

Approach to the pruritic dog

When faced with a pruritic dog in the exam room, considering the numerous possible causes of the dog's symptoms can be sometimes overwhelming. One way to approach these cases is by following a flowchart, such as in Figure 1. The flowchart emphasizes the importance of obtaining a thorough history regarding each patient (which can be enhanced by having the owner fill out a dermatology questionnaire – See Appendix) as well as performing essential diagnostic tests such as skin scrapings for mites, skin cytology for bacteria, yeast and inflammatory cells, and dermatophyte cultures to identify infections on every pruritic pet. Depending on history, seasonality, and response to treatment of parasites and infections, further assessment with skin biopsy, hypoallergenic diet trial, or allergy testing may be warranted.

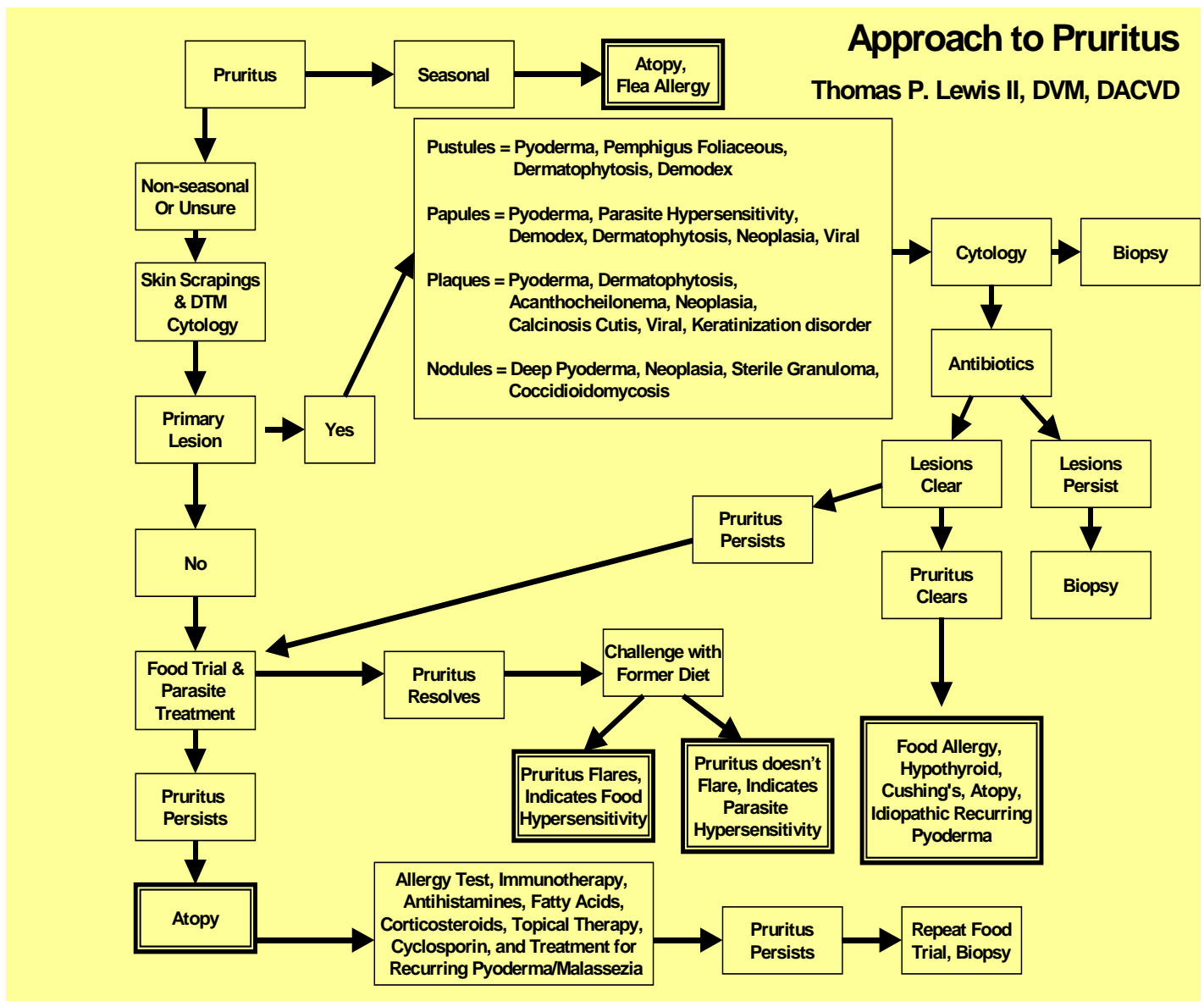


Figure 1

Another simplistic but sensible way to approach the workup of pruritic animals is to keep in mind that 90-95% of cases will be caused by only 3 main things: parasite infestation, skin infections, or allergies (or some combination thereof). The other 5-10% are less common skin diseases such as pemphigus foliaceus, sebaceous adenitis, cutaneous lymphoma, hepatocutaneous syndrome, etc. Once parasites and infections have been treated, then the only thing left to do is an allergy workup (+/- skin biopsy in the unusual cases). Whether you use a flow chart or the “rule of 3”, the important thing is that each case is approached methodically; if you use the same workup with each pruritic case, the previously overwhelming possibilities become ordered and approachable.

