**#6 Canine Food Hypersensitivity**

**Features**

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

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

**Top Differentials**

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

**Diagnosis**

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

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

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

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

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

**Treatment and Prognosis**

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

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

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

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

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

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

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

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

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

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

**8. Food Allergy Treatment**

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

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

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

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

Author’s Note

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

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

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

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

Author’s Note

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

**#7 Canine Hypothyroidism**

**Features**

This endocrinopathy is most often associated with primary thyroid dysfunction caused by lymphocytic thyroiditis or idiopathic thyroid atrophy. It is common in dogs, with highest incidence in middle-aged to older dogs. Young adult large and giant-breed dogs are also occasionally affected. Congenital hypothyroidism is extremely rare.

A variety of cutaneous symptoms can be seen. Alopecia on the bridge of the nose occurs in some dogs as an early symptom. The hair coat may be dull, dry, and brittle. Bilaterally symmetrical alopecia that spares the extremities may occur, with easily epilated hairs. Alopecic skin may be hyperpigmented, thickened, or cool to the touch. Thickened and droopy facial skin from dermal mucinosis, chronic seborrhea sicca or oleosa, or ceruminous otitis externa may be present. Seborrheic skin and ears may be secondarily infected with yeast or bacteria. In some dogs, the only symptom is recurrent pyoderma or adult-onset generalized demodicosis. Pruritus is not a primary feature of hypothyroidism and, if present, reflects secondary pyoderma, *Malassezia* infection, or demodicosis. Noncutaneous symptoms of hypothyroidism are variable and may include aggression, lethargy or mental dullness, exercise intolerance, weight gain or obesity, thermophilia (cold intolerance), bradycardia, vague neuromyopathic or gastrointestinal signs, central nervous system involvement (e.g., head tilt, nystagmus, hemiparesis, cranial nerve dysfunction, hypermetria), and reproductive problems (e.g., decreased libido, prolonged anestrus, infertility). Puppies with congenital hypothyroidism are disproportionate dwarfs with short limbs and neck relative to their body length.

**Top Differentials**

Differentials include other causes of endocrine alopecia, superficial pyoderma, *Malassezia* dermatitis, and demodicosis.

**Diagnosis**

**1.** Rule out other differentials

**2.** Hemogram and serum biochemistry panel: nonspecific findings may include a mild, nonregenerative anemia, hypercholesterolemia, or elevated creatine kinase

**3.** Dermatohistopathology: usually, nonspecific endocrine changes or findings consistent with pyoderma, *Malassezia* dermatitis, or seborrhea are seen. If present, dermal mucinosis is highly suggestive of hypothyroidism, but this can be a normal finding in some breeds (e.g., Shar pei)

**4.** Serum total thyroxine (TT4), free thyroxine (FT4) by equilibrium dialysis, and endogenous thyroid-stimulating hormone (TSH) assays: low TT4, low FT4, and high TSH are highly suggestive of hypothyroidism, but false-positive and false-negative results can occur, especially with TT4 and TSH. For example, although TT4 is a good screening test, it should not be used alone to make a diagnosis because its serum level can be artificially increased or decreased by several factors, such as nonthyroidal illness, autoantibodies, and drug therapy (Table 9-1)

**Treatment and Prognosis**

**1.** Any secondary seborrhea, pyoderma, *Malassezia* dermatitis, or demodicosis should be treated with appropriate topical and systemic therapies.

**2.** Levothyroxine 0.02mg/kg PO should be administered every 12 hours until symptoms resolve (approximately 8‑16 weeks). Some dogs can then be maintained with 0.02mg/kg PO every 24 hours; others require lifelong twice-daily dosing to maintain remission.

**3.** Dogs with concurrent heart disease should be started on levothyroxine more gradually. Treatment should begin with 0.005mg/kg PO every 12 hours; dosage should be increased by 0.005mg/kg every 2 weeks until 0.02mg/kg every 12 hours is being administered.

