

Superficial pyoderma

Definition/overview Superficial pyoderma is a bacterial infection of the superficial epidermis of the hair follicles.

Etiology The most common causes of superficial pyoderma are endocrinopathies, hypersensitivities, and ectoparasites. *Staphylococcus intermedius* is the most frequently isolated bacteria; *S. aureus* may also be found.

Pathophysiology Changes to the microenvironment lead to the development of conditions favoring the growth of pathogenic staphylococci. Inflammatory reactions to bacterial products can lead to further multiplication of bacteria.

Clinical presentation Superficial pyoderma is extremely common in canine patients presented for skin disease. Because the clinical signs are often similar to the circular lesions of ringworm in people, dogs with superficial pyoderma (32) are often mistakenly treated for ringworm (dermatophytosis). Lesions of circular alopecia, a moth-eaten appearance to the haircoat, epidermal collarettes, honey-colored crusts, papules, pustules, macules, and crusted plaques are typical for superficial pyoderma in the dog (33).

Differential diagnosis Demodicosis, dermatophytosis, pemphigus foliaceus, dermatophilosis, *Malassezia pachydermatis* infection.

Diagnosis Diagnosis is initially based on the identification of lesions. Neutrophils and intracellular cocci may be identified from cytologic examination of pustule contents. Bacterial culture and sensitivity of an intact pustule or sterile tissue sample should identify the pathogen.

Management After initial suspicion of pyoderma and appropriate antibiotic therapy for a minimum of 3–4 weeks, the patient is re-examined while receiving antibiotics with antibiotics continued one week past lesion resolution. In cases of recurrent pyoderma, antibiotics should be continued for a minimum of 4–8 weeks, with re-examination determining when treatment can be stopped. It is important to discern a true recurrence of pyoderma from a non-response based on re-examining the patient while it is receiving antibiotics and prior to discontinuing antibiotics. If the pyoderma responds, but quickly relapses once antibiotics are discontinued, antibiotic therapy was not continued for long enough. Corticosteroids are not recommended in the initial management of pyoderma for three reasons:

- It is important to assess the degree of residual pruritus once the pyoderma has cleared.
- Corticosteroids may contribute to pyoderma for several months after discontinuation. Relapses are more common and more severe with corticosteroid usage.
- Corticosteroids will mask the clinical signs of pyoderma, not allowing adequate assessment of its resolution, and may cause secondary problems.

To minimize the recurrence of pyoderma, the predisposing cause(s) must, if possible, be identified and treated.

Impetigo (puppy pyoderma)

Definition/overview Impetigo is a subcorneal pustular disease of prepubertal dogs.

Etiology Impetigo is often associated with poor husbandry conditions. *Staphylococcus* spp. are usually isolated from subcorneal pustules.

Pathophysiology Impetigo is not contagious and may occur for no apparent reason. In some young dogs it may be associated with parasitism, poor nutrition, or infectious diseases.

Clinical presentation Nonfollicular pustules are localized to the sparsely haired skin of the ventral abdomen and occasionally the axilla in puppies aged 2–9 months (34). Ruptured pustules appear as small yellowish crusts or epidermal collarettes. Pruritus may be absent.

Differential diagnosis Contact irritant dermatitis, demodicosis, dermatophytosis, food allergy.

Diagnosis Diagnosis is based on the identification of the cocci from impression smears of the pustule contents.

Management Impetigo is considered a self-limiting disease and usually requires only topical antibacterial baths combined with general health care. Severe or persistent cases may benefit from systemic antibiotics for 10–14 days. Cultures should be performed if antibacterial treatment fails.

Deep pyoderma and furunculosis

Definition/overview Deep pyoderma is a bacterial infection of the dermis, which may result from the extension of infection through the walls of ruptured follicles (furunculosis).

Etiology Deep pyoderma and furunculosis are the result of a hair follicle rupture into the dermis with subsequent liberation of keratin. This liberated keratin acts as a foreign body within the dermis to perpetuate the cycle of inflammation. Underlying causes of deep pyoderma include flea bite hypersensitivity, demodicosis, hypothyroidism, hyperadrenocorticism, immune system abnormality, and idiopathy.