The “Rule of 3”: Parasites, Infections, or Allergies

Parasites: The main skin parasites which cause pruritus in dogs include fleas, scabies, cheyletiella, and demodex.

Fleas:

Clinical signs: Fleas are small dark brown wingless insects which live on the blood meals obtained from their mammalian hosts. *Ctenocephalides felis* is the most common flea species that parasitizes dogs and cats. In most animals, fleabites cause minor pruritus and dermatitis, but in animals with fleabite hypersensitivity, a single fleabite can cause severe pruritus, excoriations, and secondary infections. Classically, animals with fleabite hypersensitivity are most pruritic on the dorsal lumbar and tail head areas and caudomedial thighs. Excessive scratching and chewing by the pet lead to self induced alopecia, excoriations, and secondary bacterial skin infections. Symptoms occur most commonly in the warm months of the year when fleas are most numerous.

Diagnosis: Clinical signs, distribution of lesions, and identification of fleas or dark flea dirt by flea combing is diagnostic of flea bite hypersensitivity. In some animals, no fleas are found, but presence of tapeworms in the stool (carried by fleas ingested when the animal grooms) may be a clue. Response to trial ectoparasiticide therapy may also be diagnostic. If an owner refuses to believe fleas are a problem, intradermal allergy testing or serology for flea IgE can be used, but false negative and false positive results can occur.

Treatment: Control of flea bite hypersensitivity involves insecticides to kill the adult fleas on all the pets in the household, as well as measures to stop the propagation of fleas in the environment. The best products for flea allergic pets are long acting products such as Frontline/Frontline Plus, Advantage/Advantix, and Revolution, some of which have effects not only on adult fleas but on flea eggs and larvae. High concentration permethrin products are also effective, but can be more toxic, especially to small dogs and cats in close contact with treated dogs. In flea allergic pets, products may need to be applied more often than the labeled monthly frequency. Program (lufenuron) inhibits chitin production and is helpful to prevent flea reproduction, but does not kill adult fleas or stop them from biting the hypersensitive pet. Most over the counter flea sprays and powders need to be applied too frequently to be very helpful, with the exception of Knockout spray by Virbac which contains both pyrethroids to kill adult fleas and pyriproxyfen (Nylar) as an insect growth regulator to prevent flea reproduction. Frontline Plus also has an insect growth regulator (methoprene) which sterilizes any fleas which may become resistant to the insecticide. Flea collars are

minimally helpful in most animals. In addition to treatment of the pets, use of an environmental insecticide with an insect growth regulator is important, as the fleas seen on the pets represent only a fraction of the flea eggs, larva and pupae in the environment. Knockout by Virbac also comes as a premise spray and fogger. Environmental products should be applied where the pets spend most of their time (beds, dog houses, carpets, under bushes and porches, etc.). With the advent of highly effective flea control products in the last 10 years, flea allergy dermatitis has become a less frequently seen problem, and is more controllable, but since fleas have a remarkable ability to become resistant to insecticides, an insect growth regulator should always be used in the flea control plan.

Other considerations in the treatment of fleas and flea allergy dermatitis are to identify and treat secondary bacterial +/- malassezia skin infections which will perpetuate pruritus even after fleas have been eliminated. Additionally, a tapering course of oral prednisone (0.5mg/kg PO BID x 5, then once daily X 5, then QOD x 10 days) is often needed to break the “itch-scratch” cycle. Antihistamines and soothing antipruritic shampoos and conditioners may also be helpful. Immunotherapy for flea allergy dermatitis has not proven to be particularly helpful, probably because the flea extract used for allergy testing and immunotherapy is manufactured from the whole flea, and the allergenic salivary components are a very small portion of the extract. There has been research into immunizing flea allergic dogs with flea salivary extracts alone, as well as a vaccine which, when given to dogs, causes production of canine antibodies which attack fleas when they take a blood meal, but the status of this research is uncertain.

Scabies:

Clinical signs: Scabies, or sarcoptic mange, is caused by the mite *Sarcoptes scabiei var. canis*. Although the mite prefers dogs, they can also cause disease in other species such as cats, foxes and humans. This mite lives within the stratum corneum, is very infectious to in-contact dogs and causes nonseasonal poorly steroid-responsive pruritus and dermatitis which can be very severe, and can mimic allergies. Affected animals classically have lesions of papules, crusts, excoriations and alopecia affecting ear margins, elbows, hocks and ventral trunk, but dogs may also have generalized pruritus and no obvious skin lesions. Some dogs can be minimally symptomatic and serve as carriers or mites.

