Parasitic Mites of Dogs and Cats

KEY FACTS

- In dogs and cats, parasitic mites can cause irritation and annoyance, may induce localized infection, and can be debilitating or fatal.
- Mites lack natural enemies and can be resistant to parasiticides; some species can lower host immunity.
- Some mite infestations are easily eliminated from the animal and environment. Others are difficult or impossible to eradicate.
- Prompt diagnosis of mite infestation and identification of the mite species as well as appropriate treatment are requisite to eliminate the parasite, to institute any ancillary treatment, and to advise the owners regarding contagion of the parasite or any possible predisposing genetic factors of the host.
- In some cases, extra label therapy with such agents as amitraz, roenell, or ivermectin is the most effective treatment. Owners must give informed consent before the extra label treatment is initiated.

Parasitic mites are members of the class Arachnida, order Arachnida (see the listing on Mite Taxonomy). Mites have a four-stage life cycle: egg, six-legged larva, eight-legged nymph, and eight-legged adult. Except for chiggers (Trombiculidae), of which only the larval stage is parasitic, mites of all four stages are parasitic.

The three suborders of mites in the order Arachnida consist of animals of diverse sizes, shapes, and distinguishing characteristics (Table I). The host anatomic areas preferred by various mite species differ (Table II) as do the geographic distribution (Table III) and potential for contagion to other animals and to humans (Table IV). Because the clinical significance and management of mite infestation depend on the mite species, this article discusses each of the commonly encountered mites separately.

DEMODEX SPECIES

Clinical Manifestations in Dogs

A small population of Demodex canis mites is a normal finding on or in the skin of healthy dogs. The elongated mite (Table I) lives its entire 20- to 35-day life cycle in the hair follicle, leaving only to cross to another hair follicle (Figure 1). The mites are transmitted to puppies by contact with the dam during the first two to three days of life. Mites can also be transmitted from one pup to another in the litter. Susceptible young dogs reportedly can contract Demodex canis mites by direct and close contact with a heavily parasitized carrier.

The presence of Demodex canis mites is considered to be a disease state when excessive numbers of mites of all four stages of the life cycle are present in a skin scraping from an animal with clinical signs. Demodicosis is also termed follicular, red, puppy, squamous, pilosebaceous, or Acarus mange. Demodex mites reproduce and lay eggs in the hair follicle and associated sebaceous and apocrine sweat glands. The mites puncture cells and ingest the cell contents; this feeding activity produces keratinization that plugs and distends the follicle. Proliferation of the mites, with subsequent rupture of the hair follicle, allows the parasites to enter the bloodstream, lymphatics, lymph nodes, and various body organs. Inflammation and foreign body response also occur.

Demodicosis occurs in three clinical forms: localized demodicosis, generalized (juvenile or adult) demodicosis, and chronic pododermatitis. The localized form is the most common and usually involves the face, skull, ear canal, forelegs, or trunk. Affected dogs may have alopecia, varying degrees of scaling, hyperpigmentation, minimal pruritus, erythema, and bacterial infection. The mites may elude discovery. Multiple scrapings from squeezed hair

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Mite Taxonomy (Order Acarina)1

Class Arachnida: Head, thorax, and abdomen are fused. Mouthparts in capitulum; antennae and mandibles missing. Six-legged larvae, eight-legged nymph and adult stages.

Order Arachnid

Suborder Prostigmata

Family Demodicidae

Demodex canis
Demodex cati
Unnamed Demodex species

Family Cheyletiellidae

Cheyletiella yasguri
Cheyletiella blakei

Family Trombiculidae

Walchia americana
Trombicula alfreduggesi
Trombicula autumnalis

Suborder Astigma: Respiratory pores absent; respiration occurs through integument

Family Sarcoptidae

Sarcoptes scabiei var. canis
Notoedres cati

Family Psoroptidae

Otodectes cynotis

Subfamily Listrophoroidea

Lynxacarus radovskyi

Suborder Mesostigmata: Respiratory pores on mid body

Family Halarachnidae

Pneumonyssoides caninum
Family Dermanyssidae

Dermanyssus gallinae

Suborder Metastigmata: Ticks

follicles on the margin of lesions may be needed.