**4.** After 2 to 4 months of therapy, the serum TT4 level should be measured 4 to 6 hours after medication administration and should be in the high normal to supranormal range. If the level is low or within the normal range and if minimal clinical improvement has been seen, the dosage of levothyroxine should be increased and the serum TT4 level checked 2 to 4 weeks later.

**5.** If signs of thyrotoxicosis from oversupplementation (e.g., anxiety, panting, polydipsia, polyuria) occur, the serum TT4 level should be evaluated. If the level is markedly elevated, medication should be temporarily stopped until adverse effects abate; it should then be reinstituted at a lower dose or a less frequent dosage schedule.

**6.** The prognosis is good with lifelong replacement thyroxine therapy, although hypothyroidism-induced neuromuscular abnormalities may not completely resolve.

**#7 Canine Hyperadrenocorticism** (Cushing’s disease)

**Features**

Spontaneously occurring hyperadrenocorticism is associated with the excessive production of endogenous steroid hormones (principally glucocorticoids, but sometimes mineralocorticoids or sex hormones) by the adrenal cortex. The disease is caused by a hyperfunctioning adrenal tumor (15%‑20% of cases) or pituitary tumor (80%‑85% of cases). Pituitary-dependent hyperadrenocorticism (PDH) is caused by the excessive production of adrenocorticotropic hormone (ACTH), usually from a pituitary microadenoma or macroadenoma. Iatrogenically induced disease occurs secondary to excessive administration of exogenous glucocorticoids. Iatrogenic hyperadrenocorticism can occur at any age and is common, especially in chronically pruritic dogs and dogs with immune-mediated disorders that are controlled with long-term glucocorticoids. Spontaneously occurring hyperadrenocorticism is also common and tends to occur in middle-aged to older dogs, with an increased incidence noted in Boxers, Boston terriers, Dachshunds, Poodles, and Scottish terriers.

The hair coat often becomes dry and lusterless, and slowly progressing, bilaterally symmetrical alopecia is common. The alopecia may become generalized, but it usually spares the head and limbs. Remaining hairs are easily epilated, and alopecic skin is often thin, hypotonic, and hyperpigmented. Cutaneous striae and comedones may be seen on the ventral abdomen. The skin may be mildly seborrheic (fine, dry scales), bruise easily, and exhibit poor wound healing. Chronic secondary superficial or deep pyoderma, dermatophytosis, or demodicosis is common and may be the client’s primary complaint. Calcinosis cutis (whitish, gritty, firm, bonelike papules and plaques) may develop, especially on the dorsal midline of the neck or ventral abdomen, or in the inguinal area.

Polyuria and polydipsia (water intake > 100mL/kg/day) and polyphagia are common. Muscle wasting or weakness, a pot-bellied appearance (from hepatomegaly, fat redistribution, and weakened abdominal muscles), increased susceptibility to infection (conjunctival, skin, urinary tract, lung), excessive panting, and variable behavioral or neurologic signs (expanding pituitary tumor) are often present.

**Top Differentials**

Differentials include other causes of endocrine alopecia, follicular dysplasia, alopecia X, superficial pyoderma, demodicosis, and dermatophytosis.

**Diagnosis**

**1.** Hemogram: neutrophilia, lymphopenia, and eosinopenia are often seen

**2.** Serum biochemistry panel: an elevated alkaline phosphatase enzyme level is typical (90% of cases). There may also be mildly to markedly elevated alanine transaminase activity, as well as elevated cholesterol, triglyceride, or glucose levels

**3.** Urinalysis: the specific gravity is usually low, and there may be bacteriuria, proteinuria, or glucosuria. Subclinical urinary tract infections are common

**4.** Urine cortisol/creatinine ratio: usually elevated. A nonspecific screening test that is not diagnostic by itself because false-positive results are common (stress-induced, seen with many other illnesses). To minimize the effects of stress, a home-collected urine sample should be used, instead of one obtained at the veterinary hospital