Diagnosis: Multiple superficial skin scrapings are taken, concentrating on crusty pinnal or pressure point lesions, or the papular ventral dermatitis, to evaluate microscopically under 4X-10X for mites or mite eggs. One single mite or egg is diagnostic for scabies infection. However, skin scrapings may be diagnostic in only 20-50% of cases, as false negative scrapings are common. An ipsilateral “pinnal-pedal” response, when the dog’s hind leg starts scratching as the edge of the pinna is scraped, is highly suggestive of scabies. A scabies serum antibody test is available in Europe, but false negative and false positive results may occur. Often the diagnosis is indirectly obtained by resolution of symptoms after trial treatment for mites.

Treatment: Options for treating scabies are either topical treatment or systemic medications. Topical treatment options include 2-3% lime sulfur dips or organophosphate dips weekly for 6 weeks, 0.025% amitraz dips every 2 weeks for 3 treatments, Frontline spray 4 pumps or 3ml/kg applied every 2 weeks for 3 treatments, or Revolution applied every 2 weeks for 3

treatments. Systemic treatment options include 1% ivermectin 0.3-0.4mg/kg PO weekly for 6 weeks or SQ every 2 weeks for 3 treatments (not in herding breeds), and milbemycin 2mg/kg PO weekly for 4-6 weeks (safe in herding breeds). 1% moxidectin 0.2 – 0.25mg/kg PO or SQ weekly for 3-6 weeks (not in herding breeds) is also effective but more adverse side effects occur with this medication. When choosing a therapy option, keep in mind that Revolution is the only labeled drug approved for the treatment of scabies in dogs. We have found both ivermectin and Revolution to be very effective, and less labor intensive than dips or sprays. For young puppies, lime sulfur dips are the treatment of choice. Regardless of treatment chosen, all in contact dogs must be treated at the same time. If treating for parasites, the entire course of treatment must be followed—since the mite life cycle is 3 weeks and scabies eggs will hatch continuously, lack of clinical response to only 1 injection of ivermectin or only one antiparasitic dip cannot be relied on to rule out scabies. Resolution of symptoms is often rapid but can take 2-4 weeks to completely resolve. Mites can live in the environment an average of 2-6 days, or longer depending on humidity and temperature, and treatment of the environment with an insecticidal spray may be needed, especially in multidog or kennel situations. Additionally, identification and treatment of secondary skin infections and a short tapering course of prednisone to decrease self trauma are usually needed.

Cheyletiella:

Clinical signs: Cheyletiella mites are non-species specific mites which live on the skin surface and cause dorsal scaling and variable pruritus. Papules and crusts may also be present. Some animals can be asymptomatic carriers. Cocker spaniels seem to be over-represented.

Diagnosis: Superficial skin scrapings, acetate tape preps, or flea combing of dorsal scale are obtained for microscopic analysis for mites and eggs under 4X –10X. Skin scrapings can be falsely negative, and an indirect diagnosis may be made with response to trial treatment.

Treatment: The same topical and systemic treatment options as listed for scabies are effective for cheyletiella, however it is important to treat all mammals in the house. This parasite is zoonotic, and owners with lesions should be referred to their physician. Mites can live up to 10 days or more in the environment, and insecticidal environmental treatment may be needed, especially in multi animal situations.

Demodex:

Clinical signs: Canine demodicosis is caused most commonly by *Demodex canis*, a normal resident of canine hair follicles, and less commonly by the larger long-bodied *Demodex injai* and by the unnamed short bodied demodex mite. Demodex is not considered transmissible except from the dam to her puppies; adult to adult dog transmission may rarely occur. Demodicosis may occur as a localized or generalized form, and may occur in puppies < 18 mo (juvenile onset) due to their immature immune system, or in older dogs (adult onset), due to underlying immunosuppression from steroids, endocrine diseases such as hyperadrenocorticism or hypothyroidism, or neoplasia. Due to a higher occurrence of generalized demodicosis in certain breeds (Shar peis, bulldogs, westies, etc.) and families of dogs, a genetic tendency and heritability is likely, and impaired T helper cell activity is suspected. Signs of demodex range from patchy alopecia with silvery scale, comedones, papules and pustules in more mildly affected dogs, to severe lichenification, crusting,

draining bullae and ulceration due to secondary deep pyoderma in more severely affected dogs. Lesions often start on the face and feet/legs, but can occur anywhere on the body. Severely affected dogs may be lethargic, febrile and have peripheral lymphadenopathy. Pruritus may be mild to marked. Demodectic pododermatitis causes interdigital erythema, swelling, crusting, draining tracts, pain and pruritus. Demodicosis can also cause ceruminous otitis. *Demodex injai* usually causes oily scaling and erythema +/- alopecia on the dorsal trunk, often in terrier breeds.

