical and cytological resolution (see Box 3-1).
3. Concurrent bathing every 2 to 7 days with an antibacterial shampoo that contains chlorhexidine or benzoyl peroxide is helpful.
4. If lesions recur within 7 days of antibiotic discontinuation, the duration of therapy was inadequate and antibiotics should be reinstituted for a longer time period and better attempts to identify and control the underlying disease should occur.
5. If lesions do not completely resolve during antibiotic therapy or if there is no response to the antibiotics, antibiotic resistance should be assumes and a bacterial culture and sensitivity submitted.
6. If antibiotics resistance is suspected or confirmed, frequent bathing (up to daily) and the frequent application of topical chlorhexidine solutions combined with the simultaneous administration of two different class antibiotics at high doses seem to produce the best results. Monitoring the infection with cytology and cultures with antibiotic sensitivities is important to determine when the treatments can be stopped. Premature discontinuation of therapy, not completely controlling the primary disease, and the use of fluoroquinolone antibiotics will likely perpetuate the resistant infection.
7. The prognosis is good if the underlying cause can be identified and corrected or controlled.

Author’s Note:
** Superficial Pyoderma is one of the most common skin diseases in dogs and almost always has an underlying cause (allergies or endocrine disease).
** Cefpodoxime, Ormetoprim/sulfadimethoxine (Primor), and Convenia provide the most consistent compliance which seem to help reduce the development of resistance when used at high doses.
** MRSA, MRSS, MRSI, and MRSP are becoming an emerging problem in some regions of the US.
   >> The most likely risk factors include previous exposure to fluoroquinolone antibiotics, sub-therapeutic antibiotic dosing, and concurrent steroid therapy.
   >> Daily baths and topical treatments can be very beneficial in the resolution of the infection.
Maximize the dose of antibiotics and consider using two antibiotics simultaneously to protect additional resistance from developing.

Practice good hygiene (HAND WASHING) to prevent zoonosis.

Consider screening dogs who visit the elderly or sick to prevent zoonosis. Cultures from the nose, lips, ears, axilla, and perianal areas are best for screening patients for MRS.

** Malasseziasis (Malassezia dermatitis) **

** Features **

*Malassezia pachydermatis* is a yeast that is normally found in low numbers in the external ear canals, in perioral areas, in perianal regions, and in moist skin folds. Skin disease occurs in dogs when a hypersensitivity reaction to the organisms develops, or when there is cutaneous overgrowth. In dogs, *Malassezia* overgrowth is almost always associated with an underlying cause, such as atopy, food allergy, endocrinopathy, keratinization disorder, metabolic disease, or prolonged therapy with corticosteroids. In cats, skin disease is caused by *Malassezia* overgrowth that may occur secondary to an underlying disease (e.g., feline immunodeficiency virus, diabetes mellitus or an internal malignancy). In particular, generalized *Malassezia* dermatitis may occur in cats with thymoma-associated dermatosis or paraneoplastic alopecia. Malasseziasis is common in dogs, especially among West Highland White terriers, Dachshunds, English setters, Basset hounds, American cocker spaniels, Shih tzus, Springer spaniels, and German shepherds. These breeds may be predisposed. Malasseziasis is rare in cats.

Moderate to severe pruritus is seen, with regional or generalized alopecia, excoriations, erythema, and seborrhea. With chronicity, affected skin may become lichenified, hyperpigmented, and hyperkeratotic (leathery or elephant-like skin). An unpleasant body odor is usually present. Lesions may involve the interdigital spaces, ventral neck, axillae, perineal region, or leg folds. Paronychia with dark brown nail bed discharge may be present. Concurrent yeast otitis externa is common.

** Diagnosis **

1. Rule out other differentials
2. Cytology (tape preparation, impression smear): yeast overgrowth is confirmed by the finding round-to-oval, budding yeasts per high power field (100x). In yeast hypersensitivity, organisms may be difficult to find

** Treatment and Prognosis **

1. Any underlying cause (allergies, endocrinopathy, keratinization defect) must be identified and corrected.
2. For mild cases, topical therapy alone is often effective. The patient should be bathed every 2 to 3 days with shampoo that contains 2% ketoconazole, 1% ketoconazole/2% chlorhexidine, 2% miconazole, 2% to 4% chlorhexidine, or 1% selenium sulfide (dogs only). Shampoos that have
two active ingredients provide better efficacy. Treatment should be continued until the lesions resolve and follow-up skin cytology reveals no organisms (approximately 4 weeks).