Skin scraping suffices to diagnose localized or generalized demodicosis; whereas in cases of pododermatitis, skin biopsy sometimes is necessary to establish a diagnosis. Localized demodicosis is most common in intact purebred dogs (76%) younger than one year of age. Nineteen percent of cases of localized mange resolve spontaneously in four to eight weeks. Underlying transient stresses (e.g., adolescence, estrus, endoparasites, growth, nutrition, vaccination, surgery, and pregnancy) have been incriminated and may contribute to the disease. Sex, haircoat length, sebaceous gland size, and hypothyroidism do not appear to be factors in the development of localized demodicosis.2,18,19

Ten percent of dogs with the localized form deteriorate to generalized juvenile demodicosis. The generalized form involves large areas of the body and causes alopecia (in more than 10 areas), erythema, and varying degrees of crusts, follicular plugging, and hemorrhage. A rancid seborrheic odor, folliculitis, furunculosis, cellulitis, deep pyoderma, pruritus, and peripheral lymphadenopathy often accompany generalized juvenile demodicosis. The condition can also occur without major associated reactions2,11-13,22 (Figure 2).

In one study of 821 dogs with generalized juvenile mange, 87% of the cases occurred in animals older than seven months of age; most of the subjects were intact purebreds (80%). Age and familial predisposition were the most important factors. Two mechanisms have been proposed to explain the inherited immunologic predisposition to generalized juvenile demodicosis. The first explanation is that a genetic immune abnormality allows massive mite proliferation. The heavy mite population induces a humoral substance (B-cell stimulation) that in turn suppresses cell-mediated (T-cell) immunity. Treatment is directed at elimination of the mites to restore T-cell function.23-25

The second proposed mechanism is that genetic predisposition to pyoderma complicates the generalized juvenile demodicosis and suppresses lymphocyte blastogenesis (B and T cells). The pyoderma suppresses serum lymphocyte immunoregulatory factors in proportion to the degree of infection. Therapy would therefore be directed at the skin infection.22 Immunosuppressive agents, such as corticosteroids, should not be used in animals with generalized juvenile demodicosis. Because of the inherited predisposition to the condition, the American Academy of Veterinary Dermatology recommends that dogs with generalized demodicosis should be sterilized.26

Demodectic pododermatitis tends to be a chronic digital and interdigital disease often complicated by pyoderma. Certain breeds are at increased risk (see the listing on Canine Breeds Predisposed to Demodicosis). Biopsy may be necessary to reveal the mites. In many cases, the condition is never cured.2,27

Adult-onset demodicosis may not be hereditary. Most cases are secondary to iatrogenic Cushing's syndrome, atopy, immune deficiencies, diabetes mellitus, heartworms, malignancies, use of immunosuppressive agents, or deep pyoderma. Patients with the adult-onset disorder must be thoroughly examined and checked for other parasites and disease processes. A complete blood count, blood profile, and endocrine screening should be performed to identify any inciting factors needing treatment. Scottish terriers and West Highland white terriers seem particularly prone to adult-onset demodicosis complicated with atopy and deep pyoderma (Figure 3). Adult-onset generalized demodicosis can necessitate euthanasia.2,27-29

Treatment of Canine Demodicosis

Therapy for canine demodicosis depends on its form and complications. Localized demodicosis has a good prognosis; 90% of cases remit spontaneously within four to eight
weeks without treatment. If the patient’s owner insists on some form of intervention, topical 1% rotenone or follicular flushing products with benzoyl peroxide may be applied.18

For the 10% of localized cases that deteriorate to generalized demodicosis, the prognosis is less optimistic and individualized therapy is indicated. From 30% to 50% of generalized juvenile demodicosis cases resolve spontaneously. Acaricidal therapy may not be necessary immediately; the clinician should monitor the patient’s progress closely. Skin scrapings performed at two- to four-week intervals indicate when increasing mite population and predominance of immature forms dictate more-aggressive treatment. Review of the animal’s pedigree could reveal the likelihood of spontaneous remission.2,17-19

Treatment of generalized demodicosis (juvenile and adult-onset) should be directed at simultaneous elimination of mites and any underlying pyoderma. Currently, amitraz is the only agent approved for use as a Demodex miticide in the United States. The manufacturer advises that 10.6 ml be diluted in two gallons of water (250 ppm = 0.025%) and applied every two weeks to the dog’s dry haircoat. Gloves must be worn during application of amitraz.

Before amitraz application, the haircoat should be shaved and cleansed with a follicular flushing shampoo or a therapeutic shampoo (e.g., with povidone-iodine or chlorhexidine). Areas of deep pyoderma and furuncles should be avoided during dipping to minimize toxicity associated with amitraz absorbance.27

Extralabel use of amitraz dramatically improves cure rates; an animal is considered cured if skin scrapings are negative and the animal lacks clinical signs six months after the last treatment. One study24 found that when 0.03% amitraz (6 ml of 19.9% amitraz in two gallons of water) was applied weekly rather than every two weeks, the cure rate for generalized demodicosis can rise from 20% to 75%. Halving the diluent (6 ml amitraz in 1 gallon of water) to double the dose to 0.06% can raise the cure rate up to 80% with minimal toxicity. Amitraz toxicity is manifested as increased sleepiness, depression, and anorexia during the first 24 hours after treatment. More-extreme signs of toxicity, such as ataxia, polyuria, polydipsia, hypothermia, seizures, pruritus, erythema, hyperglycemia, and glucose intolerance, may occur.30-31 Death is highly unlikely.30