Diagnosis: Deep skin scrapings and microscopic evaluation under 4-10X for mites are necessary for diagnosis; squeezing the scraped area and scraping until a small amount of capillary bleeding is seen on the scalpel blade are needed. In hard to scrape areas such as feet and eyelids, hair plucks and visualization of mites around the plucked hair roots is often needed. In most cases with adequately deep scrapings, mites are easy to find; exceptions may occur with severely scarred demodectic pododermatitis and in Shar peis in which tissue biopsy may be needed for diagnosis. Additionally, *D. injai* mites are usually only found in low numbers.

Treatment:

Treatment for localized demodex in puppies is often not needed, as 85% will spontaneously resolve within 4-8 weeks. Topical rotenone (Goodwinol) or localized treatment with 0.03 – 0.05% amitraz may speed resolution of localized lesions, but will not stop development of generalized infection.

Treatment options for generalized demodex are either topical or systemic antiparasitic therapy. The only effective topical therapy is weekly application of 0.03 – 0.05% amitraz (Mitaban) dips. In refractory cases, the dips may be used in a higher concentration or more frequently, but side effects may occur. It is important to clip the hair of medium to long haired dogs prior to dipping, and a benzoyl peroxide shampoo is often used to help flush hair follicles prior to the dip. Dips are given until 1 month beyond a negative skin scraping. Potential side effects of amitraz include pruritus, and excessive sedation and hypothermia (especially in small breed dogs). Severely sedated dogs can be treated with yohimbine. Lime sulfur dips and Revolution are ineffective for canine demodex.

Systemic treatment options for generalized canine demodicosis include ivermectin (not in herding breeds) and milbemycin. 1% Ivermectin is given orally at a dose range of 0.3 – 0.6mg/kg daily. The cure rate for the 0.6mg/kg dose is 85-90%. The daily dose is gradually increased to the maintenance dose to screen for sensitive animals (ie. Day 1 give 0.1mg/kg, day 2 give 0.2mg/kg, day 3 give 0.3mg/kg, day 4 give 0.4mg/kg, day 5 give 0.5mg/kg, etc.) The maintenance dose is then given orally every day until 1 month beyond a negative skin scraping. Alternate day dosing may also be effective, but once weekly ivermectin is not effective for canine demodicosis. Potential signs of ivermectin toxicity include mydriasis, blindness, ataxia, lethargy and anorexia. If any of these symptoms are seen, ivermectin should be discontinued immediately, or more severe effects such as seizures, coma or death may occur. A genetic test is available through the Washington State University College of Veterinary Medicine to screen for dogs with ivermectin sensitivity, and more information can be found at www.vetmed.wsu.edu/depts-VCPL. Milbemycin is an effective alternative to ivermectin which has fewer potential side effects but is much more expensive. The dose range for milbemycin is 0.5 – 2mg/kg PO daily, and it is currently marketed as the

heartworm preventative Interceptor. Doramectin is reported to be effective at a dose of 0.6mg/kg SQ once weekly, but the same potential adverse effects as ivermectin are possible. 1% moxidectin has also been reported to be effective at a dosage of 0.4mg/kg PO q 1-3 days, but adverse effects are common with this drug.

Regardless of the treatment chosen, it is essential to follow up treatment with skin scrapings every 3-6 weeks, and to continue treatment until 1 month beyond a negative scraping. Animals will look normal 1-3 months before they are truly cured, and premature cessation of treatment is the most common reason for relapse. Animals that relapse often become harder to treat. In dogs with adult onset demodex, identification and treatment of the underlying cause is necessary (ie. stop systemic steroids, perform labwork to screen for endocrinopathy, etc.). Other considerations in the treatment of dogs with demodex include treatment of secondary pyoderma with a 4-8 week course of oral antibiotics, and spaying/neutering intact dogs with generalized disease. Neutering is recommended not only because estrus and pregnancy can trigger or exacerbate disease, but because of the heritability of the genetic tendency for generalized demodicosis. The prognosis for cure of demodex is good for young dogs and guarded for older dogs in which an underlying cause cannot be identified and treated. In dogs which relapse repeatedly after appropriate drug doses and duration of treatment, demodicosis may be satisfactorily controlled with lifelong ivermectin or milbemycin given three times weekly.

Infections: Skin infections which commonly cause pruritus include bacterial pyoderma, malassezia dermatitis, and dermatophytosis. When dealing with animals with recurrent bacterial or yeast skin infections, it is important to identify the underlying cause of the infections. Common causes of chronic or recurrent skin infections include antimicrobial therapy which is of insufficient dose or duration, exogenous steroids causing immunosuppression, allergy (food, atopy or flea), immunosuppressive endocrinopathies such as hyperadrenocorticism or hypothyroidism, or keratinization disorders. Until the underlying cause is identified and addressed, skin infections will continually recur.

