

Mite infestations

DAMON McCLAIN*, ALI N. DANA† & GARY GOLDENBERG‡

*Bureau of Medicine and Surgery, Department of the Navy, Washington, District of Columbia, †Johns Hopkins University School of Medicine, Department of Dermatology, Baltimore, Maryland, and ‡Mount Sinai School of Medicine, Department of Dermatology, New York, New York

ABSTRACT: Mite infestations are important in dermatology because these may cause dermatologic diseases that range from papulosquamous eruptions to urticarial lesions to bullous eruptions and may spread infectious diseases. These clinical manifestations are important to recognize because mite-associated diseases may have systemic complications and may be confused with other dermatologic conditions. In treating mite infestations, oral antibiotics may be necessary. Prevention of infestation may be accomplished by pre-treating clothing with permethrin, using insect repellent N,N-Diethyl-meta-toluamide on clothing and skin, and treating animals infected with mites. This article will review etiology, clinical manifestation, and treatment of mite infestations.

KEYWORDS: DEET, infestation, mite, permethrin, treatment, typhus

Introduction

Mites are very small arthropods that may parasitize animals, humans included, or feed on organic matter. Mite infestations may cause dermatologic diseases that range from papulosquamous eruptions to urticarial lesions to bullous eruptions and may spread infectious diseases. These clinical manifestations are important to recognize because mite-associated diseases may have systemic complications and may be confused with other dermatologic conditions. In treating mite infestations, oral antibiotics may be necessary. Prevention of infestation may be accomplished by pre-treating clothing with permethrin, using insect repellent N,N-Diethyl-meta-toluamide (DEET) on clothing and skin, and treating animals infected with mites. This article will review etiology, clinical manifestation, and treatment of mite infestations.

Chiggers

Chiggers, also known as harvest bugs, harvest lice, harvest mites, jiggers, Mower's mites, and red bugs to name a few, are worldwide inhabitants (FIG. 1). The general term "chigger" refers to all larval forms of the trombiculid mite. In the United States, *Eutrombicula alfreddugesi* is abundant in the southeastern and south central regions, whereas, in Europe, the mite *Neotrombicula autumnalis* is more prevalent. The mites lay 40 eggs each month on leaves, blades of grass, or exposed ground (1). The eggs hatch in 1–2 weeks (1), and, after a dormant prelarval phase, the six-legged, red-colored larvae will cling to plants and wait for a host to brush up against them. Many mites require a relative air humidity of 80%, which is why they are typically not found on vegetation greater than 30 cm off the ground (2).

These larvae often feed on birds, reptiles, and small mammals, and, once they have fed for 3 or 4 days (1), they will drop off of their host. They feed by penetrating the stratum corneum with a feeding tube and injecting digestive enzymes that allow them to consume broken-down tissue (3). The chiggers are barely visible to the unaided eye at less than 1 mm in length and will often go

Address correspondence and reprint requests to: Gary Goldenberg, MD, Assistant Professor, Department of Dermatology and Pathology, Mount Sinai School of Medicine, 5 East 98th St 5th Floor, New York, NY 10029, or email: garygoldenbergm@gmail.com.
Conflicts of interest: none



FIG. 1. Chigger.

unnoticed even after the host has been bitten. After they finish their meal, they continue their maturation into their eight-legged nymph and adult stages (4).

The trombiculid mites prefer warm-blooded hosts, so the human can indiscriminately be a source of nourishment (5). Bites are more prevalent in the late summer and early autumn (6) and appear as grouped papules, papulovesicles, or urticarial wheals, which may progress to pustules. Although the first few exposures may be asymptomatic (5), over time, the lesions become extremely pruritic and can typically be found behind the knees, on the ankles, between the toes, and along the borders of undergarments. The mites can reside at sites where the clothing constricts and impedes their forward movement, and bites in those areas will have a characteristic circumferential distribution.

If the patient has a sufficient allergy to mites, vesicles and bullae may appear at the various bite sites. Patients may be misdiagnosed with scabies from penile and scrotal lesions. Children can present with “summer penile syndrome,” where a hypersensitivity response to chigger bites that occur during the months when they are most active and manifests as swelling, pruritus, and dysuria (6). In Asia, chigger mites are disease vectors for scrub typhus. They are also suspected to be the vectors of diseases such as hemorrhagic fever with renal syndrome, hantavirus pulmonary syndrome, ehrlichiosis, and other borrelial diseases.

Treatment of the chigger bites is aimed at relief of the symptoms. Although there is no widely accepted treatment regimen, options may include topical anesthetics, topical antipruritics, potent topical corticosteroid preparations, intralesional corticosteroid injections, oral antihistamines, or counterirritants. There have been reports that

topical anesthetics with pramoxine can be useful and that topical antipruritics with camphor and menthol can also help alleviate the accompanying intense pruritus (4). Additionally, potent topical corticosteroid treatments can also effectively treat the pruritus, but, unfortunately, the effect is not immediate, and the area typically needs to be occluded (4). Occluding the bites may actually increase the sense of itching, which may decrease patient compliance, but has had success with a technique of adding ice over the affected area to cool and soothe the pruritus (4). The topical steroid is placed first, then covered with a plastic wrap. Lastly, the ice is placed on top of the plastic (4). Many soldiers had reported symptomatic relief from the off-label use of propylene glycol as a counterirritant to the chigger bites acquired during field exercises (4).

Intralesional corticosteroids have also been used when patients did not respond to topicals (4). For atrophy at higher concentrations to be avoided, concentrations between 2.5 and 5 mg/mL can be used (4). For patients with unrelenting pruritus despite the above treatments, excision of the bite lesion may be the only option for relief (4).

Preventive methods that include avoidance of high-risk areas, as well as the use of the insect repellent DEET on both clothing and skin, and wearing clothing treated with permethrin, a synthetic pyrethroid, are very effective. Higher concentrations of DEET correspond to longer efficacy, not increased efficacy. According to the American Academy of Pediatrics, repellants containing DEET with a concentration of 10% appear to be as safe as products with a concentration of 30% (the plateau point and is the maximum concentration for infants and children) when used according to the directions on the product labels for children over 2 months of age. It is not recommended for use in children under 2 months of age. Formulations that offer sunscreen and DEET in one product should be avoided because sunscreens need more frequent application than DEET, and this may increase the chance of any potential toxic effects from the DEET.

Walking dandruff

Cheyletiella dermatitis in humans is typically the result of contact with an infected pet, with most cases resulting from an infested dog or cat. The causative mite in dogs is *Cheyletiella yasguri*; in cats, it is *Cheyletiella blakei*; and, in rabbits, it is *Cheyletiella parasitivorax*. *Cheyletiella* dermatitis is also known as “walking dandruff” (7) because



FIG. 2. *Cheyletiella blakei*.

the only symptom in these animals may be a mild dorsal dermatitis, although it may range from having heavy dandruff with mild alopecia and pruritus to completely asymptomatic. Up to 50% of rabbits on commercial colonies may be carrying *Cheyletiella* (8). The *Cheyletiella* mites do not burrow deep into the skin, have claw like anterior palps, and are six legged in the larval form and eight legged in the adult form. They are obligate parasites that live in the keratin layer of the epidermis where they feed on surface debris and tissue fluids (9) and are very difficult to see with the naked eye. They typically live out their life cycle on one animal host (10,11), where they mature from egg to adult in only 3 weeks, and the males will die within 2 days if separated from their host organism (9). Although females (and possibly the eggs) can live up to 10 days without a host (9), it is rare to find a *Cheyletiella* mite without the host animal nearby (FIG. 2). Some exceptions include one report of a boy who napped in the sleeping area of an infected dog and developed hundreds of pruritic lesions (12). Another case of *Cheyletiella* dermatitis was described where mites were discovered on the furniture, floor, and mattress (13).