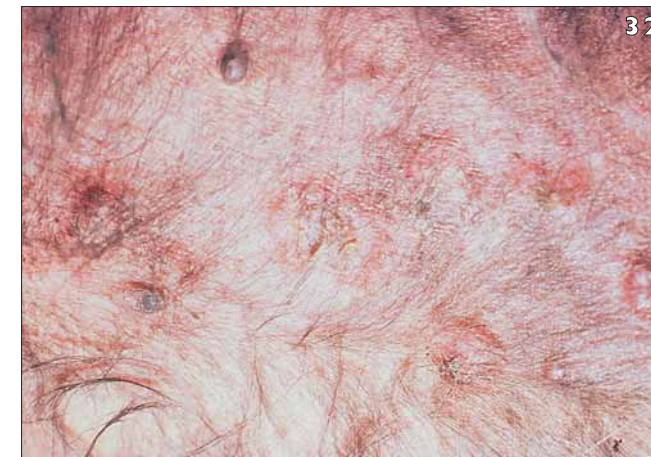
Pathophysiology Hemorrhagic bullae, ulcers, fibrosis, scarring, and cellulitis with draining, fistulous tracts may result (35).

Clinical presentation Malaise, inappetence, fever, and lymphadenopathy may be present. Regionalized furunculosis can be noted on the chin (canine chin acne) and at callus, pressure point, or interdigital areas. Due to their stubby and bristly hair shafts, short-coated breeds of dogs may be more prone to a generalized furunculosis.

Differential diagnosis Demodicosis, nocardiosis, panniculitis (infectious or sterile), deep fungal infection, mast cell tumor, foreign body.

Diagnosis Sterile preparation for culture of tissue or an unopened bulla is required to determine a causative agent. Furunculosis is more often associated with *Staphylococcus intermedius* but occasionally gram-negative bacteria may be isolated alone.

Management In mixed staphylococcal and gram-negative infections, treating the staphylococci and ignoring the gram-negative bacteria is usually adequate. Systemic antibiotics are often required for 3–6 months and longer, as lesions may relapse if they are stopped. Occasionally, lesions may be sterile. Underlying diseases must be investigated. German Shepherd Dogs can develop a severe, deep pyoderma of the hindlimbs and dorsal lumbosacral area. Immunotherapy may be helpful.



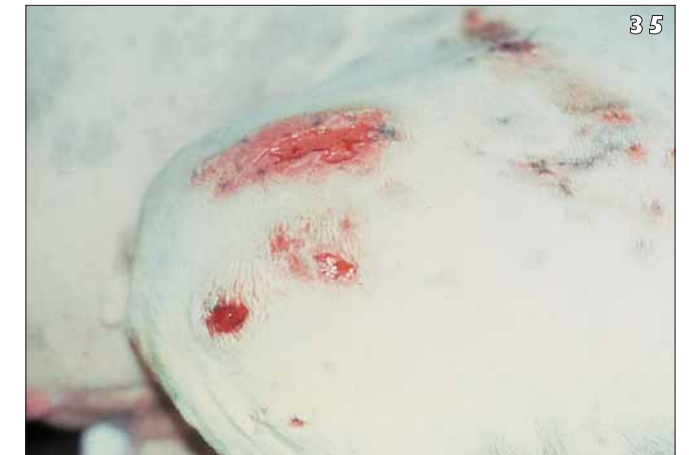
32 Circular erythema with a crust in the center in a dog with superficial pyoderma.



33 Circular areas of alopecia in a Shar Pei with superficial pyoderma due to atopic dermatitis.



34 Large, nonfollicular pustules in a puppy with impetigo. (Photo courtesy of CS Foil, DVM.)



35 Ulcers and draining tracts in a Pit Bull Terrier with deep pyoderma.

Abscesses and cellulitis

Definition/overview An abscess is a localized collection of purulent material in the dermis or subcutaneous tissues. Cellulitis is more extensive and often dissects through tissue layers.

Etiology Oral or epidermal flora are introduced by inoculation during penetration of the skin by teeth or claws. In dog bites, typical pathogens are *Staphylococcus aureus* and *Escherichia coli* whereas in cat bites these are *Pasteurella multocida*, β -hemolytic streptococci, and *Bacteroides* spp. Cellulitis may develop secondary to demodicosis in dogs.

Pathophysiology Abscess formation results from tissue damage and local infection 2–4 days after a traumatic wound when the wound site becomes promptly sealed.

Clinical presentation Subcutaneous abscesses and cellulitis are common in cats, especially intact males. Abscesses are most often present on the face, legs, base of the tail, or back, and are the result of cat bite wounds. Symptoms of fever, lameness, depression, or pain may be noted.