Bacterial pyoderma

Clinical signs: Canine pyoderma is most commonly caused by *Staphylococcus intermedius*, which is part of the normal skin flora, but becomes pathogenic due to underlying diseases such as those listed above. Lesions seen with superficial pyoderma include papules, pustules and epidermal collarettes, patchy alopecia, and crusting. Epidermal collarettes can appear similar to “ringworm” and often have central post inflammatory hyperpigmentation. Lesions can be more subtle in patients with long hair coats and be limited to a dull coat, easily epilated hair, and epidermal collarettes resulting in scale. In white-coated breeds such as Dalmations, bacterial folliculitis can appear as scattered tan patches on a previously white coat. When the areas are shaved, the underlying papules can be seen. Bacterial pyoderma can also cause lichenification and hyperpigmentation similar to malassezia dermatitis. Pruritus in dogs with superficial pyoderma ranges from mild to severe; in allergic dogs, Staph can act as a superantigen and severely exacerbate allergic pruritus.

Deep pyoderma is caused when superficial bacterial infection progresses and penetrates below the hair follicle into the dermis, causing swelling, cellulitis, bullae,

hemorrhagic crusts, ulcers, and draining tracts. Affected dogs may also be febrile or have a lymphadenopathy.

Diagnosis: The diagnosis of bacterial pyoderma is by identification of characteristic lesions, ruling out other causes of folliculitis such as demodex and dermatophyte by performing skin scrapings and dermatophyte culture, and by identification of bacteria on skin cytology. Cytology may be obtained by rupturing a pustule and smearing the contents onto a slide, or if no intact pustules are present, by raising a crust and either impressing a slide directly onto the area under the crust, or using a dry scalpel blade to gently scrape the debris under the crust. Cotton swabs may be helpful to obtain debris from intertriginous areas or between toes. This debris is then smeared onto a slide, heat fixed and stained with Diff Quick or similar stain to look for inflammatory cells and bacteria under 100X/oil immersion. In dogs with poor clinical response to appropriate antibiotics given at appropriate doses/duration, bacterial culture of a pustule or exudate under a crust is indicated to guide antibiotic therapy. In animals with deep pyoderma, analysis of exudate often reveals pyogranulomatous inflammation with few organisms; gram staining in these instances can help identification of bacteria. In dogs with chronic deep pyoderma, biopsy for histopathology and tissue culture is indicated.

Treatment: In animals with recurrent pyoderma, it is important to identify and treat the underlying cause as discussed above. Treatment of superficial pyoderma requires antibiotic therapy for a minimum of 2-4 weeks. Deep infections require 4-12 weeks of antibiotic therapy. Antibacterial shampoos such as Hexadene 1-2 times weekly can speed resolution of infection and help prevent recurrent infections, but shampoos alone are rarely able to clear bacterial pyoderma. Antibiotic therapy options for first time uncomplicated superficial bacterial pyoderma include lincocin, erythromycin and clindamycin, but bacterial resistance occurs quickly with these antibiotics. In animals with recurrent or complicated infections, more appropriate antibiotic choices include cephalosporins, Clavamox, trimethoprim sulfa or ormetoprim sulfa. Fluoroquinolone should be reserved as third line therapy in resistant infections, deep infections, or infections which have numerous rod shaped bacteria on cytology. Antibiotics which are not appropriate choices for bacterial pyoderma due to reliable resistance of staphylococcus include amoxicillin/ampicillin, non-potentiated sulfas, and tetracycline.

Yeast dermatitis:

Clinical signs: Yeast dermatitis is caused by *Malassezia pachydermatis*, which in low numbers is part of the normal skin flora in the dog, especially in the ear canals, between the toes and on the perianal area. As with bacterial infections, animals with recurrent yeast dermatitis often have an underlying immunosuppressive or allergic cause, and dogs with yeast dermatitis often have concurrent bacterial pyoderma. Unlike human medicine, antibiotic therapy is not associated with secondary *Malassezia* dermatitis. Lesions of *Malassezia* dermatitis include erythema, alopecia, lichenification, hyperpigmentation, greasy yellow scale, and odor. Commonly affected areas include the ventral neck, axillae and inguinal areas, and flexural surfaces of the elbows and tarsi. Affected dogs are usually very pruritic and pruritus is not steroid responsive. Some dog breeds appear predisposed to yeast dermatitis, including Westies and cocker spaniels, but this breed predisposition may also reflect the high incidence of allergies in these breeds.

Diagnosis: The diagnosis of yeast dermatitis is by clinical signs and by ruling out other infectious organisms such as demodex and bacterial pyoderma with skin scrapings and cytology, and by identification of the peanut shaped yeast organisms on cytology. In addition to the same methods used to obtain cytology as described for bacteria, clear acetate tape pressed to affected skin then applied to a slide on top of a drop of blue stain and examined under 40-100X can be diagnostic.