These mites are very contagious, and 20% of pet owners with infested animals will become infected as well (14). The reports of *Cheyletiella* infection in humans typically have been on females, 40 years old or younger who present with grouped pruritic papules in areas that would have been in contact with the infested pet (15). Less common are widespread papules, urticarial wheals, vesiculobullous lesions, and one report of systemic manifestations of joint pain and numb fingertips with decreased motion of amplitude. The *Cheyletiella* mites do not take the time to burrow into or reproduce on humans, and this presents a diagnostic challenge. The mites typically bite the human and quickly

return to the animal host, which has been called "bite and run" behavior (12).

A veterinarian should perform the search for mites on the suspect animal. Simple diagnostic tools such as adhesive tape can be attempted to see if mites or eggs can be visualized (16). Rapid-acting knockdown insecticidal sprays can be used on the areas of the pet's dermatitis to help with the collection of the mites. Caution should be used when choosing a spray, as certain formulations may be toxic in some animals. The sprayed areas of dandruff can then be carefully brushed with a fine-toothed comb or a toothbrush, and all crust, scale, and hair should be collected and placed in a sealable plastic bag. With the addition of alcohol to the contents of the bag, the mites tend to float although most of the debris should sink. Vacuum cleaners with in-line filters can also be used to gather the mites. The filter samples are then treated with hot potassium hydroxide and then suspended in a concentrated solution to retrieve the mites (17).

Treatment for *Cheyletiella*-infested animals has included the use of ectoparasitic shampoos and dips. Canine cheyletiellosis has also been treated with a combination of permethrin and fipronil sprays (18) as well as topical amitraz (19). With reports of mites occasionally being found off the host animal, a thorough cleaning of the pets' sleeping area would be wise to prevent possible reinfection. The lesions on the owners should disappear within 3 weeks of their pet being appropriately treated, and no additional treatment for the humans should be necessary (20).

House mouse mite

The organism that causes rickettsialpox, *Rickettsia akari*, is transmitted via the house mouse mite *Liponyssoides sanguineus* (formerly known as *Allodermanyssus sanguineus*), to the common house mouse (*Mus musculus*) (21). The mouse then serves as a reservoir for the disease. The mite's life cycle consists of a larval form, which passes through two nymph stages before reaching adulthood (22). The nymphs and adults feed on blood every 3 or 4 days, although they can do so much less frequently and still survive (22). Each feeding typically only takes 1–2 hours, and the mite swells to many times its previous size and changes from colorless to bright red (22). The *R. akari* are passed transovarially in the mite, and *L. sanguineus* tend to lay eggs after feedings. The common house mouse may not be the only host for *L. sanguineus*;

the Korean vole, Mongolian gerbils, and Egyptian gerbils may also be hosts for this parasite (23).

With a healthy population of mice to parasitize, *L. sanguineus* is unlikely to seek out a human host. Cases of humans infected with rickettsialpox have been noted more commonly in urban areas where the mice population has been eradicated. The human is usually unaware that they have been bitten, as the bite is typically painless, and a small eschar or dark crust will develop at the site of the bite within 1–2 days (24). This initial lesion may have some mild erythema, but it is rarely painful and will typically heal in 2–3 weeks on its own and leave a small scar at the site of the bite. In a study of 13 patients with rickettsialpox, eschars were biopsied in five patients, and direct fluorescent-antibody testing revealed organisms consistent with *R. akari* (25).

A few days after the initial lesion, almost all patients develop fever and malaise. Most fevers run between 101 and 104°C (26) and are often accompanied by headache, stiff neck, myalgias, and photophobia. Cerebrospinal fluid studies are typically normal (27). Conjunctival injection, pharyngitis, cough, nausea, vomiting, and lymphadenopathy may also be seen. The stereotypical generalized rash of rickettsialpox (named so because of the relationship to chickenpox) may begin at the same time as the constitutional symptoms or up to 9 days later. The rash may start as maculopapular and usually (but not always) progresses to papulovesicular lesions (28). These lesions may be numerous and vary in location but will scab over and fall off without scarring.

Though similar to varicella, they can be differentiated by the presence of eschars and papules, versus the purely vesicular appearance of chickenpox. During the febrile phase of the disease, patients may also demonstrate leukopenia (27,29). Diagnosis can be made with biopsy of a lesion, isolation of the organism in the blood, or serologic testing. There is also an antibody available that detects an antigen that is common to the group of spotted fevers.

Treatment is not typically needed for rickettsialpox, as it will resolve on its own in most situations, but, in some cases, antibiotics have been used and shortened the duration of symptoms. If antibiotics are used, doxycycline is preferred at the dosage of an initial 200 mg orally and then 100 mg every 12 hours until the patient is afebrile and feeling well for 2–3 days. Erythromycin, tetracycline, and chloramphenicol (29) have also been shown to be effective, although some feel that, because the illness is self-limited, it is unreason-

able to use chloramphenicol and risk the chance of an adverse reaction (26).

Scrub typhus

Scrub typhus is also known as tsutsugamushi disease because it is caused by the obligate gram-negative coccobacillus *Orientia tsutsugamushi* (previously called *Rickettsia tsutsugamushi* but is now known to have a different genetic makeup and antigens from the *Rickettsia* genus) (30).

The bacterium is introduced through the skin by the bite from a trombiculid mite chigger, with a major role played by the genus *Leptotrombidium*. The first signs typically include headache, high fever, and myalgias, and these begin after a roughly 1- to 3-week incubation period (31). Additional symptoms may include nausea, vomiting and/or diarrhea, and cough. Approximately 5–8 days after the initial symptoms, a nonpruritic macular or papular rash may be seen on the trunk, which can later spread to the face, arms, and legs. Lymphadenopathy may also be present around the area of the bite or may even be generalized (31–33). Pathologically, scrub typhus is a focal or disseminated vasculitis and may involve multiple organ systems. Neck stiffness, tremor, delirium, and nervousness have also been reported (31,34). The presence of an eschar is important for diagnosis; it starts as a small papule that enlarges, develops a central necrotic area with ulceration, and later develops a cigarette-burn appearance with an erythematous halo surrounding a blackened crust (32,35). The eschar is found in the areas where you would expect a chigger bite, such as the waist where the waistband lays, or any other area that would constrict the clothing.