If a doubled dose of amitraz is administered weekly, hyperglycemic and insulin-suppressing effects require special caution for diabetic animals.31 Humans with diabetes must wear gloves when applying amitraz. Weekly administration of 0.05% amitraz seems most efficacious; but pet owners must be advised that this regimen is extralabel.30

In France, amitraz concentrations of 0.05% and 0.10% have reportedly been effective and produced no toxic side effects.32 Also in France, an amitraz-impregnated collar is marketed for Demodex and tick control.33 Treatment failures may be related to premature cessation of treatment or failure to correct underlying stress or infection. Months of weekly dipping may be needed to effect a cure; there is no prescribed number of dips.

Extralabel use of ronnel can be tried to treat generalized juvenile demodicosis if amitraz fails. Ronnel is mixed as a 4% solution with propylene glycol (Scott’s formula).24 The agent can be dispensed in a 4-ounce childproof amber medicine bottle containing 18 ml of 33.3% ronnel solution plus 100 ml of propylene glycol. The preparation is stable for about one month. One third of the dog’s body is painted daily so that each area is treated once every three days in sequence. Ronnel is effective in 90% of cases but is irritating to the skin and highly toxic to animals and humans.19 Dogs to be treated with ronnel (or any organophosphate) should be free of heartworm infection before treatment is initiated. Demodex canis is resistant to ectoparasiticides less potent than ronnel.

Demodectic pododermatitis can be treated with weekly immersion in double-strength amitraz (0.05%) or 4% topical ronnel. Fresh daily amitraz can be applied (1 ml of 19.9% amitraz per 30 ml of propylene glycol); however, oxidation makes this topical preparation unstable. Aqueous trichlorfon (3%) solutions may be used as a topical therapy but are less effective than ronnel19; this use of trichlorfon is also extralabel.

For all miticidal therapies, skin scrapings must be monitored at two- to four-week intervals. Counts or estimates of the numbers of live and dead mites should be made and therapy continued for three to four weeks after scrapings are negative. Recurrence is possible, and lifelong amitraz dips may be necessary.28,33

Concurrent pyoderma must be treated simultaneously. Skin culture and sensitivity studies are indicated in complicated cases of demodicosis. Coagulase-positive staphylococci are the most common isolate, but Proteus or Pseudomonas organisms may be present. Pseudomonas seriously changes the prognosis because it can cause fatal septicemia.18 An antibiotic, such as erythromycin, lincomycin, oxacillin sodium, cephalosporin, or chloramphenicol, may be used until the culture and sensitivity report returns. Antibiotics should be continued weeks beyond healing.29 Clients should be advised of the zoonotic potential of the pet’s infected skin.

Vitamin E (up to 400 U orally twice a day) and fatty acid supplements may quell skin inflammation in dogs with generalized demodicosis and are useful for treating any underlying atopy.33 Immune stimulants, such as levamisole or injectable bacterins, have not been successful against mites.
TABLE I

<table>
<thead>
<tr>
<th>Mite</th>
<th>Size of Adult Female (μm)</th>
<th>Characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Demodex canis</em></td>
<td>40 x 225</td>
<td>Spindle-shaped eggs; elongated body, striations</td>
</tr>
<tr>
<td><em>Demodex cati</em></td>
<td>30 x 200</td>
<td>Oval eggs; smaller and slimmer body than <em>Demodex canis</em></td>
</tr>
<tr>
<td>Unnamed <em>Demodex</em> species</td>
<td>30 x 110</td>
<td>Short, blunt abdomen; superficial skin parasite</td>
</tr>
<tr>
<td><em>Cheyletiella yasguri</em></td>
<td>265 x 500</td>
<td>Cone-shaped sensory organ</td>
</tr>
<tr>
<td><em>Cheyletiella blakei</em></td>
<td>265 x 500</td>
<td>Huge mouthhooks, heart-shaped sensory organ; surface mite</td>
</tr>
<tr>
<td>Trombiculidae (chiggers)</td>
<td>210 x 400</td>
<td>Orange-red, six-legged parasitic larva; hairy body; surface mite</td>
</tr>
<tr>
<td><em>Sarcoptes scabiei var. canis</em></td>
<td>250 x 400</td>
<td>Terminal anus; long, unsegmented pedicel</td>
</tr>
<tr>
<td><em>Notoedres cati</em></td>
<td>200 x 240</td>
<td>Dorsal anus, body striations</td>
</tr>
<tr>
<td><em>Otodectes cynotis</em></td>
<td>300 x 400</td>
<td>Long legs, short pedicel; males have pretarse on all four pairs of legs; surface mite</td>
</tr>
<tr>
<td><em>Lynxacarus radovskyi</em></td>
<td>250 x 515</td>
<td>Hair-clasping sternal plates, elongated body; surface mite</td>
</tr>
<tr>
<td><em>Pneumonyssoides caninum</em></td>
<td>150 x 400</td>
<td>Long legs, small palps; often confused with <em>Dermanyssus</em> mites</td>
</tr>
<tr>
<td><em>Dermanyssus gallinae</em></td>
<td>150 x 400</td>
<td>Long legs, stocky palps; deeply embedded, red poultry mites; surface mites</td>
</tr>
</tbody>
</table>