Treatment: As with recurrent bacterial pyoderma, it is important to identify and treat the underlying immunosuppressive or allergic disease in dogs with recurrent yeast dermatitis. Identification and treatment of concurrent bacterial pyoderma is also usually necessary. Treatment of the *Malassezia* infection involves topical antifungal therapy such as miconazole based cream, shampoo or rinses, ketoconazole shampoo, chlorhexidine shampoo (> 2% concentration), or benzoyl peroxide shampoo which is degreasing as well as antimicrobial. Topical therapy alone may be sufficient in mild or localized cases, but in more severely affected dogs, topical antifungal therapy should be combined with systemic antifungal medications such as ketoconazole, itraconazole or fluconazole 5-10mg/kg PO daily for 2-4 weeks. After a loading dose of 1-2 weeks of daily therapy, itraconazole can be used as weekend “pulse” therapy due to its lipophilicity and retention in the skin for prolonged periods. Griseofulvin is ineffective for malassezia dermatitis.

Dermatophytosis:

Clinical signs: Dermatophytosis is a fungal infection of the hair and stratum corneum caused most commonly by *Microsporum canis*, *M. gypseum*, and *Trichophyton mentagrophytes*. Clinical signs include patchy, usually asymmetrical alopecia (which may or may not be in a “ring”), crusting, and scaling. Some animals will develop a fungal kerion, which is a raised, erythematous nodule or plaque composed of pyogranulomatous inflammation and fungus. Lesions can occur anywhere on the body. Affected animals are usually minimally to mildly pruritic, but some animals may be moderately to severely pruritic. Severe cases may develop a deep furunculosis with draining tracts. Dermatophytosis is more common in young or immunosuppressed animals, and in certain breeds such as Yorkies, Jack Russell terriers, and Persian cats. Dermatophytosis is the most common cause of folliculitis and hair loss in cats.

Diagnosis: Dermatophytosis is definitively diagnosed by dermatophyte culture. Hair plucks with hemostats can miss infected hairs, and the preferred method of fungal culture is to brush a new toothbrush through the fur and over the affected areas, and then gently impress the bristles of the brush onto a dermatophyte culture medium to deposit collected hair and scales. Sab-duets or Derm-duets by Bacti-lab (1-800-227-3700 or www.bacti-lab.com) are rectangular fungal culture plates which are easier to use than tubes, and have both a color change side and a side more conducive to fungal spore formation for fungus identification. Fungal cultures are incubated in a warm, dark, moist place for 2-3 weeks, and should be checked daily for fungal growth. Dermatophyte colonies are white to tan, and the fungal culture media changes color at the same time as the fungus grows. Contaminant fungi can be any color including white, green, and grey, but the fungal culture media may change color only several days after the fungal colony starts growing. True dermatophytes will never have dark brown, black or green pigment. Cytology of suspect fungal colonies should be performed by touching clear acetate tape to the colony, then sticking the tape to a slide on

top of a drop of blue stain to evaluate microscopically under 10-40X for the boat or cigar shaped macroconidial spores diagnostic of dermatophytosis. Other methods of dermatophyte diagnosis include skin biopsy and histopathology and cytological identification of fungal ectothrix spores on a trichogram of infected hair, but false negative results may occur. Wood's lamp examination is not recommended as the sole diagnosis of dermatophytosis, as only 50% of *M. canis* strains will fluoresce, and false negative and false positive results are common.

Treatment: Treatment of localized dermatophytosis involves clipping the area and application of a topical antifungal cream or ointment BID until healed. Topical medication options include miconazole, clotrimazole, thiabendazole, ketoconazole and terbinafine. In animals with generalized dermatophytosis, topical antifungal treatments (lime sulfur dip or miconazole rinse 1-2 times weekly; shampoos are less effective) are combined with oral antifungal medication. Oral medication options include ketoconazole (not in cats), itraconazole, or fluconazole 5-10mg/kg PO daily, terbinafine 30-40mg/kg PO SID, or griseofulvin. Because of its lipophilic nature, itraconazole is also effective when used as pulse therapy. The loading dose of 5-10mg/kg PO daily is given for 2 weeks, then on weekends, or on a "week on/week off" basis. Unfortunately, despite an initial promising case report, in multiple subsequent controlled studies involving both experimentally and naturally exposed cats (as well as out own personal case experiences), Lufenuron has not been shown to be effective for the treatment of dermatophytosis. Regardless of treatment chosen, antifungal therapy is continued until 2 negative fungal cultures, and fungal cultures by toothbrush technique are rechecked every 2-4 weeks (the recheck fungal cultures should be monitored for 3 weeks, as antifungal therapy will slow fungal growth).

In addition to treatment of the infected animal, in contact animals should also be toothbrush cultured (especially with *M. canis* infection), and culture negative animals should be isolated from infected pets if possible. Exposed animals should also be prophylactically treated with antifungal dips while results of their cultures are pending. Environmental decontamination should be performed by washing all bedding and hard surfaces in dilute bleach and water, disposing of grooming equipment, cat trees and other disposable things which cannot be disinfected, and frequently vacuuming and disposing of the vacuum bags.