**5.** Dermatohistopathology: often shows nondiagnostic changes consistent with any endocrinopathy. Dystrophic mineralization (calcinosis cutis), thin dermis, and absent erector pili muscles are highly suggestive of hyperadrenocorticism, but these changes are not always present

**6.** Abdominal ultrasonography: may demonstrate adrenal hyperplasia or tumor

**7.** Computed tomography (CT) or magnetic resonance imaging (MRI): may detect a pituitary mass

**8.** Adrenal function tests:

n ACTH stimulation test (cortisol): an exaggerated poststimulation cortisol level is highly suggestive of endogenous hyperadrenocorticism, but false-negative and false-positive results can occur. In iatrogenic cases, an inadequate response to ACTH stimulation is typical. *Note*: Reconstituted cosyntropin (ACTH solution) can be stored frozen at -20°C in plastic syringes for up to 6 months with no adverse effects on its bioactivity

n ACTH stimulation test (17-hydroxyprogesterone): exaggerated basal and poststimulation 17-hydroxyprogesterone levels may be seen in endogenous hyperadrenocorticism, but false-negative and false-positive results can occur. 17-Hydroxyprogesterone, a progestin, is an adrenal gland–produced precursor of cortisol

n Low-dose (0.01mg/kg) dexamethasone suppression test: inadequate cortisol suppression is highly suggestive of endogenous hyperadrenocorticism, but false-negative and false-positive results can occur. Suppression at 4 hours followed by escape from suppression at 8-hour sampling is characteristic of PDH

n High-dose (0.1mg/kg) dexamethasone suppression test: used to help differentiate between adrenal neoplasia and pituitary-dependent hyperadrenocorticism. A lack of cortisol suppression is suggestive of adrenal neoplasia, whereas cortisol suppression suggests pituitary disease

n Endogenous ACTH assay: used to help differentiate between adrenal neoplasia and pituitary-dependent hyperadrenocorticism. An elevated ACTH level is suggestive of pituitary disease, whereas a depressed ACTH level is suggestive of adrenal neoplasia

**Treatment and Prognosis**

**1.** Any concurrent infections (e.g., pyoderma, demodicosis, urinary tract infection) should be treated with appropriate therapies. 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 disinfect the skin and ear canals, preventing the secondary infections from recurring.

**2.** Treatment of choice for **iatrogenic Cushing's** cases is to progressively taper, then discontinue glucocorticoid therapy.

**3.** Treatment of choice for **adrenal neoplasia** is adrenalectomy.

1. Dogs with inoperable adrenal tumors or metastases may benefit from mitotane or trilostane therapy.

n Mitotane for adrenal tumors: One should give 50mg/kg PO every 24 hours with food for 7 to 14 days. An ACTH stimulation test is performed every 7 days. If inadequate cortisol suppression persists, increase the mitotane dosage to 75 to 100mg/kg/day for an additional 7 to 14 days, monitoring with ACTH stimulation tests weekly. When adequate adrenal suppression is demonstrated, maintenance mitotane therapy is initiated as described below

**4.** The traditional (historic) medical treatment of choice for **PDH** is mitotane 50mg/kg PO administered every 24 hours with food. The daily dosage is continued until the basal serum or plasma cortisol level normalizes and does not increase following ACTH stimulation. Control is usually achieved within 5 to 10 days of initiation of therapy, so the patient should be closely monitored with ACTH stimulation tests performed every 7 days. Monitoring water and food intake before and during induction may be useful. Water and food intake often markedly decreases when adequate adrenal suppression has been achieved. If signs of adrenal insufficiency (e.g., anorexia, depression, vomiting, diarrhea, ataxia, disorientation) develop, mitotane therapy should be stopped and hydrocortisone 0.5 to 1.0mg/kg PO every 12 hours administered, until symptoms resolve.

To maintain remission following mitotane induction, mitotane PO with food 50mg/kg administered once weekly, or 25mg/kg twice weekly. Dogs that relapse during maintenance therapy should be reinduced with daily mitotane for 5 to 14 days or until recontrolled, then maintained with 62 to 75mg/kg once weekly, or 31 to 37.5mg/kg twice weekly. A great deal of patient variability occurs, requiring close monitoring.