but may aid pyoderma therapy. 

The extralabel use of ivermectin has produced disappointing results against *Demodex* mites, although it is highly effective against other mites. Ivermectin paralyzes susceptible mites by potentiating the neuroinhibitor \( \gamma \)-aminobutyric acid (GABA), thus blocking postsynaptic neuromuscular potentials, and is lethal to the parasite. Ivermectin does not cross mammalian blood-brain barriers to reach the GABA receptors. Ivermectin can cause potentially fatal idiosyncratic reactions in collies, sheepdogs, and related crossbreeds and should not be used in these animals. Dogs should be screened for heartworm infection before receiving ivermectin therapy; the precise weight of all animals, dogs or cats, should be measured to determine the correct dose.

A 1% preparation of ivermectin provides 10 mg/ml (10,000 μg/ml). In one study, two 400-μg/kg injections of ivermectin given two weeks apart were effective against *Demodex canis*. Another trial of the same dose at weekly intervals did not resolve the infestation but seemed to provide some reduction of clinical signs. The efficacy of milbemycin oxime against *Demodex canis* is under study.

**Clinical Manifestations in Cats**

Feline demodicosis is caused by the follicular mite *Demodex cati* (Figure 4) and a much less common unnamed *Demodex* species, which is found only in the stratum corneum of the cat's skin. The size and shapes of these mites differ from those of *Demodex canis* (Table I). Both species are rare. Feline demodicosis manifests itself in two forms. The localized form exhibits erythematous patches of alopecia along with scaling and crusting of the eyelids, face, chin, or neck (Figure 4). Ceruminous otitis may be seen. Multiple skin scrapings disclose the parasite. Spontaneous remission can occur. Generalized feline demodicosis is usually associated with an underlying disease (e.g., diabetes mellitus, hyperadrenocorticism, endocrine alopecia, feline leukemia virus infection, feline immunodeficiency virus, or an autoimmune disease).

Generalized feline demodicosis may be more common in
Figure 1A
Figure 1-(A) Characteristic *Demodex canis* mite measuring 40 μm × 225 μm. (x60)

Figure 1B
Demodex canis egg. (x60)

Figure 2-A
10-month-old female akita with generalized juvenile demodicosis. Pyoderma was minimal relative to the extent of *Demodex canis* lesions.

Burmese and Siamese cats than in other feline breeds. Extensive lesions of erythema, alopecia, crusting, and hyperpigmentation can involve the head, neck, legs, and trunk. Eosinophilia and pyoderma may occur. Intense pruritus can occur with infestations of the unnamed *Demodex* species.

**Treatment of Feline Demodicosis**

The prognosis for treatment of feline demodicosis is good. Treatments include 2.5% lime-sulfur dips, carbaryl shampoos, and malathion dips. Carbamate products, however, are not approved for use on kittens or nursing puppies. Organophosphates should only be used cautiously in cats. An extralabel regimen of weekly half-strength amitraz dips (0.0125%) is effective against feline demodicosis. Sedation and salivation are transient side effects, especially when the amitraz preparation is used full-strength. Diabetic cats with demodicosis should be treated with miticides other than amitraz.

**CHEYLETIELLA SPECIES**

**Clinical Manifestations**

*Cheyletiella yasguri* of dogs and *Cheyletiella blakei* of cats are large, surface-feeding mites that live in keratin (Table I). Their 35-day life cycle is spent entirely on the dog or cat. The mite has enormous hooked mouthparts and combed legs; it attaches its eggs to hair. The mites are very mobile and contagious by direct contact; the so-called walking dandruff can spread rapidly through a kennel or cattery. Puppies and kittens are most susceptible.