Allergies: If pruritus persists once parasitic and infectious causes have been ruled out or treated, or if skin infections recur after appropriate treatment and there is no immunosuppressive condition present, then underlying allergy is likely. Atopy and food allergy cause very similar symptoms, and can be present concurrently. It is important to get a thorough history, including any seasonality or a history of a good response of pruritus to steroids suggestive of atopy (keeping in mind that atopy can also be non-seasonal, and if secondary skin infections or parasites are present can also be non-steroid responsive), gastrointestinal symptoms or poor response to steroids suggestive of food allergy, etc. The client questionnaire in Figure 2 is a good start.

Atopy:

Symptoms: Atopy is a hypersensitivity reaction to percutaneously absorbed and inhaled allergens (such as pollens, molds, housedust mites, dander etc.) in genetically predisposed dogs. Age of onset ranges from 6 months to 6 years of age, but in most atopic dogs symptoms begin between 1 and 3 years of age. Symptoms may occur seasonally or non-

seasonally, and it is common for seasonal signs to progress to non-seasonal symptoms. Atopy can occur in any breed of dog, including mixed breeds, but is more common in breeds such as terriers, Labradors, golden retrievers, shar peis, and spaniels. The primary sign of atopy is pruritus, especially of feet, axillae, inguinal area, face and ears. Pruritus is often steroid responsive, unless secondary skin infections are present. Due to pruritus and self-trauma, as well as to abnormal cutaneous immunity, secondary bacterial and malassezia skin and ear infections are common in atopic dogs, and worsen pruritus.

Diagnosis: The diagnosis of atopy is by considering clinical signs and by ruling out other causes for pruritus such as parasites, skin infections, and food allergy with appropriate diagnostic testing. Because allergy tests (both serologic and intradermal) can have irrelevant positive reactions in dogs with food allergy, skin parasites or skin infections, allergy testing is performed after the clinical diagnosis of atopy is made, in order to determine pertinent allergens to include in a desensitization vaccine. Intradermal allergy testing is considered the “gold standard” in the diagnosis of pertinent allergens, with fewer false positive reactions compared to most serologic tests. The down sides of intradermal allergy testing are that it requires sedation and shaving, usually requires referral to a dermatologist, and that steroid and antihistamine therapy will hinder interpretation of test results. A dog should be off oral steroids for 4 weeks, withdrawn from long acting injectable steroids for 6-8 weeks, and off antihistamines for 1-2 weeks prior to intradermal allergy testing. Additionally, oral fatty acids and topical steroids (skin or ear products) should be discontinued 2-3 weeks prior to testing. Oral cyclosporin does not seem to interfere with intradermal allergy test results, and can be used to enable discontinuation of steroids prior to testing. Serologic testing for atopy is more convenient, does not require sedation and shaving, or referral to a dermatologist, however allergy serology is not as accurate as intradermal testing, and false positive reactions are common in most tests. The Heska serologic allergy test seems to have good correlation of positive serologic reactions with intradermal reactions, but false negative reactions seem to be more common with this test. Serologic allergy testing may be less affected by steroid or antihistamine therapy compared to intradermal testing, however several companies are now recommending that steroids and antihistamines be discontinued for several weeks prior to serologic testing, negating this advantage. Both intradermal and serologic allergy testing are expensive, and allergy immunotherapy is labor intensive. Success of immunotherapy depends on selection of the right allergens, adjusting immunotherapy vaccine content, dose, or frequency based on the individual dog’s needs, and experience of the veterinarian to know how and when to make these adjustments. It is therefore to the benefit of most allergic dog and allergic dog owners to be referred by their regular veterinarian to a veterinary dermatologist for allergy testing and immunotherapy, to avoid poor therapy results and owner dissatisfaction.

Treatment: Treatment of atopy is multifaceted, and includes controlling secondary skin and ear infections, avoidance of allergens if possible, and treating symptoms medically with antihistamines, fatty acids, frequent bathing, and occasional topical and oral steroids. Long acting injectable steroids are inappropriate for dogs. Short-term oral steroids are safer and can be titrated to the lowest possible dose for comfort, ideally < 0.5mg/kg prednisone PO QOD, administered for less than 2-3 months/year. Atopica or oral cyclosporin can be used in many dogs as a less toxic alternative to steroids to control pruritus, but cyclosporin is expensive, especially for larger dogs, and should still be considered lifelong immunosuppressive therapy. We use Atopica or cyclosporin not as a first line therapy in the

treatment of atopy, but as an alternative to steroids in dogs in which allergy immunotherapy is not helpful, and in older dogs which are not appropriate candidates for allergy testing and immunotherapy. We also use cyclosporin to enable dogs to discontinue steroids prior to allergy testing, and, in some dogs, to control pruritus until immunotherapy has time to take effect. Allergy testing and immunotherapy are indicated in atopic dogs whose symptoms cannot be controlled with benign medical management options, or who need steroids for longer than 2-3 months/year. Allergy immunotherapy is helpful in 50-80% of allergic dogs to decrease clinical signs and need for medications, and can take 2-12 months for maximal response. Immunotherapy treats the cause of the symptoms, not just the symptoms themselves, and side effects are rare. We will cover these treatment options in more detail in the next lecture.