Millions of people have been infected with scrub typhus throughout Asia, with endemic areas including China, India, Japan, Korea, Taiwan, Thailand, Pakistan, and Malaysia. Anyone presenting with an eschar, lymphadenopathy, and a maculopapular rash in or recently from this region of the world should be considered to possibly have the disease. The differential diagnosis includes rickettsialpox, brucellosis, dengue fever, leptospirosis, and murine typhus. Indirect immunofluorescence antibody tests and Weil–Felix slide agglutination tests, which show high levels of serum antibody titer, are diagnostic (31–33,36), although, in the first 4–5 days of illness, antibody titers are often low. Polymerase chain reaction has also been used but may not be as readily available in all labs. Another difficult aspect to obtaining a diagnosis is that up to 77% of people living in an endemic area may have the antibody to *Ori. tsutsugamushi* (37–39).

Without treatment, scrub typhus lasts for 2–3 weeks and has a variable fatality rate. Successful treatment regimens have included doxycycline, tetracycline, chloramphenicol, and azithromycin. Chloramphenicol was the first medication proven to effectively treat scrub typhus. It is given in doses of 250–500 mg orally or intravenously every 6 hours and is an effective treatment still used in some endemic regions. Doxycycline is now considered the drug of choice. Studies have been carried out that compare the efficacy of doxycycline and chloramphenicol and were based on how long it took the patients to become afebrile and the relapse rate. There were no significant differences between the two treatment regimens (40). Doxycycline is given 100 mg orally or intravenously twice daily.

Tetracycline has also been tried and was compared with a short duration of doxycycline. In a 116-patient multicenter trial, scrub typhus was treated for 7 days with tetracycline or 3 days of doxycycline. The tetracycline patients were medicated four times daily at a dose of 500 mg each and the doxycycline patients were given 100 mg twice daily. The tetracycline group had a 100% cure rate, while the doxycycline group cured 94%. No relapses were noted in either group (41).

Azithromycin has also been considered for the treatment of scrub typhus, and it has been compared with treatment with doxycycline in 93 patients with mild disease in South Korea. Azithromycin-treated patients were given a one-time 500-mg dose compared with doxycycline-treated patients who took 200 mg once daily for 7 days. Both groups had no incidence of relapse and both regimens successfully reduced fever at a similar rate (42). Azithromycin has also been used in pregnant women with scrub typhus because of its relative safety profile. One study reported that eight women who were infected were given the one-time 500-mg dose of azithromycin, and all recovered and delivered healthy babies (43). Some strains of *Ori. tsutsugamushi* are known to be less susceptible to doxycycline and tetracycline, and azithromycin may be a reasonable alternative in areas where those strains are prevalent (44).

Combination therapy of doxycycline and rifampin was compared with doxycycline alone in areas of Thailand known for having strains of *Ori. tsutsugamushi* with reduced susceptibility to tetracyclines. Those treated with doxycycline alone had an average fever clearance time of 52 hours, whereas those who added daily doses of 900 mg of rifampin were afebrile on average after 22.5 hours, and those treated with a lower dose (600 mg) of

rifampin were clear after an average of 27.5 hours (45).

The ideal duration of therapy to prevent relapse has not been firmly established. One study of a 3-day therapy with either doxycycline or chloramphenicol showed relapse in three of six patients treated with doxycycline and three of seven treated with chloramphenicol within 4 weeks of completing therapy. However, 37 patients treated with either drug for 5 or more days had no relapses, which suggests that shorter courses of those drugs are less effective in preventing relapse (46).

There is currently no vaccine available for scrub typhus. Thus, mite control and chemoprophylaxis are the two avenues for infection prevention. DEET and permethrin are very effective in preventing bites from mites. One report of the effects of chemoprophylaxis had 20 individuals living and working in an environment where scrub typhus was endemic, with half of the individuals taking a once-weekly 200-mg dose of doxycycline, and the control group receiving no chemoprophylaxis. Both groups developed antibodies to scrub typhus (47). Only 1 of the 10 individuals treated with doxycycline developed clinical disease in contrast to the 9 of 10 who developed disease symptoms from the group that did not receive prophylaxis (47).

Avian, snake, bat, and tropical rat mites

Zoonotic acariasis, often referred to as gamasoidosis, is described as a human skin disease caused by nonburrowing hematophagous mites that typically feed on birds and other animals. These mites belong to the families Dermanyssidae and Macroonyssidae. The dermanyssid mites are ectoparasites of rodents and birds, whereas the macronyssid mites prey on reptiles, birds, and mammals. Avian mites belong to the two genera *Dermanyssus* (Family Dermanyssidae) and *Ornithonyssus* (Family Macroonyssidae). These include the following mites that have been associated with humans: *Dermanyssus gallinae* (chicken mite or pigeon mite), *Dermanyssus americanus* (American bird mite), *Dermanyssus hirundinis*, *Ornithonyssus sylviae* (Northern fowl mite), and *Ornithonyssus bursa* (tropical fowl mite), respectively. The two most isolated avian mites involved in human disease are *Derman. gallinae* and *Orn. sylviae* (48). Both of these are cosmopolitan mites with wide geographic distributions. Although the Tropical Fowl mite is found in both tropical and temperate zones, the Northern Fowl Mite is restricted to

temperate zones. Most commonly affecting chickens and pigeons, these mites infest a range of wild and domestic birds such as ducks, canaries, sparrows, robins, swallows, tiger finches, parakeets, starlings, and white wagtails (49,50). In the United States, these avian mites have had a significant economical impact on the poultry industry because of increased morbidity secondary to infestation (51).

Infestation in humans has been referred to as “avian mite dermatitis,” “bird mite rash,” “gamasoidosis,” and even the misnomer “chicken tick rash.” In 1809, Willan documented the first suspected case of human infestation with an avian mite, and it was not until 20 years later that an avian mite was isolated from human skin (52). Human cases typically result from occupational exposure and proximity to nesting birds and, occasionally, domestic pets. In poultry workers, these mites are responsible for producing dermatitis and asthma (53,54). Because *Dermanyssus* spp. are nocturnal feeders, most bites are incurred during the night or in darkened buildings. Numerous outbreaks in humans have been linked to infested birds’ nests, particularly pigeons, which were located on window ledges or air-conditioner in domiciles and hospitals (55). Most of these cases occur in the late spring and early summer when the fledgling birds leave their roost and the mites need to find an alternative food source. In addition to canaries and parakeets, pet rodents such as gerbils have been identified as a source of avian mite infestation (50).

The bite of *Derman. gallinae* may be painful and results in an erythematous papular rash that is often excoriated because of pruritus. Less commonly, vesicles, urticarial plaques, or diffuse erythema may be present. Upon close inspection, the mite may be identified as a small, red speck in the center of the lesion. However, in contrast to *Sarcoptes scabiei*, avian mites are difficult to recover from the skin, as they do not burrow and often emigrate rapidly from the host following a blood meal. Additionally, exposed areas including the face and interdigital web spaces of the hands and feet are not usually involved (56). Typically, the areas covered by clothing are affected including the arms and upper trunk with rare occurrences in the axillary and pubic regions. Although closely related to *Derman. gallinae*, the American bird mite *Derman. americanus* is rarely associated with human bites but may cause an acute generalized, eczematous dermatitis in the late spring (48). Another related avian mite, *Derman. hirundinis* has been documented to cause an urticarial der-

matitis (57,58). The tropical fowl mite *Orn. bursa* is associated with sharply painful bites and persistent itching. This avian mite can be found at all times on the host and, as a result, may induce more irritation than other avian mites manifesting as intensely pruritic erythematous papules. Avian mite dermatitis, regardless of the species of mite, may be treated topically with corticosteroids, and the itching may subside with oral antihistamines (50,56,57,59). For symptomatic relief, topical pramoxine in conjunction with topical corticosteroids and hydroxyzine has also been tried (60).