3. The treatment of choice for moderate to severe cases is ketoconazole (dogs) or fluconazole 10mg/kg PO with food every 24 hours. Treatment should be continued until lesions resolve and follow-up skin cytology reveals no organisms (approximately 4 weeks).

4. Alternatively, treatment with terbinafine 5-40mg/kg PO every 24 hours or itraconazole (Sporonox) 5-10mg/kg every 24 hours for 4 weeks may be effective.

5. Pulse therapy protocols have been published using several drugs and a variety of schedules; however, these often take longer to resolve the active infection.

5. The prognosis is good if the underlying cause can be identified and corrected. Otherwise, regular once- or twice-weekly antiyeast shampoo baths may be needed to prevent relapse. This disease is not considered contagious to other animals or to humans, except for immunocompromised individuals.

Authors’s Note:
** Yeast dermatitis is currently the most commonly missed diagnosis in US general practices. Any patient with leathery, elephant-skin like lesions on the ventrum should be suspected of having Malassezia dermatitis.
** Cutaneous cytology is not always successful for finding Malassezia organisms requiring the clinician to rely on clinical lesion patterns to make a tentative diagnosis.
** Yeast dermatitis is severely pruritic with owners reporting an itch level of 10 on a 0-10 visual analog scale.

**Canine Generalized Demodicosis**

**Features**

Canine generalized demodicosis may appear as a generalized skin disease that may have genetic tendencies and can be caused by three different species of demodectic mites: *D. canis*, *D. injai*, and an unnamed short-bodied *Demodex* mite. *D. canis*, a normal resident of the canine pilosebaceous unit (hair follicle, sebaceous duct, and sebaceous gland), is primarily transmitted from the mother to neonates during the first 2 to 3 days of nursing, but adult-to-adult transmission may rarely occur. *D. injai*, a recently described, large, long-bodied *Demodex* mite, is also found in the pilosebaceous unit, but its mode of transmission is unknown. Mode of transmission is also unknown for the short-bodied unnamed *Demodex* mite, which, unlike the other two species, lives in the stratum corneum. Depending on the dog’s age at onset, generalized demodicosis is classified as juvenile-onset or adult-onset. Both forms are common in dogs. Juvenile-onset generalized demodicosis may be caused by *D. canis* and the short-bodied unnamed *Demodex* mite. It occurs in young dogs, usually between 3 and 18 months of age, with highest incidence in medium-sized and large purebred dogs. Adult-onset generalized demodicosis can be caused by all three mite species and occurs in dogs older than 18 months of age, with highest incidence in middle-aged to older dogs that are immunocompromised because of an underlying condition such as endogenous or iatrogenic hyperadrenocorticism, hypothyroidism, immunosuppressive drug therapy, diabetes mellitus, or neoplasia. To date, only adult-onset disease has been reported with *D. injai*, with highest incidence noted in terriers.
Clinical signs of infestation with either *D. canis* or the unnamed *Demodex* mite are variable. Generalized demodicosis is defined as five or more focal lesions, or two or more body regions affected. Usually, patchy, regional, multifocal, or diffuse alopecia is observed with variable erythema, silvery grayish scaling, papules, or pruritus. Affected skin may become lichenified, hyperpigmented, pustular, eroded, crusted, or ulcerated from secondary superficial or deep pyoderma. Lesions can be anywhere on the body, including the feet. Pododemicosis is characterized by any combination of interdigital pruritus, pain, erythema, alopecia, hyperpigmentation, lichenification, scaling, swelling, crusts, pustules, bullae, and draining tracts. Peripheral lymphadenomegaly is common. Systemic signs (e.g., fever, depression, anorexia) may be seen if secondary bacterial sepsis develops.

*D. injai* infestations are typically characterized by a focal areas of greasy seborrhea (seborrhea oleosa), especially over the dorsum of the trunk. Other skin lesions may include alopecia, erythema, hyperpigmentation, and comedones. Small breeds and terriers seem to predisposed to Demodex *injai* infections.

**Diagnosis**

1. Microscopy (deep skin scrapes): many demodectic adults, nymphs, larvae, and ova are typically found with *D. canis* and the short-bodied, unnamed demodectic mite, although *D. canis* may be difficult to find in fibrotic lesions and in feet. With *D. injai*, mites may be low in number and difficult to find requiring skin biopsies.