Food allergy:

Symptoms: Food allergy is an immunologic reaction or intolerance to food or food additive. Any food can be allergenic, but most common allergenic foods include wheat, corn, beef, dairy and chicken. Food allergy can occur at any age, including puppies and old dogs, even after eating the same food for years. Predisposed breeds include Labradors, cocker spaniels, golden retrievers and Shar peis. The symptoms of dogs with food allergy are similar to atopy, with pruritus often directed at feet, ears, perineum, face, axillae and inguinal area. The pruritus is nonseasonal and variably, often minimally, steroid responsive. 20-30% of dogs with food allergy have concurrent gastrointestinal symptoms such as constant or intermittent vomiting and diarrhea, excessive flatulence, and increased frequency of bowel movements. As with atopy, secondary skin and ear infections are common in food allergic dogs, and recurrent pyoderma can be the only sign in some food allergic dogs.

Diagnosis: Diagnosis of food allergy is by consideration of clinical signs, ruling out other causes of pruritus such as skin parasites and skin infections, and by performing a hypoallergenic diet trial for a minimum of 6-8 weeks. Serologic or intradermal allergy testing for food allergy are not accurate and are not recommended. Hypoallergenic diets can be either based on a novel protein source (we have had the most success with Royal Canin rabbit/potato formula), or a hydrolyzed formula. Some dogs respond better to one diet type versus the other, and sometimes multiple diets have to be tried to find the diet that works. Additionally approximately 10% of food allergic dogs will only respond to a home cooked hypoallergenic diet, possibly due to a reaction to a food additive or preservative. During the hypoallergenic diet trial it is essential to remove treats, rawhides, chewable supplements including vitamins and chondroitin supplements, chewable heartworm preventatives, etc. Also, make sure owners are not giving pills in cheese, peanut butter, bread, pill pockets etc. Giving pills in the canned hypoallergenic diet is recommended. Acceptable treats include baked slices of the canned hypoallergenic food. For dogs that like to chew, dried rabbit ears or fish skins as an alternative to rawhides can be found on the internet (www.sitstay.com). After the minimum 6-8 week trial, the diagnosis of food allergy is made by challenging the dog with its prior diet and monitoring for flare of pruritus within 1-10 days of the challenge. If pruritus flares, the hypoallergenic diet is immediately restarted. Options at that point are either to feed the prescription hypoallergenic diet lifelong, to try one of the over the counter hypoallergenic diets to see if they control symptoms as well as the prescription food, or, to identify specifically what a dog is allergic to, perform sequential diet challenges with individual food allergens every 3-4 weeks.

Treatment: The treatment of food allergy is to avoid offending food allergens and feed the hypoallergenic diet lifelong. If pruritus flares despite the hypoallergenic diet, common causes include dietary indiscretion, exposure to skin parasites such as fleas or scabies, development of secondary bacterial or yeast skin infection, or development of concurrent atopy. Less commonly, dogs can develop a new hypersensitivity to a component in the hypoallergenic diet, and a repeat hypoallergenic diet trial may be necessary.

Conclusion: Although there are many potential causes of pruritus in dogs, the diagnostic work up is the same for every pruritic dog:

1. Screen for ectoparasites with flea combing and both superficial and deep skin scrapings.
2. Perform skin cytology to identify bacterial and yeast skin infections, +/- a dermatophyte culture in suspect lesions.
3. Treat what you know is there (ie. fleas, infections) and trial treat for scabies even if skin scrapings are negative for mites.
4. If skin lesions such as plaques, crusts, papules, pustules, nodules, ulcers, depigmentation or follicular casts remain after appropriate antibiotics and parasite trials, skin biopsies should be performed to look for less common skin diseases such as pemphigus, cutaneous lymphoma, sebaceous adenitis or hepatocutaneous syndrome. Perform multiple biopsies of different lesions/areas, and send the biopsies with a complete written history including clinical signs and current medications to a dermatopathologist for most diagnostic results.
5. If pruritus persists after treatment of skin infections and parasites, or if skin or ear infections recur and there is no underlying immunosuppressive drug or condition, then the only remaining option is workup for allergies with a hypoallergenic diet trial +/- allergy testing/desensitization depending on the animal's response to the diet trial and the severity of clinical signs.
6. Don't be afraid to call or refer to a veterinary dermatologist for your tough dermatology cases, we're here to help both you and your clients. Referral can often result in a faster diagnosis and less expense and frustration for the owner.