5. A more recent treatment option and the current recommendation for the medical treatment of **PDH** is trilostane. At this writing, its optimal dosing regimen has not yet been determined, but many investigators are using the following protocol:

n Dogs <5kg: give 30mg PO with food q 24 hours

n Dogs between 5 and 20kg: give 60mg PO with food q 24 hours

n Dogs between 20 and 40kg: give 120mg PO with food q 24 hours

n Dogs >40kg: give 240mg PO with food q 24 hours

  Assess efficacy by monitoring clinical signs and evaluating results of ACTH stimulation tests 10 days, 4 weeks, and 12 weeks after the start of therapy, then every 3 months thereafter. ACTH stimulation tests should be performed 4 to 6 hours after trilostane dosing. A post-ACTH cortisol level <150 nmol/L (but >20nmol/L) is usually consistent with good control. However, optimal clinical control has also been reported with post-ACTH cortisol concentrations between 150 and 250 nmol/L, so blood work results should always be interpreted alongside clinical signs. If the dog is not clinically well controlled and post-ACTH cortisol concentrations are >150 nmol/L, the dose of trilostane should be increased. Dose adjustments should be made in increments of 20 to 30mg/dog. A wide range of trilostane doses to induce and maintain remission have been reported in dogs, with the therapeutic dose for most dogs being between 4 and 20mg/kg/day. Some dogs may require twice-daily dosing if duration of effect is inadequate. Clinical signs such as polydipsia/polyuria/polyphagia often start to improve within the first 10 days of treatment, but alopecia and other skin changes may take 3 or more months to improve. If signs of adrenal insufficiency (depression, inappetence, vomiting, diarrhea) develop at any time during therapy, or if post-ACTH cortisol concentrations (measured 4‑6 hours after trilostane dosing) are <20 nmol/L, trilostane should be stopped for 5 to 7 days, then reinstituted at a lower dose. *Note*: Although trilostane appears to be well tolerated by most dogs, sudden death has been reported in dogs with concurrent heart problems. Trilostane is also contraindicated in pregnant and lactating dogs, dogs with primary hepatic disease, and dogs with renal insufficiency.

**6.** Other alternative, but less consistently successful, medical treatments for PDH include the following:

Ketoconazole 15mg/kg PO with food q 12 hours

or Selegiline (L-deprenyl) 1‑2mg/kg PO q 24 hours

**7.** An effective treatment (where available) for PDH is microsurgical transsphenoidal hypophysectomy. This procedure requires a highly skilled neurosurgeon and specialized veterinary facilities that have access to advanced pituitary imaging techniques. Postoperative complications may include hypernatremia, keratoconjunctivitis sicca, diabetes insipidus, and secondary hypothyroidism.

8. For calcinosis cutis, adjunctive topical treatment with dimethyl sulfoxide (DMSO) gel every 24 hours may help resolve the lesions. During DMSO therapy, serum calcium levels should be monitored periodically because hypercalcemia is a potential adverse effect of this treatment.

**9.** The prognosis ranges from good to poor, depending on the cause and severity of the disease, with the average survival time for dogs with PDH being approximately 2.5 years after diagnosis.

**#8 Acral Lick Dermatitis** (lick granuloma)

**Features**

Acral lick dermatitis is first noted as excessive, compulsive licking at a focal area on a limb, resulting in a firm, proliferative, ulcerative, alopecic lesion. The causes of the licking are multifactorial, and, although environmental stress (e.g., boredom, confinement, loneliness, separation anxiety) may be a contributor, other factors are usually more important (Box 13-1). This dermatitis is common in dogs, with the highest incidence in middle-aged to older, large-breed dogs, especially Doberman pinschers, Great Danes, Golden retrievers, Labrador retrievers, German shepherds, and Boxers.