*Cheyletiella* mites are not host specific, and a reference text is advisable for correct identification of the species. The hallmark of *Cheyletiella* is the moving white flakes along the dorsal midline and head of the dog and cat. Scruping, combing, or applying transparent adhesive tape to the suspect areas aids in disclosing the large, visible mites. Magnifying loupes and selective removal of questionable flakes or hairs represent the quickest method. Some fastidious cats may require skin scrapings or fecal flotations to reveal mites.

Dogs experience more pruritus associated with cheyletiellosis than do cats. The canine disease may resemble seborrhea oleosa or flea bite dermatitis. Affected dogs have scaly, oily skin. Cats may appear to have seborrhea sicca with scales and red scabs, or they may show no signs at all. Humans, especially cattery personnel, are highly susceptible to *Cheyletiella* infestation. Pruritus, erythematous papules, and pustules appear on the chest, arms, thighs, and abdomen. The human disease is transient (three weeks) without reexposure.

**Treatment**

Treatment of *Cheyletiella* mite infestation can be accomplished by using lime-sulfur dips, carbaryl shampoo or 5% dust, 0.5% malathion dips, or pyrethrins weekly for four or five treatments. Although amitraz has not been approved for use against *Cheyletiella*, this agent is effective.
### TABLE II
Host Anatomic Areas Preferred by Canine and Feline Mites

<table>
<thead>
<tr>
<th>Mite</th>
<th>Preferred Location on Host</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Demodex</em> species</td>
<td>Localized: follicles of face, skull, ear canal, forelegs; Generalized: anywhere and everywhere</td>
</tr>
<tr>
<td><em>Cheyletiella</em> species</td>
<td>Surface of dorsal midline</td>
</tr>
<tr>
<td>Trombiculidae</td>
<td>Surface of head, legs, and ventral areas</td>
</tr>
<tr>
<td><em>Sarcoptes scabiei</em> var. canis</td>
<td>Epidermis of ear tips, elbows, and glabrous areas</td>
</tr>
<tr>
<td><em>Notoedres cati</em></td>
<td>Epidermis of ears, face, and feet</td>
</tr>
<tr>
<td><em>Otodectes cynotis</em></td>
<td>Surface of ear canals and perilauricular areas</td>
</tr>
<tr>
<td><em>Lynxacarus radovskyi</em></td>
<td>Surface of perineum, tailhead, and tail tip</td>
</tr>
<tr>
<td><em>Pneumonyssoides caninum</em></td>
<td>Nasal sinus, nasal passages</td>
</tr>
<tr>
<td><em>Dermanyssus gallinae</em></td>
<td>Surface of legs and back</td>
</tr>
</tbody>
</table>

TROMBICULIDAE
Clinical Manifestations

Trombiculiasis is infestation by parasitic, six-legged, larval chiggers. Dogs, cats, and humans can be parasitized by 20 of the more than 700 species of chiggers. Infestation is most prevalent during the late summer and fall and in animals frequencing fields or woodlands where the larval mites are contracted directly (Table III). The nymphs and adult chiggers are free-living and not parasitic. The bright orange-red mites are surface feeders; their salivary secretions induce intense pruritus that lasts longer than the mites are present on the body. Alopecia, pustules, and scaling of the head, ears, ventral trunk, legs, and paws can occur. The entire body can be afflicted; self-inflicted injury is often a problem.

Diagnosis and Treatment

Diagnosis can be made by scraping the mite from the lesion, but the mite may already have detached from the animal. Subcutaneous ivermectin (300 μg/kg) repeated after a five-week interval is effective and useful for miticide-resistant cases and may be ideal for cattery and kennel infestations. Regardless of the treatment used against *Cheyletiella*, all of the animals in the household or facility must be treated.

### Generalized Juvenile Demodicosis

Afghan hound, beagle, Boston terrier, boxer, Chihuahua, Chinese shar pei, chow chow, collie, dalmatian, dachshund, Doberman pinscher, English bulldog, German shepherd, Great Dane, Old English sheepdog, pointer, pit bull terrier, pug, Staffordshire bull terrier

### Chronic Demodicetic Pododermatitis

Great Dane, Newfoundland, Old English sheepdog, Saint Bernard

### Generalized Adult-Onset Demodicosis

Cocker spaniel, Old English sheepdog, Scottish terrier, West Highland white terrier

Canine Breed Predisposed to Demodicosis

Scabies caused by the mite *Sarcoptes scabiei* var. *canis* is often misdiagnosed as contact allergy, food allergy, atopy, or flea bite dermatitis. Many pruritus-producing skin diseases can resemble scabies. This mite usually appears on dogs, occurs only rarely in cats, and spends its 17- to 21-day life cycle in epidermal pockets. The large female mite lays three to five eggs per day in freshly burrowed tunnels (Table I). The male mite, along with mites in the larval and nymph stages, lives in vacated burrows. The male dies after copulation. *Sarcoptes* mites have a long, unsegmented pedicel and a terminal anus.