Differential diagnosis Penetrating foreign body, panniculitis, nocardiosis or actinomycosis, subcutaneous and deep mycoses, feline leprosy and opportunistic mycobacterial infection, canine demodicosis, *Rhodococcus* or L-form bacteria, neoplasia, cuterebriasis or dracunculiasis, dermatophytic mycetoma.

Diagnosis A complete history and examination of the site of the abscess usually lead to the diagnosis. Recurrent formation of abscesses necessitates a more thorough investigation to determine the cause. If an underlying immunosuppression or endocrinopathy is suspected, appropriate tests should be conducted. Other tests include cytologic examination of discharge and fungal, mycobacterial, and bacterial culture and sensitivity.

Management Adequate drainage and antibiotics resolve cat bite abscesses in 10–14 days. Any abscess that does not heal or recurs should be investigated for opportunistic mycobacteria, especially if present in the inguinal or lumbar area (36). Deep tissue wedges (which includes subcutaneous fat) should be submitted promptly for mycobacterial culture. Additionally, aerobic, anaerobic, and fungal culture of tissue may be indicated in recurrent cases.



36 Punctate ulcers and draining tracts in the inguinal area of a ten-year-old DSH cat with panniculitis due to *Mycobacterium fortuitum*.

FUNGAL AND YEAST INFECTIONS

Dermatophytosis

Definition/overview Dermatophytosis is a fungal infection of the skin, hair, or claw.

Etiology Dermatophytosis is caused by infection with species of keratinophilic fungi. In cats the fungus commonly implicated is *Microsporum canis* and in dogs it is *M. canis* and *M. gypseum*. Other common fungal agents include *Trichophyton* spp.

Pathophysiology A cell-mediated and humoral response is elicited after infection. The inflammatory reaction leads to increased epidermal proliferation. Persian cats may develop a nodular phase of deep furunculosis.

Clinical presentation In cats, lesions may range from alopecia, scale, miliary dermatitis, or nodules (37) to none at all (asymptomatic carriers). In dogs, lesions may include papules, pustules, and draining tracts. Dermatophytosis is more common in very young or old animals, immunosuppressed animals, and in cattery situations.

Differential diagnosis

- Regional/generalized lesions. In cats: flea bite hypersensitivity, telogen/anagen defluxion, psychogenic alopecia. In dogs: demodicosis, superficial pyoderma, immune-mediated diseases, deep mycotic lesions.
- Focal lesions. In cats: cat bite abscess, cheyletiellosis, demodicosis. In dogs: superficial pyoderma, demodicosis, defects in keratinization, alopecia after injection.



37 Erythematous plaques and nodules in a one-year-old Persian cat with generalized dermatophytosis due to *Microsporum canis*.

Diagnosis Although not a common disease, dermatophytosis is an overdiagnosed disease when clinical signs or color change on dermatophyte test medium (DTM) alone are used. Once hair or scale applied to fungal culture media exhibits nonpigmented colony growth concomitant with red color change, the colony MUST be identified microscopically to confirm the presence of a dermatophyte. Nondermatophytes may cause the red color change similar to a dermatophyte (24). Identification of the dermatophyte assists the veterinarian, the client, and the patient in the following ways:

- It avoids a false-positive diagnosis of dermatophytosis and unnecessary treatment.
- The source of the dermatophyte may be identified and removed or treated, and the potential need for environmental decontamination can be evaluated.
- Along with signalment and clinical presentation of the patient, dermatophyte identification guides the selection of appropriate therapy – systemic and topical or topical alone.
- Culture of hair on DTM is recommended for all cats presented for skin disease and for all dogs with evidence of primary lesions. Culture of tissue is recommended when fungal or hyphal structures present in tissue cannot be readily identified on histopathologic examination and culture of hair or scale is negative.

Management Clipping away affected hair and burning the clippings should be performed in all cases of generalized dermatophytosis and dermatophytosis in longhaired cats to decrease environmental and human exposure. In these situations topical treatments should be used with systemic antifungal agents. Griseofulvin is the treatment of choice, although itraconazole, ketoconazole, and lufenuron have success as well. Treatment should be continued for at least six weeks and until two or three fungal cultures are negative. In cases of *M. canis* infection, owners should be advised to vacuum the animal's environment daily and to disinfect cages and other surfaces with bleach.

Malassezia dermatitis

Definition/overview *Malassezia* dermatitis is a pruritic condition associated with the presence of the yeast *Malassezia pachydermatis*.