A host parasite, *Orn. sylvarum* completes the entirety of its life cycle on the host but can also be found in the surrounding habitat. A nocturnal predator, *Derman. gallinae* feeds on the host at night and hides in nesting material or the cracks and crevices of edifices during the day and therefore may not be found on the host during the day. Eradication of avian mites involves the removal of the infested birds and nesting material. For *Ornithonyssus* to be successfully eradicated, both the host and nesting material need to be treated, in contrast to *Derman. gallinae* infestation, which may only require treatment of the host. Ancillary house cleaning and fumigation with pyrethroid, organo-phosphorus, and carbamate-based acaricides may be helpful (61). More so for *Ornithonyssus* than for *Dermanyssus* infestations, affected individuals may be treated with topical acaricides such as gamma benzene hexachloride 1%, crotamiton, pyrethroid (e.g., Permethrin) or organophosphate (e.g., Malathion) compounds (60,62–64).

Avian mites have been implicated in the maintenance of several arboviruses in animal reservoirs and possible human transmission. Both *Derman. gallinae* and *Orn. bursa* have been associated with Western equine encephalitis, *Orn. sylvarum* and *Derman. gallinae* have been linked to Eastern Equine Encephalitis, and there are studies of *Derman. gallinae* and St. Louis encephalitis (65–67). However, although laboratory transmission has been demonstrated and arboviruses have been isolated from naturally occurring mites, the role of these mites in human transmission is unknown.

In addition to the *Ornithonyssus* avian mite species, the macronyssid mites that bite humans include *Ophionyssus bacoti* (tropical rat mite), *Ophionyssus natricis* (snake mite), and *Chiroptonyssus robustipes* (free-tailed bat mite). Although coined the “Tropical Rat Mite,” *Op. bacoti* (*Liponyssus bacoti*) affects wild and domestic rodents from temperate and tropical regions but not from the arctic and antarctic regions (68,69).

Primarily, this mite preys on the brown Norway rat (*Rattus norvegicus*) and the black roof rat (*Rattus rattus*). The snake mite *Op. natricis* affects both captive and sylvatic snakes. Preying on the Brazilian free-tailed bat (*Tadarida brasiliensis*), *Ch. robustipes* has a limited range involving Central America and the southern United States. These mites have been isolated from bat colonies in Alabama and Georgia (70,71).

The first documented case of human dermatitis that resulted from rat mites occurred in Australia in 1913 (72). In the United States, human cases have been identified in 36 states, the majority of which occur in the spring (73,74). Similar to that of the avian mite bites, the bite of *Op. bacoti* may be mildly painful and is followed by the development of pruritic small single or grouped 4- to 10-mm erythematous papules and occasional vesicles or urticarial plaques or nodules (73). Typically, bites occur where clothing fits tightly such as on the neck, shoulders, and waist, although all areas may be affected. The snake mite *Op. natricis* has been documented as the cause of a vesiculopapular eruption, mainly affecting the upper extremities, in a family who owned an infested python (75). One case of dermatitis secondary to bat mite infestation in California has been documented (76). Although there are case reports of the snake mite *Op. natricis* and the bat mite *Ch. robustipes* causing human dermatitis, these most likely represent rare occurrences associated with exotic pets and handling free-tailed bats or proximity to buildings with bat infestations in contrast to the tropical rat mite infestations, which are more prevalent (74–76).

Because *Op. bacoti* is a nest parasite and feeds on the host for a relatively short duration, treatment of the host with an acaricide is often unnecessary. Once the exposure is eliminated, the dermatitis is self-limited (77). Bathing will immediately remove *Op. bacoti* from humans and domestic animal. Treatment of rat mite dermatitis is symptomatic and may include antihistamines or topical corticosteroids (72,78). Previously used topical pesticides include gamma benzene hexachloride 1% (Lindane), alpha-chlordane and gamma-chlordane mixture (Chlordane), and topical benzyl benzoate 25% and 1% tannic acid (Acarosan) (73,79). When the primary host is not available, the rat mite is capable of surviving for prolonged periods of time and may travel several hundred feet in search of a blood meal (80). To prevent reinfestation to be prevented, the source of infestation should be eliminated. Rats should be exterminated, and the environment can be treated with 25% benzyl benzoate and 1% tannic acid,

malathion, methylcarbamate, and lindane, or fumigated with fluoridated silica aerogel dust (81). Similarly for snake mite and bat mite dermatitis, the affected individual may be treated symptomatically, and the source of infestation needs to be eliminated.

The tropical rat mite has been shown experimentally to transmit the following pathogens: murine typhus, rickettsialpox, Q fever, tularemia, plague, eastern equine encephalitis, epidemic hemorrhagic fever (Haantan), coxsackievirus, and Langat virus (81–85). This mite was implicated in the human transmission of murine typhus in 1931, but no cases since then have been reported (79).

Another Mesostigmatan mite, *Proctolaelaps pygmaeus* (Family Ascidae) was implicated in a case of an erythematous papular dermatitis in New Zealand (86,87).

Itch mites

Pyemotid mites are ectoparasites of insects, particularly the larval stages of beetles, hymenopterans, and lepidopterans that infest stored grains such as wheat, barley, straw, beans, peas, cottonseed, tobacco, hay, grass, and broom corn. The family Pyemotidae includes several species of mites that affect humans including *Pyemotes tritici* (*Pediculoides ventricosus*, *Pyemotes ventricosus*), *Pyemotes zwoelferi*, and *Pyemotes herfsi*. These mites have an unusual life cycle. The gravid female may swell up to 2 mm in diameter. Pyemotes are oviparous, and gravid females will give birth to more than several hundred immature adults. Males are born first and fertilize the females. The females then migrate to a new source of food (88). These mites will infest humans as incidental hosts, particularly if their natural prey is eliminated with prolonged storage of grain or because of environmental conditions.