**Treatment and Prognosis**

1. If adult-onset, any underlying conditions should be identified and corrected. All steroid containing therapies should be discontinued as steroid administration is the most common cause of adult onset demodicosis.
2. Intact dogs, especially females, should be neutered. Estrus or pregnancy may trigger relapse.
3. Any secondary pyoderma should be treated with appropriate long-term (minimum 3-4 weeks) systemic antibiotics that are continued at least 1 week beyond clinical resolution of the pyoderma.
4. Topical shampoo therapy using a 1-3% benzoyl peroxide shampoo every 3-7 days will help speed resolution and enhance the mitacidal treatments.
5. Effective Mitacidal therapies include the following:
   * Ivermectin 0.2-0.6mg/kg PO every 24 hours is often effective against generalized demodicosis. Initially, ivermectin 0.1mg/kg PO is administered on day 1, then 0.2mg/kg PO is administered on day 2, with oral daily increments of 0.1mg/kg until 0.2-0.6mg/kg/day is being administered, assuming that no signs of toxicity develop. The cure rate for 0.4mg/kg/day ivermectin is 85% to 90%.
   * Milbemycin oxime, 0.5 to 2mg/kg PO every 24 hours. The cure rate is 85% to 90%.
   * Doramectin is also reported to be effective against canine demodicosis at a dose of 0.6mg/kg SC once weekly. The cure rate is approximately 85%. Adverse effects are uncommon but include, as for ivermectin, dilated pupils, lethargy, blindness, and coma.
   * For dogs ≥20kg, the use of 9% amitraz collars may be effective. In small dogs, use of 9% amitraz collars alone may be as effective as ivermectin (0.6mg/kg/day PO).
* Topical application of Promeris (topical metaflumizone and amitraz solution) every two weeks has demonstrated good efficacy.

*Moxidectin has demonstrated variable efficacy when applied every 2-4 weeks.

Historical Treatment Include:

Traditional miticidal treatment entails the following:
- Total body hair coat clip if dog is medium- to long-haired
- Weekly bath with 2.5% to 3% benzoyl peroxide shampoo, followed by a total body application of 0.03% to 0.05% amitraz solution. The cure rate ranges from 50% to 86%.
- For demodectic pododermatitis, in addition to weekly amitraz dips, foot soaks in 0.125% amitraz solution should be performed every 1 to 3 days.

6. Regardless of the miticidal treatment chosen, therapy is administered over the long term (weeks to months). Treatments should be continued for at least 1 month beyond the time when the first follow-up skin scrapings becomes negative for mites (total of two negative skin scrapings).

7. The prognosis is good to fair. Relapses may occur, requiring periodic or lifelong treatment in some dogs. The use of glucocorticosteroids in any dog that has been diagnosed with demodicosis should be avoided. Because of its hereditary predisposition, neither female nor male dogs with juvenile-onset generalized demodicosis should be bred. *D canis* is not considered contagious to cats or to humans. It is transmitted from bitch to newborn puppies during the first 2 to 3 days of nursing, and possibly between adult dogs that are close cohabitants. The mode of transmission for *D injai* and the unnamed short-bodied *Demodex* mite is unknown.

Author’s Note:
Steroids are the most common cause of adult onset Demodicosis.
Products containing amitraz tend to be the most toxic usually due to the product vehicle.
Aggressive treatment should be tried for up to six months before giving up.
One of the most common causes of treatment failure is that the patient will look greatly improved before negative skin scrapes are achieved. Many owners will discontinue treatment prematurely resulting in relapse.
The average time to achieve clinical improvement is 4-6 weeks; the first negative skin scrape usually occurs around 6-8 weeks; and most patients need approximately 3 months of treatment to resolve the infection based on two negative skin scrapes at least 3 weeks apart.
1. **WHAT ARE THE INFECTIONS?**
   Perform 3-Slide Technique™ during the physical exam on multiple sites/lesions.
   - **Slide 1** Skin Scrape (hairplucks): Positive for / Negative
   - **Slide 2** Ear Swab: Positive for / Negative
   - **Slide 3** Tape Prep/Impression Smear: Positive for / Negative
     - Pyoderma
     - Demodex
     - Dermatophytosis (if suspected, confirm with DTM culture)
     - Otitis (Cocci, Yeast, Pseudomonas)
     - Pododermatitis (Cocci, Yeast)
     - Yeast Dermatitis

2. **COMMON ALLERGIC SIGNS**

   A. **LUMBAR DERMATITIS**
   **Flea Allergy:** (very reliable pattern)
   1. Caudal 1/3 of body
   2. Flea comb identifying fleas or flea dirt
   3. Multiple animals involved or humans affected
   4. Variable response to steroids
   5. Fall and Spring are often worse but can be year-round