Etiology Increased numbers of yeast organisms or a hypersensitivity to surface yeast may be associated with diseases that may induce seborrheic conditions on the skin. Predisposing factors for *Malassezia* dermatitis include: hypothyroidism; flea bite hypersensitivity; food hypersensitivity; atopic dermatitis; superficial pyoderma; primary keratinization disorders; long-term antibiotic or glucocorticoid therapy; and breed (Terriers, Basset Hound, Poodle, American Cocker Spaniel, Shih Tzu and Lhasa Apso, German Shepherd Dog).

Pathophysiology The precise pathogenesis is unclear, although *M. pachydermatis* thrives in areas of skin with increased lipid content and may be more prevalent in geographic regions where humidity is high.

Clinical presentation Moist, erythematous, hyperpigmented, lichenified lesions typify those of *Malassezia* dermatitis and are often located in the ventral neck fold, axilla, lip fold, ears, claw folds, and interdigital spaces (38). Pruritus is present and often constant. *Malassezia* dermatitis in cats may be associated with otitis externa, feline acne, generalized keratinization defects, FIV, thymoma, and exfoliative erythroderma.

Differential diagnosis Demodicosis, atopic dermatitis, food hypersensitivity, sarcoptic mange, superficial pyoderma, idiopathic defects in keratinization.

Diagnosis Peanut-shaped budding yeast are visible on acetate tape preparations from affected skin (25).

Management Topical degreasing and antifungal products may temporarily clear the disorder; oral ketoconazole (5–10 mg/kg q24–12h for 2–4 weeks), itraconazole, or fluconazole may be necessary. It is important to investigate and correct underlying diseases as mentioned above to minimize recurrence.



38 Erythematous pododermatitis due to *Malassezia pachydermatis* in a ten-year-old Poodle.

PARASITIC DERMATOSES

Canine demodicosis

Definition/overview Demodicosis, an intrafollicular parasitic disease caused by demodicid mites, is probably the most serious non-neoplastic dermatologic condition in animals.

Etiology Demodicosis is caused by mites of the genus *Demodex*. A hereditary factor predisposes an animal to develop juvenile-onset demodicosis.

Pathophysiology Typically the mites reside in the hair follicles, although some have been found in apocrine and sebaceous glands adjacent to follicles. Mites feed mostly on follicular debris and cells and occasionally on sebum. It seems that lymphocyte suppression, possibly influenced by secondary bacterial infection, allows the mites to proliferate.

Clinical presentation Lesions comprise one or several areas of either scaling, thinning of hair, hyperpigmentation, alopecia, or erythema with alopecia (39). Lesions may appear on any part of the body but typically affect the face and forelimbs. About 10% of localized cases progress to generalized disease. Juvenile-onset demodicosis is limited to onset <18–24 months of age and is considered hereditary. Adult-onset demodicosis occurs after two years of age and is often associated with an underlying disease (iatrogenic Cushing's, hypothyroidism, infectious diseases).

Differential diagnosis Color dilute alopecia, alopecia areata, sebaceous adenitis, deep or superficial pyoderma, injection site reaction, deep mycotic infection, cutaneous T-cell lymphoma, pemphigus foliaceus, drug eruption, zinc-responsive dermatosis, dermatophytosis, *Malassezia* dermatitis, endocrine disorders.

Diagnosis Determining the extent of disease in demodicosis is one of the most important steps in diagnosis. Acquiring deep skin scrapings from five body sites is helpful in determining the extent of disease (lipfold, fore and hind foot, two additional lesions) (Table 3). Noting which life cycle stages are present

and their relative numbers can give the veterinarian an idea as to the activity of the disease (40) (numerous eggs, few adults – active disease; numerous dead adults, no eggs – less active disease, potentially a better prognosis).

Generalized disease involves positive skin scrapings from more than one body region. Localized disease is limited to a few lesions in one body region. Skin scrape sites can be re-scraped biweekly to assess response to therapy. A skin biopsy may be necessary in the Shar Pei and in cases of pododermatitis if skin scrapings are negative.

Management The most effective therapies to date include topical amitraz weekly (0.125–0.250 ppm), oral milbemycin daily (2 mg/kg), and oral or parenteral ivermectin daily (300–600 µg/kg). Topical alternate day (500 µg/kg) and oral alternate day (450 µg/kg) ivermectin therapy has given 7% and <70% cure rates, respectively. Appropriate miticidal therapy should be continued until three consecutive negative deep skin scrapings are achieved two weeks apart, rather than until the dog is clinically normal.