The lesion usually begins as a small area of dermatitis that slowly enlarges because of persistent licking. The affected area becomes alopecic, firm, raised, thickened, and plaquelike to nodular, and it may be eroded or ulcerated. With chronicity, extensive fibrosis, hyperpigmentation, and secondary bacterial infection are common. Lesions are usually single but may be multiple, and they are most often found on the dorsal aspect of the carpus, metacarpus, tarsus, or metatarsus.

**Top Differentials**

Differentials include demodicosis, dermatophyte kerion, fungal or bacterial granuloma, and neoplasia.

**Diagnosis**

**1.** Usually based on history, clinical findings, and ruling out other differentials

Box 13-1

Underlying Causes of Acral Lick Dermatitis

 Hypersensitivity (atopy, food)

 Fleas

 Trauma (cut, bruise)

 Foreign body reaction

 Infection (bacterial, fungal)

 Demodicosis

 Hypothyroidism

 Neuropathy

 Osteopathy

 Arthritis

**2.** Dermatohistopathology: ulcerative and hyperplastic epidermis, mild neutrophilic and mononuclear perivascular dermatitis, and varying degrees of dermal fibrosis

**3.** Bacterial culture (exudates, biopsy specimen): *Staphylococcus* is often isolated. Mixed gram-positive and gram-negative infections are common

**Treatment and Prognosis**

**1.** The underlying causes should be identified and corrected (see Box 13-1).

**2.** One should treat for secondary bacterial infection with long-term systemic antibiotics (minimum, 6-8 weeks, and as long as 4-6 months in some dogs). Antibiotic therapy should be continued at least 3 to 4 weeks beyond regression of the lesion. The antibiotic should be selected according to bacterial culture and sensitivity results.

**3.** Anecdotal reports suggest good efficacy with combined antibiotic, amitriptyline (2mg/kg q 12 hours), and hydrocodone (0.25mg/kg q 8-12 hours) administered until lesions resolve. Then, one drug should be discontinued every 2 weeks until it can be determined which drug (if any) may be required for maintenance therapy.

**4.** Topical applications of analgesic, steroidal, or bad-tasting medications every 8 to 12 hours may help stop the licking but response is unpredictable and often disappointing.

**5.** When no underlying cause can be found, treatment with behavior-modifying drugs may be beneficial in some dogs (Table 13-1). Trial treatment periods of up to 5 weeks should be used until the most effective drug is identified. Lifelong treatment is often necessary.

6. Alternative medical treatments such as cold laser therapy or acupuncture have been beneficial in some patients.

**7.** Mechanical barriers such as wire muzzles and bandaging, Elizabethan collars, and side braces may be helpful.

**8.** Surgical excision or laser ablation is not recommended because postoperative complications, especially wound dehiscence, are common. Laser ablation may help sterilize the lesion and deaden nerve endings; however, response is highly variable.

**9.** The prognosis is variable. Chronic lesions that are unresponsive or extensively fibrotic and those for which no underlying cause can be found have a poor prognosis for resolution. Although this disease is rarely life threatening, its course may be intractable.

Table 13-1

Drugs for Psychogenic Dermatoses in Dogs

**Drug Dose**

**Anxiolytics**

Phenobarbital 2-6 mg/kg PO q 12 hours

Diazepam 0.2 mg/kg PO q 12 hours

Hydroxyzine 2.2 mg/kg PO q 8 hours

**Tricyclic Antidepressants**

Fluoxetine 1 mg/kg PO q 24 hours

Amitriptyline 1-3 mg/kg PO q 12 hours

Imipramine 2-4 mg/kg PO q 24 hours

Clomipramine 1-3 mg/kg PO q 24 hours

**Endorphin Blocker**

Naltrexone 2 mg/kg PO q 24 hours

**Endorphin Substitute**

Hydrocodone 0.25 mg/kg PO q 8 hours

**Topical Products**

Fluocinolone acetonide

   flunixin meglumine

Deep Heet  Bitter Apple

#9 Canine Scabies (sarcoptic mange)

Features

Canine scabies manifests as a disease that is caused by *Sarcoptes scabiei* var. *canis*, a superficial burrowing skin mite. Mites secrete allergenic substances that elicit an intensely pruritic hypersensitivity reaction in sensitized dogs. Canine scabies is common in dogs. Affected dogs often have a previous history of being in an animal shelter, having contact with stray dogs, or visiting a grooming or boarding facility. Wildlife such as fox and coyotes are often the source of initial infection and possible repeated infections. In multiple-dog households, more than one dog is usually affected.