In 1909, the first case of *Pyemotes* dermatitis in the United States was reported in association with hay mattresses in Indiana (89). The most common species affecting humans, *Py. tritici* is also known as the “grain itch mite,” “hay itch mite,” and the “straw itch mite” (FIG. 3). This mite is globally distributed, and outbreaks of pruritic human dermatitis have occurred in Europe, Africa, Eastern Asia, Australia, and the United States. (88,90–93). Human bites are typically associated with individuals handling straw or hay, such as agricultural workers, but may also affect individuals handling the same raw materials used in decorative arts and crafts (93). Massive outbreaks have occurred

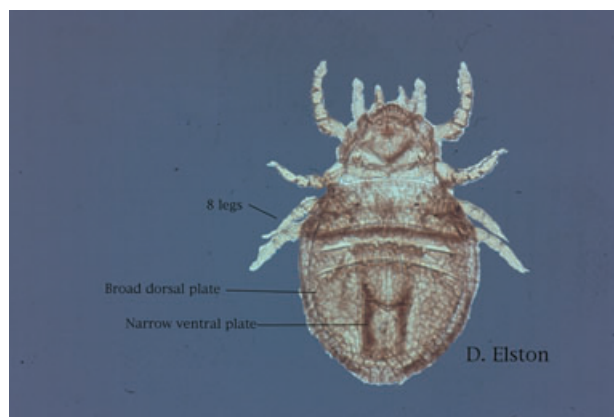


FIG. 3. Straw itch mite.

including more than 1000 Indiana State Fair participants who developed dermatitis secondary to *Pyemotes*-infested straw that had been used for animal bedding (94). Additional outbreaks of *Pyemotes tritici* have been associated with termite-infested wood and infestation with the common furniture beetle (95).

First identified in Europe in 1936, *Py. herfsi* is an itch mite that preys on oak leaf gall-making midge larvae. *Py. herfsi* has been reported as a cause of papular dermatitis in Czechoslovakian workers exposed to *Pyemotes*-infested dust that had accumulated in the loft above a porcine food shed (96). Although not indigenous to the United States, *Py. herfsi* was implicated as the cause of the outbreak of a pruritic rash in Kansas because of infested leaf galls in surrounding pin oak trees (97). The majority of the affected individuals had an erythematous papular rash, although one-fifth presented with pustules, macules, or confluent erythema. Almost half of the individuals had a rash on the neck and arms, with the trunk less commonly affected. Typically, pruritus developed 10–16 hours after incurring mite bites. Systemic symptoms including fever, wheezing, and rhinorrhea were absent. The rash resolved within 1 week, and symptomatic treatment with calamine lotion, an oral antihistamine, and a topical hydrocortisone cream was recommended. Suggested preventative measures included using DEET before going outdoors, wearing light-colored and tightly woven clothing, and keeping windows closed in wooded areas with leaf galls. Insecticide spraying was not performed because the mites are unexposed within the leaf galls (98).

In addition to *Py. tritici* and *Py. herfsi*, *Py. zwoelferi* has also been reported to cause human dermatitis. An erythematous vesicopapular eruption lasting 2–3 weeks occurred in individuals who

came in contact with mite-infested bunches of dried everlasting flowers, *Helichrysium angustifolium*, that had been imported from Yugoslavia (99,100).

The bites of *Pyemotes* spp. result in a pruritic dermatitis that has been referred to as “Barley Itch,” “Grain Itch,” “Straw Itch,” “Hay Itch,” etc., depending on the infested source. The bites, which are numerous and intensely pruritic, typically affect covered sites especially the trunk and proximal extremities (95). Human bites are associated with intense itching following piercing of the skin and envenomation with potent neurotoxin peptides, Txp-I and TxpII, which result in muscle contraction and paralysis of the mites’ insect prey, that is, moth larvae (101–103). Vesicular urticarial lesions, often described as varicelliform, may form in which a vesicle develops on an erythematous base in the center of a white wheal. The mite may be identified in the center of the vesicle as a tiny white speck. In 1–2 days, the vesicle may become turbid and contain a purulent material (95). If the source of the mites is removed, the eruption is self-limited and will resolve in 1–3 weeks. Systemic signs are rare but may include fever, chills, malaise, and diarrhea (93). Treatment is symptomatic with topical corticosteroids and antihistamines and resolution depends on the elimination of the source of the infesting mites (104).

To eliminate *Pyemotes* mites, infested areas may be treated with acaricides. Carbamate insecticides such as methyl bromide and carbaryl have been used as pesticides, although methyl bromide was removed from the market in 2005 (88,90). Pyrethrin insecticides have also been used with some success (88,93,105). Sulfur or gamma benzene hexachloride ointment, combined with olive oil application, has been reported as a repellent (94,106). And, more recently, dusting sulfur on an infested area may be used to eliminate the mite population (107).

Another Prostigmatan mite, *Balaustrium murorum* (Family Erythraeidae) has rarely been associated with human dermatitis. Insectivorous and herbivorous, this mite typically dwells outdoors on plants and soils, although, owing to its bright red appearance, it can be conspicuous on the walls of buildings during summer. The first documented human case of *Balaustrium* dermatitis occurred in 1951 and involved an entomologist working in a museum who was bitten on the upper left arm (108). He had developed an erythematous pruritic papule that persisted for 3–4 days. In 1956, an outbreak of human dermatitis due to *Balaustrium* mites occurred in a Los Angeles hospital in

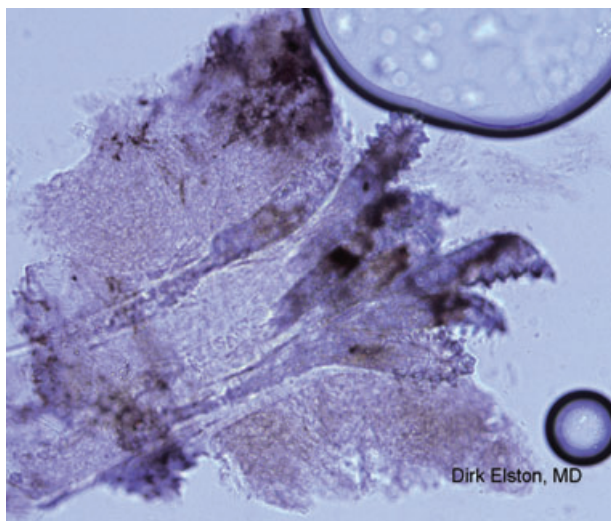


FIG. 4. *Demodex* mites demodectic folliculitis.

which pediatric patients were bitten because of their proximity to heavily mite-infested hospital grounds (108). In 1998, in Japan, a debilitated woman developed an erythematous urticarial, popular, and ecchymotic rash. *Balaustrium murorum* was identified in the patient's hospital room, and, following eradication, the rash resolved (109). In addition to nosocomial attacks and incidental exposure, environmental exposures may predispose individuals to *Balaustrium* attacks as evidenced by workers who had been attending a mite-infested apple orchard (109).

Follicle mites

The follicle mites include *Demodex folliculorum* and *Demodex brevis*. Infestation with *Demodex* spp., or Demodicosis, may present with a spectrum of clinical findings ranging from asymptomatic erythema to severe dermatitis with alopecia. Both feeding on sebum, *Dem. folliculorum* typically resides in hair follicles, whereas *Dem. brevis* survives in sebaceous glands adjacent to hair follicles (FIG. 4). These mites populate facial areas such as the forehead, eyebrows, eyelids, and nose. Occasionally, they may be found on the perioral mucosa, ear canal, chest, and other body regions. The mites are part of the normal flora of human skin and only present a problem when present in large numbers or with altered host immunity. In heavy infestations, the mites plug the follicular openings with subsequent inflammation and pathologic changes. Clinical variants that result from demodex infestation include pityriasis folli-



FIG. 5. Papular demodicosis of the face.

culorum, Rosacea-like demodicidosis, demodex blepharitis, pustular folliculitis, papulopustular scalp eruptions, perioral granulomatous dermatitis, solitary granuloma, papular demodicosis of the face (FIG. 5), follicular spinulosus, seborrheic dermatitis-like lesions, nonspecific facial pruritus with or without erythema, and pseudozoster (110–115).