Because *Staphylococcus* is considered immunosuppressive in dogs with demodicosis, identifying and treating secondary bacterial infections helps minimize pyoderma as a contributing factor of demodicosis. When deep pyoderma is present, intact bulla or tissue culture with antibiotic sensitivity is financially prudent due to the long duration of antibiotic therapy required.

Generalized juvenile-onset demodicosis is hereditary and can be a serious and expensive disease to treat. All dogs with juvenile-onset generalized demodicosis must be neutered as soon as practical. Mature dogs that develop generalized demodicosis may have an associated endocrine abnormality. Corticosteroids are absolutely contraindicated in dogs with any form of demodicosis.

Feline demodicosis

Definition/overview Feline demodicosis is an uncommon parasitic disease due to increased numbers of demodicid mites in the skin.

Etiology Feline demodicosis is caused by a follicular mite *Demodex cati* (which looks similar to *D. canis*) or a surface mite *D. gatoi* (shorter with a blunt, rounded abdomen) (41). An inapparent carrier state and contagiousness between cats has been reported with *D. gatoi*.

Pathophysiology Some cases of feline demodicosis have been associated with other diseases. These have included food allergy, feline acne, FIV, diabetes mellitus, and actinic dermatitis.

Clinical presentation Cats may present with pruritus and fur pulling, localized or symmetrical alopecia, erythema, and excoriations.

Differential diagnosis Bacterial folliculitis/furunculosis, psychogenic alopecia, dermatophytosis, atopic dermatitis, food hypersensitivity, flea bite hypersensitivity, infestation with *Cheyletiella* spp. or *Notoedres cati*, contact dermatitis.

Diagnosis If skin scrapings are negative, topical treatment with lime sulfur should be considered in any cat that presents with the above symptoms.

Management Most cases respond to weekly dips with 2% lime sulfur for 4–6 weeks. Failing this, 0.0125% amitraz may be used weekly as a dip and continued for three weeks after a skin scraping has given negative results. All cats in the household should be treated simultaneously when *D. gatoi* is found or suspected.



39 Patch of perifollicular hyperpigmentation and comedones in a five-year-old Afghan Hound with adult-onset generalized demodicosis.



40 Adult and larval stages of *Demodex canis*.

Table 3. Common external parasites affecting the skin of dogs and cats

Parasite	Depth of skin scraping	Location	Treatment
<i>Cheyletiella</i>	S	Trunk	I, P, L, A
<i>Notoedres</i>	S	Head	I, L, A
<i>Sarcoptes</i>	S	Ear margin, elbows, hocks	I, L, M, A, Se
<i>Otodectes</i>	S	Head, rump	I, P
<i>Demodex</i> *	D**	Face, feet, trunk	I, M, A
<i>Demodex</i> †	D, S	Head, trunk	A, L
I Ivermectin	M Milbemycin	S Superficial	* Dog
L Lime sulfur	A Amitraz	D Deep	† Cat
P Pyrethrin	Se Selamectin		** Hair plucks are also useful



41 Adult mite of *Demodex gatoi*. (Photo courtesy of SR Merchant, DVM.)

Canine scabies

Definition/overview Canine scabies (sarcoptic mange) is a contagious dermatosis of dogs, and rarely cats, caused by the mite *Sarcoptes scabiei* var. *canis*.

Etiology Caused by the highly contagious mite *Sarcoptes scabiei* var. *canis*, sarcoptic mange is one of the most pruritic skin diseases of dogs. The mite has also been reported to cause disease in cats, foxes, and humans.

Pathophysiology Most of the pruritus may be caused by a hypersensitivity reaction to the mite and its secretions.

Clinical presentation Canine scabies is a ventrally distributed disease with the ear margins and the elbows, hocks, and abdomen typically involved (42). The pinnal-pedal reflex may be positive in 25–90% of dogs with scabies (20).

Differential diagnosis Flea bite hypersensitivity, atopic dermatitis, food hypersensitivity, *Malassezia* dermatitis, *Pelodera strongyloides* dermatitis.

Diagnosis Apart from puppies, fewer than 25–50% of dogs with symptoms of sarcoptic mange are positive for mites on superficial skin scrapings (43). The diagnosis is often ultimately made by response to appropriate therapy.

Management The most effective therapies are topical lime sulfur rinse (weekly), ivermectin (oral, pour-on, or subcutaneous weekly), milbemycin (every other day for 14 days or weekly), selamectin (twice, two weeks apart), and topical amitraz rinses (weekly). Therapy should be continued for 4–6 weeks. All in-contact animals must be treated as well as the environment. Variable success has been noted with either topical organophosphate rinses or fipronil spray.