Canine scabies is a nonseasonal intense pruritus that responds only variably to corticosteroids. Lesions include papules, alopecia, erythema, crusts, and excoriations. Initially, less-hairy skin is involved, such as on the hocks, elbows, pinnal margins, and ventral abdomen and chest. With chronicity, lesions may spread over the body, but the dorsum of the back is usually spared. Peripheral lymphadenomegaly is often present. Secondary weight loss may occur. Heavily infested dogs may develop severe scaling and crusting. Some dogs may present with intense pruritus but no or minimal skin lesions. Although they are less common, asymptomatic carrier states are possible in dogs; however, in multiple-dog households, a range of symptoms (severe to nonpruritic) may exist..

Top Differentials

Differentials include hypersensitivity (food, atopy, flea), *Malassezia* dermatitis, pyoderma, demodicosis, dermatophytosis, and contact dermatitis.

Diagnosis

1. History, clinical findings, and response to scabicidal treatment

2. Pinnal-pedal reflex: rubbing of the ear margin between thumb and forefinger may elicit a scratch reflex. This reflex is highly suggestive of a scabies infection with approximately 80% accuracy.

3. Microscopy (superficial skin scrapings): detection of sarcoptic mites, nymphs, larvae. or ova. False-negative results are common because mites are extremely difficult to find; approximately 20% accurate.

4. Serology (enzyme-linked immunosorbent assay [ELISA]): detection of circulating immunoglobulin (Ig)G antibodies against *Sarcoptes* antigens. This is a highly specific and sensitive test, but false-negative results can occur in young puppies and in dogs receiving corticosteroid therapy. Also, false-positive results may be seen in dogs that have been successfully treated for scabies because detectable antibodies may persist for several months after treatment cessation

5. Dermatohistopathology (usually nondiagnostic): varying degrees of epidermal hyperplasia and superficial perivascular dermatitis with lymphocytes, mast cells, and eosinophils. Mite segments are rarely found within the stratum corneum

Treatment and Prognosis

1. Affected and all in-contact dogs should be treated with a scabicide. Failure to treat all dogs results in reinfection and persistent pruritus.

2. 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 antimicrobial shampoo every 3-7 days will help speed resolution and enhance the mitacidal treatments.

5. Systemic treatments are the most effective due to the accurate dosing and better compliance. Effective systemic treatments include the following:

\*Selamectin applied 6 to 12mg/kg every 2 weeks at least four times may be more effective.

 Ivermectin 0.2‑0.4mg/kg PO q 7 days, or SC q 14 days, for 4‑6weeks

\* Doramectin 0.2-0.6 mg/kg SQ q 7 days for 4-6 weeks.

 Milbemycin oxime 0.75mg/kg PO q 24 hours for 30 days, or 2mg/kg PO q 7 days for 3‑5 weeks

  Moxidectin applied every 2-4 weeks for 4-6 weeks; frequent application may lead to increased adverse effects.

6. Topical treatments may be effective but due to the poorer compliance, treatment failures are more common. Effective topical products include the following:

 \* 0.025%‑0.03% amitraz solution applied to the entire body three times at 2-week intervals, or once weekly for 2‑6 weeks

  Fipronil spray 3 mL/kg, applied as pump spray to the entire body three times at 2-week intervals, or 6 ml/kg applied as sponge-on once weekly for 4-6 weeks

 2%‑3% lime sulfur solution

 Organophospates (malathion, phosmet, mercaptomethyl phtalimide). Organophosphates are the most toxic and least effective therapies available

5. If the animal is severely pruritic and mites have been identified, steroids may be used for the first 2 to 5 days of scabicidal treatment may be helpful. Use of steroids without the finding of mites makes it impossible for the practitioner to determine response to scabicidal therapy.