INTENSE pruritus induced by hypersensitivity to burrowing female mites is a consistent clinical sign. The mite targets the pinnae, elbows, back, chest, abdomen, or hairless areas. The entire body can be affected. Alopecia, hemorrhagic crusts, and papules are worsened by self-inflicted injury. The skin becomes lichenified, and secondary infection and lymphadenopathy occur. The animal can become debilitated; but well-nourished, well-groomed dogs can have scabies. Frequent grooming of dogs and frequent insecticidal shampooing can cause and mask scabies in some pampered pets; the resultant condition has been called scabies incognito. Rubbing the infested animal's ear tip may elicit scratching movements of the hindlimb; this pinna-pedal reflex suggests scabies.
Multiple deep skin scrapings from scattered reddened crusty areas may reveal mites or eggs. To obtain an adequate skin scraping, the clinician must scrape hairless areas deeply enough for the area to ooze blood. In 35% to 50% of cases, no mites are found and diagnosis is based on history, clinical examination, and response to treatment. A history of sudden-onset pruritus, exposure to a kennel or pet shop, or pruritus among humans in the household suggests scabies. The mite is highly contagious by direct contact to other dogs; up to 50% of canine cases result in human disease. Humans evidence pruritic lesions on the arms, legs, and trunk. The canine parasite in humans is self-limiting and transient without reexposure.

**Treatment**

Treatment of infected dogs includes cleansing shampoos to remove debris as well as systemic antibiotics and anti-inflammatory agents to treat infection and self-inflicted injury. Laundering the animal’s bedding eliminates a source of reinfestation. Weekly malathion or phosmet dips have been suggested but may be ineffective because of resistant mites. Lime-sulfur dip (2.5%) is safe and effective; five dips are given at five-day intervals. Lime sulfur is also antifungal, antibacterial, and antipruritic. Amitraz is effective against *Sarcoptes* mites but has not been approved for that use. A single application of amitraz (0.025%) is effective in 98% of cases. Some dogs require a second or third application. Extralabel use of oral or injected ivermectin (200 μg/kg) is effective; a single treatment kills the mites. Regardless of the method of treatment, all dogs in a household must be treated.
TABLE III
Geographic Distribution of Canine and Feline Mites in the United States\textsuperscript{2,5,7-10}

<table>
<thead>
<tr>
<th>Mite</th>
<th>Distribution</th>
</tr>
</thead>
<tbody>
<tr>
<td>Demodex species</td>
<td>Throughout the United States</td>
</tr>
<tr>
<td>Cheyletiella species</td>
<td>Throughout the United States</td>
</tr>
<tr>
<td>Trombiculidæ</td>
<td>Uncommon; fields and woodlands, especially in the central United States</td>
</tr>
<tr>
<td>Sarcoptes scabiei var. canis</td>
<td>Throughout the United States</td>
</tr>
<tr>
<td>Notoedres cati</td>
<td>Uncommon; scattered enzootic areas</td>
</tr>
<tr>
<td>Otodectes cynotis</td>
<td>Throughout the United States</td>
</tr>
<tr>
<td>Lynxacarus radovskyi</td>
<td>Rare; Hawaii, Puerto Rico, southern Florida</td>
</tr>
<tr>
<td>Pneumonyssoides caninum</td>
<td>Rare; throughout the United States</td>
</tr>
<tr>
<td>Dermanyssus gallinae</td>
<td>Rare; bird nests, henhouses, pigeon coops</td>
</tr>
</tbody>
</table>

\textbf{NOTOEDRES CATI Clinical Manifestations}

\textit{Notoedres cati} is the mite of feline scabies. The mite is smaller than \textit{Sarcoptes} mites and has a dorsal anus and evident body striations (Figure 5 and Table I). Its life cycle is similar to that of \textit{Sarcoptes} mites. \textit{Notoedres cati} is highly contagious by direct contact and is present in large numbers on the cat. All of the cats in a household or neighborhood may become infected. Although \textit{Notoedres} mites are not a common parasite of cats, enzootic feline populations create cluster outbreaks with high morbidity\textsuperscript{2,9,10,15}

\textbf{CLINICAL signs include intense pruritus attributable to a hypersensitivity response to the burrowing female mites as well as alopecia, crustings, and scaling of the face, ear tips, and distal extremities. Hyperpigmentation, lichenification, self-inflicted injury, and secondary skin infection can be severe (Figure 5). Young and chronically infected cats become debilitated\textsuperscript{2,9,10,47} (Figure 6). Humans contacting infected cats can develop pruritic skin disease. Papules, crusts, and excoriations of the arms, legs, and trunk can occur. The human disease is self-limiting and transient without reexposure\textsuperscript{4,10}. Dogs rarely become parasitized by \textit{Notoedres} mites\textsuperscript{2,47}

Skin scrapings of lesions reveal numerous mites and eggs (Figure 7). The mites are active and rapidly spread over the cat’s body. Diagnosis is usually easy.