42 Pinnal alopecia due to canine scabies in a black Labrador Retriever.

Otodectic acariasis

Definition/overview Otodectic acariasis is a contagious, parasitic, otic or cutaneous disease caused by the psoroptid mite *Otodectes cynotis*.

Etiology *O. cynotis* is the most common cause of otitis externa in young cats. Its incidence in dogs is less common. The mites can also transiently affect humans.

Pathophysiology *Otodectes* mites feed on epidermal debris and tissue fluid, exposing the host to mite antigen. The ear canal epidermis becomes irritated, producing excessive cerumen and blood.

Clinical presentation Ear pruritus with black, granular debris in the external ear canal is a common symptom. *O. cynotis* may also exist outside of the ear canal and be a cause of head and tail pruritus, especially in cats (44). Papules, crusts, and excoriations may be evident.

Differential diagnosis Foreign bodies, bacterial infection, yeast infection, *Pseudomonas* spp. infection, defects in keratinization, autoimmune diseases, hypersensitivities.

Diagnosis Diagnosis may be made by visualization of the mites in the ear canal, mineral oil ear swabs, or on skin scrapings or acetate tape preparations (45). However, mites may be difficult to demonstrate in the ear canal as immunity to the salivary antigens of the mite may develop and the ensuing inflammation may destroy the mites or cause them to leave the ear canal.

Management Aural and topical parasiticides applied to the ears and entire body, respectively, at varying intervals, for a total of 30 days is required. Ivermectin (oral weekly, pour-on or subcutaneous biweekly) is also effective. All in-contact animals must be treated as well.



43 Adult *Sarcoptes scabiei* var. *canis* mite.



44 Alopecia and scale due to *Otodectes cynotis* dermatitis in a cat. (Photo courtesy of CS Foil, DVM.)



45 Adult *Otodectes cynotis* mite from the ear of a cat. (Photo courtesy of E Greiner, PhD.)

HYPERSENSITIVITIES

Canine atopic dermatitis

Definition/overview Atopic dermatitis is an inherited tendency to respond to environmental allergens by developing a type I hypersensitivity.

Etiology Canine atopic dermatitis results from a genetic predisposition to become sensitized to environmental allergens. Although the route of allergen access is still controversial, inhalation and percutaneous absorption of these allergens seem likely.

Pathophysiology The pathophysiology is still unclear in animals. In humans atopic dermatitis is associated with increased activation of T lymphocytes, defective cell-mediated immunity, hyperstimulatory Langerhans cells, and overproduction of B-cell IgE.

Clinical presentation Atopic dermatitis usually affects young adult dogs (onset at 1–3 years old) with a seasonal pruritus. It is important to discern the presence, intensity, and frequency of itch; the aspect, distribution, and progression of cutaneous lesions; flea eradication programs; and previous treatment and effect. Pruritus should be evident and affect one or more of the following areas: face, extensor and flexor skin surfaces, axilla, pinna, and groin (46).

Differential diagnosis Sarcoptic mange, cheyletiellosis, pediculosis, contact allergic dermatitis, food hypersensitivity, flea bite hypersensitivity, *Pelodera strongyloides* dermatitis, *Malassezia* dermatitis, superficial pyoderma, xerosis.

Diagnosis Diagnosis of atopic dermatitis is based on compatible historical and clinical information, as well as ruling out other causes of pruritus. Secondary diseases may also contribute to the pruritic threshold in the atopic patient and these can be recurrent problems which must be continually addressed; otitis externa/media, superficial pyoderma, acute moist dermatitis, keratinization disorder, *Malassezia* dermatitis, flea bite hypersensitivity (common in dogs with atopic dermatitis), acral lick dermatitis, and fibropruritic nodules (noted in some dogs with flea bite hypersensitivity).

Management When allergen avoidance is not possible, hyposensitization is the treatment of choice for animals with a prolonged allergy season. Hyposensitization based on *in vitro* or *in vivo* allergy testing is effective in decreasing pruritus or reducing the need for other medications in 60–80% of dogs. The success of hyposensitization may not be noted for 3–12 months.

Symptomatic therapy includes the use of antihistamines and essential fatty acids, topical antipruritic therapy, oral alternate day corticosteroids, and avoidance (if possible), as well as managing secondary problems.



46 Papules, erythema, alopecia, and excoriations on the palmar metacarpal area of a Weimaraner with atopic dermatitis and superficial pyoderma.