6.. In kennel situations, bedding should be disposed of and the environment thoroughly cleaned and treated with parasiticidal sprays.

8. The prognosis is good. *S scabei* is a highly contagious parasite of dogs that can also transiently infest humans and, rarely, cats. Reinfection can occur leading to chronic pruritic disease.

Author’s Note:

Scabies infections can very closely mimic food allergy and atopy.

The pinnal-pedal reflex (ear scratch test) is the easiest and most suggestive test for scabies.

If reinfection is suspected, long-term scabicidal therapy may be beneficial unless the source can be identified and treated.

# 10 Dermatophytosis (ringworm)

Features

Dermatophytosis is an infection of hair shafts and stratum corneum caused by keratinophilic fungi. It occurs commonly in dogs and cats, with highest incidences reported in kittens, puppies, immunocompromised animals, and long-haired cats. Persian cats and Yorkshire and Jack Russell terriers appear to be predisposed.

Skin involvement may be localized, multifocal, or generalized. Pruritus, if present, is usually minimal to mild but occasionally may be intense. Lesions usually include areas of circular, irregular, or diffuse alopecia with variable scaling. Remaining hairs may appear stubbled or broken off. Other symptoms in dogs and cats include erythema, papules, crusts, seborrhea, and paronychia or onychodystrophy of one or more digits. Rarely, cats present with miliary dermatitis or dermal nodules (see “Dermatophytic Granulomas and Pseudomycetomas”). Other cutaneous manifestations in dogs include facial folliculitis and furunculosis resembling nasal pyoderma, kerions (acutely developing, alopecic, and exudative nodules) on the limb or face, and truncal dermal nodules (see “Dermatophytic Granulomas and Pseudomycetomas”). Asymptomatic carrier states (subclinical infection) are common in cats, especially among long-haired breeds. Asymptomatic disease, although rare in dogs, has been reported in Yorkshire terriers.

Top Differentials

Dogs

Differentials in dogs include demodicosis and superficial pyoderma. If nodular, neoplasia and acral lick dermatitis should be included.

Cats

Differentials in cats include parasites, allergies, and feline psychogenic alopecia.

Diagnosis

1. Rule out other differentials

2. Ultraviolet (Wood’s lamp) examination: hairs fluoresce yellow-green with some *Microsporum canis* strains. This is an easy screening test, but false-negative and false-positive results are common

3. Trichogram (hairs or scales in potassium hydroxide preparation): Search for hair shafts infiltrated with hyphae and arthrospores. Fungal elements are often difficult to find

4. Dermatohistopathology: variable findings may include perifolliculitis, folliculitis, furunculosis, superficial perivascular or interstitial dermatitis, epidermal and follicular orthokeratosis or parakeratosis, or suppurative epidermitis. Fungal hyphae and arthrospores in stratum corneum or hair shafts.

5. Fungal culture: *Microsporum* or *Trichophyton* spp

6. PCR analysis may simplify the diagnosis where available.

Treatment and Prognosis

 1. If the lesion is focal, a wide margin should be clipped around it and topical antifungal medication applied every 12 hours until the lesion resolves. (Some dermatologists believe that clipping spreads lesions onto animals and further contaminates the environment.) Effective topicals for localized treatment include products that contain the following:

  terbinafine cream

  clotrimazole cream, lotion, or solution

  enilconazole cream

  ketoconazole cream

  miconazole cream, spray, or lotion

 2. If response to localized treatment is poor, the animal should be treated for generalized dermatophytosis.

 3. Topical antifungal rinse or dip should be applied to the entire body one or two times per week (minimum 4‑6 weeks) until follow-up fungal culture results are negative. Bathing the animal with a shampoo that contains chlorhexidine **and** (miconazole or ketoconazole) immediately preceding the antifungal dip may be helpful. Dogs with generalized dermatophytosis may be cured with topical therapy alone, whereas cats almost always require concurrent systemic therapy. Effective topical antifungal solutions include the following:

 Enilconazole 0.2% solution

 Lime sulfur 2%‑4% solution

1. For cats with dermatophytosis and dogs that are unresponsive to topical therapy alone, topical therapy for generalized infection should be combined with long-term systemic antifungal therapy and continued until 3 to 4 weeks beyond negative follow-up fungal culture results. The average duration of therapy is 8-12 weeks.

 Effective systemic antifungal drugs include the following:

 Terbinafine 30‑40mg/kg PO q 24 hours

 Ketoconazole 10mg/kg PO q 24 hours with food (dogs)

 Fluconazole 10mg/kg PO q 24 hours with food.

 Itraconazole (Sporonox) 5‑10mg/kg PO q 24 hours with food

Less effective systemic antifungal drugs include the following:

 Microsized griseofulvin at least 50mg/kg/day PO with fat-containing meal

 Ultramicrosized griseofulvin 5‑10mg/kg/day PO with fat-containing meal

 5. Alternatively, pulse therapy may be almost as effective and multiple protocols have been published using various drugs. Pulse treatments should be continued until two consecutive follow-up fungal cultures taken 2 to 4 weeks apart are negative.

 6. All infected animals, including asymptomatic carriers, should be identified and treated. Exposed, noninfected cats and dogs should be treated prophylactically with weekly topical antifungal rinse or dip for the duration of treatment of the infected animals.

 7. The environment should be thoroughly cleaned by removing all contaminated materials and disinfected with bleach (vacuums may further contaminate the environment).

 8. For endemic infections involving multianimal homes, catteries, or animal facilities, treatment should be provided according to the recommendations outlined in Box 4-1.

9. Lufenuron has not demonstrated consistent efficacy in treating or preventing infection.

10. The prognosis is generally good, except for endemically infected multicat households and catteries. Animals with underlying immunosuppressive diseases also have a poorer prognosis for cure. Dermatophytosis is contagious to other animals and to humans.

Box 4-1

Treating Dermatophytosis in Multianimal Homes, Catteries, and Animal Facilities

 Culture all animals to determine the extent and location of animal infections.

 Culture the environment (cages, counters, furniture, floors, fans, ventilation units, etc) to map the infected areas to be disinfected.

 Treat all infected animals with systemic antifungals until each animal has two negative fungal cultures taken at least 1 month apart.

 Treat all infected and exposed animals with topical 2% to 4% lime sulfur solution every 3 to 7 days to prevent contagion and zoonosis. Continue until all animals have two negative fungal cultures taken at least 1 month apart. Do not clip cats as this contaminates the clippers and facility and worsens the risk of contagion.

 Dispose of all infected material. Remove any clutter from animal facilities or other infected areas.

 Clean and disinfect all surface areas every 3 days. Continue until all animals have two negative fungal cultures taken at least 1 month apart. Enilconazole (Clinafarm EC disinfectant, American Scientific Laboratories, Union, NJ) is a very effective environmental disinfectant, but it is licensed only for poultry farm use in the United States. Household chlorine laundry bleach (5% sodium hypochlorite) diluted 1:10 in water is an effective, inexpensive environmental disinfectant.

BOX

Author’s Note:

*\*\* Microsporum canis* is one of the most common zoonotic diseases in veterinary medicine.

\*\* Adopted kittens should be screened for infection during the first veterinary wellness visit.

\*\* Chronicly infected animals likely have contaminated the home requiring aggressive cleaning and disinfection of the environment.

\*\* Even long-standing and severe infections can be resolved with aggressive and persistent treatments.

\*\* The discontinuation of therapy MUST be based on negative cultures.

#11 Malasseziasis

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 (100). 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.