TABLE IV
Zoonotic Mites of Dogs and Cats\textsuperscript{2,4,7,11-16}

<table>
<thead>
<tr>
<th>Mite</th>
<th>Human Anatomy Affected</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cheyletiella species</td>
<td>Arms, thighs, trunk</td>
</tr>
<tr>
<td>Trombiculidæ</td>
<td>Legs, arms</td>
</tr>
<tr>
<td>Sarcoptes scabiei var. canis</td>
<td>Arms, legs, trunk, neck</td>
</tr>
<tr>
<td>Notoedres cati</td>
<td>Arms, trunk, neck</td>
</tr>
<tr>
<td>Otodectes cynotis</td>
<td>Arms, trunk</td>
</tr>
<tr>
<td>Lynxacarus radovskyi</td>
<td>Arms, forearms</td>
</tr>
<tr>
<td>Dermanyssus gallinae</td>
<td>Arms, chest</td>
</tr>
</tbody>
</table>

\textsuperscript{2}Extralabel use of ivermectin in dogs and cats kills these mites; ivermectin is contraindicated for collies and sheepdogs.

\textbf{Treatment}

Treatment includes cleansing shampoos to remove scales and crusts. Antibiotics for secondary skin infection and corticosteroids to treat pruritus and self-inflicted injury may be needed. Four to six weekly dips with 2.5 % lime sulfur are safe and effective and alleviate pruritus\textsuperscript{2,9,11}. Amitraz dips (0.0125%) repeated at two-week intervals are reportedly effective, but this regimen is extralabel\textsuperscript{11}. Stronger amitraz dips (0.025%) have reportedly been effective\textsuperscript{32,58}. Malathion dip also is effective\textsuperscript{2}. All cats in the household must be treated.

Extralabel use of ivermectin is highly effective against \textit{Notoedres} mites\textsuperscript{11,33,59-61}. I have treated an epizootic of \textit{Notoedres} mange using subcutaneous ivermectin (300 \mu g/kg) on cats as young as four weeks. Over 200 cats have been treated; most need only one ivermectin injection. No adverse effects have been apparent\textsuperscript{10}.

\textbf{OTODECTES CYNOTIS Clinical Manifestations}

Dogs and cats share the same ear mite species, \textit{Otodectes cynotis}. The mite is a large and mobile surface feeder (Table I). Transmission is by direct contact between pets; the mite is not host specific. \textit{Otodectes cynotis} mites have a 21-day life cycle on the host; the life cycle features a distinct stage involving deutonymphs. Deutonymphs are a sexually undifferentiated stage and will be joined end to end by an adult male mite (Figure 8). If the deutonymph molts to become a female, it will be fertilized. If a male develops or if a deutonymph fails to be joined by a male, no reproduction occurs. Ear mites have characteristic long legs that extend beyond the body as well as short pedicels on these legs\textsuperscript{7,2,47} (Figure 8 and Table I).

Ear mites irritate the ear canal and hypersensitize the pet. The mite antigen induces antibody production soon after the pet is parasitized\textsuperscript{2}. The result is pruritus; head shaking; and a heavy, dark, discolored wax discharge. Ear
mites do not thrive in infected ears; patients with purulent otitis are usually devoid of ear mites. The mites can leave the ear canal and irritate the neck, head, rump, and tail.2,9 Head shaking can induce aural hematomas, and ear scratching can cause localized moist dermatitis and traumatic skin excoriations. Humans can develop papules on the arms and trunk; this condition is self-limiting without reexposure.6,62 Diagnosis of Otodectes cynotis is by direct visualization or microscopic examination of a swab smear.