One of the first variants described, Pityriasis folliculorum presents with faint facial erythema with a burning sensation and with fine scaly follicular plugs. This condition was associated with middle-aged women who used heavy moisturizers compounded with infrequent washing. *Demodex* has been implicated in the pathogenesis of rosacea, although the association has not been unequivocally determined. A distinct clinical condition that closely mimics rosacea, Rosacea-like demodicidosis, presents with follicular scale and superficial erythematous vesiculopustules that involves the lateral as well as the central face. Unlike rosacea, clinically, there are no telangiectasias, and, historically, patients deny flushing, persistent erythema, or photosensitivity. In demodicidosis gravis, erythematous papulonodules are present with dermal granulomas containing mite remnants phagocytized by foreign-body giant cells and showing central caseation necrosis on histology (116). Around the eye, *Dem. folliculorum* is found in the follicles of the eyelashes, whereas *Dem. brevis* inhabits the sebaceous gland of the eyelashes and the meibomian glands (117). The association of demodex with blepharitis, similar to rosacea, has not been fully delineated (118,119). demodex infestation in the eyelashes has been associated with cyclical dandruff, trichiasis, meibomian gland dysfunction with lipid tear deficiency, and conjunctival inflammation (120).

Human demodectic mange resembles canine demodectic mange, and individuals may present with erythema, hyperkeratosis, and scaling in addition to alopecia. Scalp demodectic mange may present with crusted erythematous scaly plaques and mimic favus (121). In patients with human immunodeficiency virus, especially when the CD4 count is lower than 200/mm³, *Demodex* mites have been associated with pruritic eruptions of the face, presternal, and interscapular areas. *Demodex* may also present a problem in other immunocompromised individuals, such as those with malignancies, that is, acute myelocytic leukemia (122–126).

Because these mites are universally present, pathogenicity is not thought to be secondary to mite presence unless five or more mites are identified in a single, low-power field prepared with potassium hydroxide preparation, or more than 5/cm² in a standardized skin biopsy (127,128). Various treatments have been used in demodicidosis including topical salicylic acid, metronidazole, crotamiton, lindane, and sublimed sulfur, oral metronidazole, oral ivermectin together with topical permethrin, and oral or topical retinoids (110,111,129–131). Topical sulfur is recommended as an initial treatment, even prior to permethrin and ivermectin (132). Permethrin 5% has been successful in treating some patients with this condition with human demodectic mange (133). A case of scalp demodex folliculitis that did not improve with clindamycin 1% gel eventually resolved with topical sulfacetamide 10% and sulfur 5% cream in addition to selenium sulfide 2.5% shampoo (134). One case exists of a demodex abscess that did not respond to topical therapy but eventually cleared with oral metronidazole 250 mg three times a day for 2 weeks (135). Facial demodex also responds to oral metronidazole used in conjunction with topical dilute camphor oil (136). *Demodex* infestation of the eyelids may be treated with topical tea tree oil 50% weekly and daily tea tree shampoo to eradicate this mite infestation (120).

Immunocompromised individuals may present with more challenging cases of demodicidosis. In patients with Human Immunodeficiency Virus (HIV), ivermectin has been successfully used to clear follicle mite infestation (137). In a recent case series of children with leukemia undergoing chemotherapy, none of the five children cleared with topical permethrin 5% cream, and four received limited benefit with topical metronidazole. Complete resolution in one child occurred with a formulation of topical sodium sulfacetamide 10% and sulfur 5% (125). In another case, a child with acute lymphoblastic leukemia was partially treated with

topical metronidazole applied twice a day for 2 weeks and completely cleared with oral erythromycin and two overnight applications of 1% lindane cream, each 1 week apart (123).

Storage (food) and dust mites

Storage mites are a significant cause of occupational allergies in agricultural workers. Storage mites, upon repeated exposure, may cause an allergic contact dermatitis, inhalational allergy, bronchial asthma, and even anaphylaxis. The mites most commonly implicated in human exposures belong to the families Acaridae, Carpoglyphidae, and Glycyphagidae. These mites proliferate in a warm, humid environment with relative humidity of 80% and a temperature between 25 and 30°C. Herbivorous and fungivorous, they subsist on fungi and are pests of stored food products with high moisture content. Dermatitis may be secondary to bites, as the mite attempts to find moisture or to contact with allergens from cast exoskeletons, feces, or ova.

The mites belonging to the family Acaridae include *Tyrophagus longior*, *Tyrophagus putrescentiae*, *Acarus siro*, *Acarus farris*, and *T. casei*. In the early 1900s, Castellani first described an occupational exposure to a storage mite. He identified the dermatitis among workers handling coconut kernel (copra) as copra itch, and the causative agent of the papulopustular eruption was later identified as the mite *T. longior* (138). This mite is also known as a “mold mite,” given its close relation to *T. putrescentiae*, also known as the “Mold Mite.” Fungivorous, *T. putrescentiae* infests comestibles with a high lipid and protein content such as cured ham, cheese, smoked herring, dried fruit, and grains (139,140). Ubiquitous, this mite has been found in roots of plants, bulbs, and mushrooms in addition to stored foods (141). This storage mite has caused allergic dermatitis in handlers of cured ham in addition to other foods (140). Additionally, *T. putrescentiae* is responsible for occupational inhalational allergies associated with farms, dairy barns, bakeries, and grain elevator stores (142–144). And, airborne contact dermatitis has been reported as occurring from this storage mite (145).

The most important acarid mite in agricultural occupational disease is *A. siro* (*Tyrophagus siro*). This mite is considered one of the most abundant mites in stored plant products (146). This mite is responsible for food spoilage in addition to affecting human health. *A. siro* is associated with contact allergies in agricultural workers, bakers, and

cheese producers (147). This mite may cause “grocer’s itch,” “baker’s itch,” and “vanillism.” Additionally, this pest is increasingly identified in house dust and may contribute to bronchial asthma and environmental allergies (148). A sibling species complex, *Acarus* spp. is considered three species with identical morphological characteristics but different ecological preferences; *A. farris* and *Acarus immobilis* occur primarily outdoors, whereas *A. siro* prefers indoor storage sites (149,150). *A. farris* has been reported as a cause of human dermatitis in hay workers in Europe (147).

The cheese mite, *Tyrolichus casei* (*Tyrophagus casei*) may be found in other stored foods including grains and flour but is most commonly found on cheese. Typically, this mite is often found in addition to other species of storage mites (151). Cheese mites have been associated with dermatitis in cheese handlers (141). At an ambient temperature of 70 °F, these mites will complete their life cycle in 2 weeks, whereas, at 40°F, an entire month may lapse prior to the completion of a life cycle (152). Although cheese mites can live at low temperatures, they will not survive in the refrigerator. Additionally, a thin layer of paraffin wax is typically used as a protective covering for cheeses.

A by-product of the flour milling of grain, wheat pollard has been associated with infestations of *Suidasia nesbitti*, another Astigmatan mite. The dermatitis that workers develop after handling wheat pollard is known as “wheat pollard itch” (147).