   B. **EAR-SCRATCH TEST**
   **Scabies:** (1-2 are highly reliable)
   1. Positive pinnal pedal reflex is 80% diagnostic
   2. Ear margin, distal legs, lateral elbow, ventrum
   3. Variable responsive to steroids
   4. Confirmed by response to treatment
   5. Skin Scrapes are often falsely negative

   C. **PERIANAL DERMATITIS**
   **Food Allergy:** (less common but 1-5 increase probability)
   1. Perianal dermatitis
   2. GI symptoms; more than 3 BM/day, diarrhea, vomiting, flatulence
   3. Less than 1 year or older than 5 years at onset
   4. Labradors and German Breeds may be predisposed
   5. Variable response to steroids

   **Hypothyroidism:** (can mimic allergic dermatitis)
   1. Recurrent infection may cause pruritus
   2. Lethargy, weight gain, dry coat, hypotrichosis
   3. Nonpruritic when infections are resolved

   D. **FOOT LICKING**
   **Atopic Dermatitis:**
   (1-5 are highly reliable)
   1. Started at 6 months – 3 years of age
   2. Front feet affected
   3. Inner ear pinnae erythema
   4. Lives indoors
   5. Ruling out Scabies (ear margin dermatitis) and Flea allergy (lumbar dermatitis)
   6. Seasonal symptoms progressing to year-round
### Treat the Acute Flares:

<table>
<thead>
<tr>
<th>Cause</th>
<th>Recommended Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bacterial Pyoderma</td>
<td></td>
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<tr>
<td>Yeast Infections</td>
<td></td>
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<tr>
<td>Otitis</td>
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<tr>
<td>Flea Infestation</td>
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<tr>
<td>Scabies Treatment</td>
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<tr>
<td>Steroid “Crisis” Therapy</td>
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<tr>
<td>Topical Short-Term Steroid</td>
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</tbody>
</table>

### Treatment, Control and Prevention of Future Flares:

<table>
<thead>
<tr>
<th>Cause</th>
<th>Recommended Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atopy</td>
<td></td>
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<tr>
<td>Immunotherapy</td>
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<tr>
<td>Allergy Vaccine</td>
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<tr>
<td>Atopica® (Cyclosporine capsules, USP) MODIFIED</td>
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<tr>
<td>Thyroid Supplementation bid</td>
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</tbody>
</table>

### Avoiding the Triggers:

<table>
<thead>
<tr>
<th>Cause</th>
<th>Treatment</th>
<th>Recommended Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bacteria</td>
<td>Regular bath with an antimicrobial shampoo. Wipe off affected areas (feet, face, etc.) as often as possible</td>
<td></td>
</tr>
<tr>
<td>Yeast</td>
<td>Routine Ear Treatment/cleaning</td>
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<tr>
<td>Pollens</td>
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<tr>
<td>Otitis</td>
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<tr>
<td>Flea and Intestinal Parasites</td>
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<tr>
<td>Food Triggers</td>
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<tr>
<td>House Dust Mites</td>
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</tbody>
</table>

### Promote Skin Health and Restore Barrier Function:

<table>
<thead>
<tr>
<th>Cause</th>
<th>Recommended Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Essential Fatty Acids</td>
<td></td>
</tr>
<tr>
<td>Antihistamines</td>
<td></td>
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<tr>
<td>Soothing, Leave on Conditioner</td>
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### Recheck Appointment:

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Almost all dermatology patients have a primary/underlying disease which causes secondary infections. The infections must be eliminated and prevented, but will recur unless the primary disease is identified and controlled.

Most skin cases seen in practice can be successfully managed if these 2 question can be answered. Once the etiology of a patients dermatosis is known, it is a simple matter of therapeutic followthrough to resolve the problem.

The recognition of the basic patterns allows a practical approach to most of the common skin diseases.

10 Clinical Patterns

What are the infections? (Always secondary)
1. Folliculitis
2. Pododermatitis
3. Otitis
4. Yeast Dermatitis

Why are they there? (The key to preventing relapse of infections)
5. Pruritus
6. Nonpruritic Alopecia (endocrine)
7. Autoimmune Skin Disease
8. Keratinization Defects
9. Lumps, Bumps, and Draining Tracts
10. Weirdopathies

Case example: 2 year old male Labrador that has seasonal pruritus (foot licking) and a moth-eaten hair coat.

What are the Infections?
- Folliculitis
  *pyoderma, demodex, dermatophytes*
- Pododermatitis
  *bacterial, yeast*
- Otitis
  *bacterial, yeast*
- Yeast dermatitis

Why are they there?
- Allergies
  - Atopy
  - Food allergy
  - Scabies
- Endocrinopathy
  - Hypothyroidism
  - Cushing’s