Treatment
Miticidal therapy is preceded by gentle cleansing of the ear canals with a cerumenolytic agent, such as mineral oil or squalene. Such miticides as topical thiabendazole, rotenone in oil, or methylcarbaryl are all effective. Treatment intervals are staggered to increase kill of wandering mites; periauricular areas can also be treated to kill any mobile mites. Because the mites are so contagious, all animals in the household, kennel, or cattery should be treated.2,47

EXTRALABEL use of ivermectin (injection of 200 to 400 μg/kg) can successfully eliminate Otodectes cynotis mites in dogs and cats. Ivermectin may be the ideal remedy for chronic infestations in a kennel or cattery.4,5,63 Extralabel use of amitraz (0.05% on the body and 0.5% diluted with 50% propylene glycol in the ear canals) is reportedly effective against Otodectes cynotis.32 Amitraz at a concentration of 0.025% is also effective.48

LYNXACARUS RADOVSKYI
Clinical Manifestations
Lynxacarus radoskyi is the cat fur mite. It is a large hair-clasping surface feeder that can be easily seen through a head loupe (Table I). The mite has modified sternal plates that allow it to clasp a hair and travel along hair shafts over the cat’s body1 (Figure 9).
Although *Lynxacarus radovskyi* is not common throughout the United States, it is enzootic in a few areas (Table III). The mite has an affinity for the tail tip, tailhead, and perineum. When this parasite is present in large numbers, the haircoat can feel granular and appear peppered. Small populations of fur mites cause no clinical signs; but heavily parasitized cats have pruritus, regional alopecia, and areas of shortened fur and develop a dry, rust-colored coat with such secondary signs as gingivitis or hair balls. The mite is contagious to cats and humans by direct contact. One of my clients had a papular forearm rash that resolved when her heavily parasitized cat was cleansed. Diagnosis is swiftly made by plucking suspect hairs to reveal the mite or attached eggs (Figure 9).

**Treatment**

Treatment is easily accomplished using 2.5% lime-sulfur dips or pyrethrin shampoos, powders, or sprays.25
Figure 10A

Figure 10B

Figure 10C

Figure 10—(A) This two-year-old male English bulldog has nasal discharge and pruritic facial irritation resulting from Pneumonyssoides caninum infestation. (B) A typical Pneumonyssoides caninum mite embedded in dried nasal exudate from the dog. (C) A long-legged nasal mite found wandering on the dog's muzzle. (×60)

collars do not work. House cats should be separated from feral cats to prevent reinfestation. Subcutaneous ivermectin (300 μg/kg) has been effective in killing Lynxacarus radovskyi in an ongoing epizootic I am treating.

PNEUMONYSSOIDES CANINUM
Clinical Manifestations

Pneumonyssoides caninum is the nasal or sinus mite of dogs. As with the previously mentioned cat fur mite, little is known about the complete life cycle of Pneumonyssoides caninum. Like fur mite infestation, Pneumonyssoides caninum infestation responds favorably to treatment. The sheltered habitat of the nasal sinus mite, however, protects it from injury and investigation. Larval and adult stages are usually seen, and the female may be ovoviviparous. The mode of contagion is unknown but is probably direct contact. The mite may cause no signs; however, rhinitis, serous nasal discharge, facial pruritus, sneezing, nocturnal restlessness, stertorous breathing sounds, and orbital infection have been reported. The mite has reportedly been found in lung, liver, and perirenal tissue (Figure 10).

Diagnosis is made by finding the long-legged mite crawling on the dog's muzzle, in nasal discharge, or in the nasal passages (observed by rhinoscopy). The mite could be confused with Dermanyssus mites or larval ticks.

Treatment

The mite can be killed by ivermectin injections (200 μg/kg); two doses three weeks apart is an effective regimen. Before this extralabel use of ivermectin was made, therapies for nasal sinus mite infestations included ether, rotenone, cresol, and organophosphates administered by nasal drops, inhaler bags, or insecticide strips.

DERMANYSSUS GALLINAE
Clinical Manifestations

The red poultry mite, Dermanyssus galliniae, infests dogs, cats, and humans contacting infested bird nests,
henhouses, or pigeon coops. The eight-legged nymph and adult stages are parasitic. The mite has long anterior legs. It tends to infest the back and legs of pets. Pruritus, papules, crusts, and erythema can be present. The mite is diagnosed by skin scrapings.

**Treatment**

Treatment is accomplished with lime sulfur, pyrethrins, or any safe acaricide. Ivermectin injections are effective in killing Dermanyssus gallinae on poultry; ivermectin may be as effective when used in the rare canine and feline cases of infestation. Avoidance of the source of the parasites or treatment of the source prevents reinfection.

**CONCLUSION**

Parasitic mite infestations of dogs and cats can be difficult or impossible to cure. Inherited factors, the contagious nature of the parasites, and their zoonotic potential complicate the management of these parasites. Because extralabel or nonapproved use of therapeutics may be necessary, owner awareness and cooperation are critical. Milbemycin, which has recently been approved for use in the United States, might someday be used to treat parasitism that is resistant to ivermectin or that has occurred in animals unable to tolerate ivermectin.

**About the Author**

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