Other mites that cause contact dermatitis belong to the family Carpglyphidae, (*Carpoglyphus lactis*) and Glyciphagidae (*Glyciphagus domesticus*). *Ca. lactis* (*Carpoglyphus passularum*)-infested dates were responsible for a scabies-like eruption of the arms and face in two women who were sorting the partially damaged dates (153). A similar rash was described in a dock-worker handling figs infested with *Ca. lactis*. On both of his forearms and hands, he had developed 1- to 2-mm, erythematous papules with scabs reminiscent of human scabies (154). Also known as the “furniture mite,” *G. domesticus* causes vesicular dermatitis in skin (155).

Historical accounts exist of a mite *Rhizoglyphus parasiticus* as a cause of “water-itch” or “tea dermatosis,” a vesicular eruption that affected the feet of workers in the Assam tea plantations during the wet season (154). However, it was later believed that hookworm infestation accounted for the dermatitis. However, commonly found on plant bulbs, *Rhizoglyphus* spp. have been associated with “bulb dermatitis” (147). Additionally, *Rhizog-*

lyphus has been recently identified as a cause of dermatitis in a cheese handler (156).

For both occupational and domestic exposures, the dermatitis is self-limited, and the elimination of the source of the mite is the primary concern. Storage mite dermatitis may also be symptomatically treated with a topical corticosteroid and oral antihistamine. Elimination of storage mites has previously relied on spraying with residual pesticides such as the organophosphorus compounds. Given concern about their potential human toxicity, alternative control measures are being investigated. These include insect growth regulators, inert dusts, and plant-derived acaricidal products, among others (157). Inert dusts such as silica gel or diatomaceous earth have been used successfully as stored-grain protectants (158). Plant products such as Benzyl benzoate, derived from the Peruvian tree *Myroxylon balsamum* var. *pereirae* Harms (Family Fabaceae) has also been employed with success in the control of storage mites in addition to dust mites (157). Another plant-derived acaricide, pyrethroids are also effective against storage mites and degrade slowly (157). Control measures for *A. siro* have included waxing the surface of the cheese and fumigation with methyl bromide (152). Although lindane has been used, there has been at least one account of lindane resistance in *A. siro* (159).

The allergens of pyroglyphid and other storage mites are also involved in dermatitis and inhalational allergies. Astigmatan mites responsible for dust mite allergy belong to the families Pyroglyphidae, Acaridae, Glyciphagidae, and Chortoglyphidae. More than 10 different species of mites have been identified in house dust. The American house-dust mite *Dermatophagoides farinae*, European house-dust mite *Dermatophagoides pteronyssinus*, and *Euroglyphus maynei* are the most common sources of allergen (160). Additionally, *Blomia tropicalis* (Family Echymopodidae) has been identified as an important contributor to dust mite allergies in tropical climates (161). *Dermatophagoides scheremetewskyi* causes feather pillow dermatitis, which presents as erythematous papules on the face, neck and head, has been associated with infested feather pillows (162,163). Storage mites also are found in dust and may contribute to allergenicity including *Glyciphagus domesticus*, *Lepidoglyphus destructor*, *T. putrescentiae*, *A. siro*, and *Chortoglyphus ancutatus*.

In addition to inhalational allergy and asthma, atopic dermatitis has been associated with sensitization to mite allergens (161). Individuals with atopic dermatitis may exhibit immediate and

delayed-type skin reactivity to mite antigens (164). Although the role of dust mite sensitization in atopic dermatitis is not completely known, allergen avoidance measures have been recommended given clinical improvement in eczema following dust mite control (165). Reduction of mite population and allergens is achieved by following several recommendations. Environmental relative humidity should be maintained below 50%, and carpets, draperies, and upholstery that may retain moisture and debris should be discarded to limit ideal breeding conditions for mites. Mite allergens may be decreased by encasing mattresses and pillows in protective coverings and washing sheets, mattress pads, and other bedding once weekly in hot water, 130°F. In addition, weekly carpet vacuuming may decrease dust mite populations (161).

In addition to inhalational and cutaneous allergies, storage and dust mites may cause anaphylaxis following ingestion of contaminated foods. Four cases of anaphylaxis have been reported with the ingestion of *T. putrescentiae* and *Dermat. farinae* contaminated “okonomiyaki,” a flour-containing Japanese dish (166–168). Several cases of ingested-mite anaphylaxis have resulted from storage mite-infested wheat flour pancakes, and the syndrome has been designated “the pancake syndrome” (169). In addition to accidental ingestion of *Dermat. farinae*, ingestion of contaminated foods with storage mite *Blomia freemani* and *S. nesbitti* may cause anaphylaxis (170–172).

Mange and scabietic mites

Astigmatid mites, *Psoroptes* and *Sarcoptes* mites cause scabietic and mange disease in domestic and wild animals. Humans who come in contact with infested animals may be inadvertently affected. Scab mites belong to the family Psoroptidae and parasitize domestic and wild mammalian hosts. These mites pierce the skin to feed on blood or ingest sloughed skin, survive on the epidermis, and do not burrow. Psoroptic mange results from the inflammation and tissue damage inflicted by the scab mites with extensive pruritus, oozing and crusting, and hair loss. Originally, five species of mites (*Psoroptes cervinus* (elk scab mite), *Psoroptes cuniculi* (rabbit ear mite), *Psoroptes natalensis* (cattle mite), *Psoroptes ovis* (sheep scab mite), *Psoroptes equi* (horse mite)) were identified as causes of mange depending on the host preference; however, molecular evidence suggests that these species may not be separate entities (173–175). *Ps. ovis*, the cause of sheep scab, may also affect

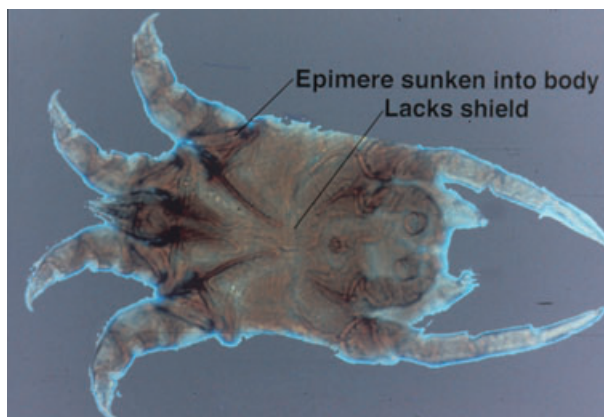


FIG. 6. *Psoroptes* mite.

humans. In sheep, *Ps. ovis* causes inflammation with pruritus and subsequent oozing and serous crusting secondary to rubbing and scratching. These “scabs” affect wool-covered areas, and eventually the inflammation leads to dislodged wool with inadvertent shedding. The animals become emaciated and may die (176). In humans, Psoroptic skin disease resembles scabies infestation. Typically, the distribution of the dermatitis is dependent upon the areas that come in close contact with the animals. In previous study of sheep scab affecting humans, the most heavily infested sites were the upper extremities then the lower extremities, with the face, ears, genitalia, and abdomen less commonly involved (FIG. 6)(177).

Individuals with Psoroptic infestation should be treated with a topical acaricide, such as permethrin 5% cream (176). Although there are no published human accounts of Psoroptic mange treated with ivermectin, psoroptic mange in animals has been treated with the endectocides ivermectin, doramectin, and moxidectin (178). Two doses of ivermectin 1 week apart may be required, given that *Ps. ovis* females can survive off the body for up to 15 days. Infested animals should also be treated with either oral or topical acaricides. Topical Selamectin has been shown to significantly limit psoroptic and sarcoptic mite infestation in rabbits (179). Additionally, the nesting or bedding material should be discarded and disinfected.

Zoonotic scabies are *Sarcoptes* spp. that primarily affect other animals including dogs, pigs, and cats but may also infest humans. The zoonotic mites are closely related to the human scabies mite, and studies suggest that *Sarcoptes scabiei* mites probably infected man originally, and then were transmitted to as many as 39 different animal species (180). Individuals with zoonotic scabies are involved in animal husbandry or own pets and

Table 1. Summary of mite infestations

Mite	Common name (s)	Clinical disease	Clinical findings
<i>Cheyletiella yasguri</i> (dogs)		In humans: Dermatitis	In humans: Grouped pruritic papules in areas of pet contact, less common are widespread papules, urticarial wheals, and vesiculobullous lesions
<i>Cheyletiella blakei</i> (cats)		In animals: "Walking dandruff"	
<i>Cheyletiella parasitivorax</i> (rabbits)			In animals: Mild to heavy dorsal dermatitis with or without alopecia and pruritis
<i>Liponyssoides sanguineus</i> (formerly <i>Allodermanyssus sanguineus</i>)	House mouse mite	Rickettsialpox	Initial eschar at site of bite, followed by fever and malaise. A generalized maculopapular rash that progresses to papulovesicular lesions that scab over and fall off
Trombiculid mite larvae such as <i>Leptotrombidium akamushi</i>	Chigger Harvest bug Harvest lice	Scrub typhus (also known as tsutsugamushi disease)	Headache, high fever, and myalgias, possibly nausea, vomiting, diarrhea, and cough. Rash starts as a nonpruritic macular or papular rash on trunk that may spread to face, arms, and legs. May have an eschar, and lymphadenopathy may be present.
<i>Leptotrombidium deliense</i>	Jigger		
<i>Leptotrombidium fletcheri</i>	Mower's mite		
<i>Leptotrombidium arenicola</i>	Red bug		
<i>Leptotrombidium pallidum</i>	Scrub mite		
<i>Leptotrombidium pavlovskyi</i>			
<i>Leptotrombidium scutellare</i>			
<i>Dermanyssus gallinae</i> (formerly <i>D. avium</i>)	Red poultry mite Chicken mite Pigeon mite	Avian mite dermatitis Eastern equine encephalitis ^a Western equine encephalitis ^a St. Louis encephalitis ^a	Painful bite Erythematous papules, excoriations, and less commonly, vesicles, urticarial plaques, or diffuse erythema affecting areas covered by clothing, with sparing of the axillary and genital region and not the face and web spaces of the hands and feet
<i>Ornithonyssus bursa</i>	Tropical fowl mite	Avian mite dermatitis Western equine encephalitis ^a	
<i>Ornithonyssus sylvarium</i>	Northern fowl mite	Avian mite dermatitis Eastern equine encephalitis	
<i>Dermanyssus americanus</i>	American bird mite	Avian mite dermatitis	Acute generalized, eczematous dermatitis
<i>Dermanyssus hirundinis</i>		Avian mite dermatitis	Urticarial dermatitis
<i>Chiroptonyssus robustipes</i>	Free-tailed bat mite	Bat mite dermatitis	Erythematous papules and excoriations
<i>Ophionyssus bacoti</i> (formerly <i>Liponyssus bacoti</i>)	Tropical rat mite	Rat mite dermatitis Murine typhus ^a Rickettsialpox ^a Tularemia ^a Plague ^a Coxsackievirus ^a Q fever ^a Epidemic hemorrhagic fever (Hantaan virus) ^a Langat Virus ^a	Painful bite Erythematous papules, excoriations, and less commonly, vesicles, urticarial plaques, or diffuse erythema affecting areas covered by clothing, with sparing of the axillary and genital region and not the face and web spaces of the hands and feet
<i>Ophionyssus natricis</i>	Snake mite	Snake mite dermatitis	Erythematous papules and vesicles

Table 1. Continued

Mite	Common name (s)	Clinical disease	Clinical findings
<i>Proctolaelaps pygmaeus</i>		Proctolaelaps dermatitis	Erythematous papules
<i>Demodex folliculorum</i> <i>Demodex brevis</i>	Follicle mite	Demodicidosis	Pleomorphic presentation with erythematous papules, pustules, nodules, vesicles with facial erythema +/- burning sensation and pruritus, typically affecting the face and scalp
<i>Pyemotes tritici</i> (formerly <i>Pediculoides ventricosus</i> , <i>Pyemotes ventricosus</i>) <i>Pyemotes zwoelferi</i> <i>Pyemotes herfsi</i> <i>Balaustium murorum</i>	Straw itch mite Hay itch mite Grain itch mite	Straw itch (hay itch, grain itch)	Pruritic erythematous urticarial papules surmounted by pinpoint vesicles that become purulent
<i>Acarus siro</i> (formerly <i>Tyrophagus siro</i>) <i>Acarus farris</i>	Flour mite Cereal mite Grain mite	Storage mite dermatitis Baker's itch Grocer's itch Vanilla itch Hay itch	Erythematous pruritic papules with possible urticaria and ecchymoses Contact: Erythematous pruritic papules without burrows, excoriations, and vesicles on exposed areas including hands and face
<i>Tyrophagus putrescentiae</i> <i>Tyrophagus longior</i>	Mold mite Grain stack mite	Storage mite dermatitis	Inhaled: Asthma, rhinitis, rhinoconjunctivitis, and possible anaphylaxis
<i>Tyrolichus casei</i>	Cheese mite	Storage mite dermatitis Cheese mite dermatitis	
<i>Suidasia nesbitti</i> <i>Carpoglyphus lactis</i> (formerly <i>C. passularum</i>)	Wheat Pollard mite Fruit mite	Wheat Pollard itch Storage mite dermatitis Dried-fruit-mite dermatitis	
<i>Glyciphagus domesticus</i> <i>Lepidoglyphus destructor</i> <i>Rhizoglyphus</i> spp.	Grocer's itch mite House mite Cosmopolitan food mite Dust mite, hay mite Bulb mite	Storage mite dermatitis Grocer's itch Storage mite dermatitis	
<i>Dermatophagoides farinae</i> <i>Dermatophagoides pteronyssinus</i> <i>Dermatophagoides schereemetewski</i> <i>Euroglyphus maynei</i> <i>Blomia tropicalis</i> <i>Blomia freemani</i>	Dust mite	Storage mite dermatitis Bulb dermatitis Contact and inhalational allergy	Contact: Exacerbation of atopic dermatitis. Inhaled: Asthma, rhinitis, rhinoconjunctivitis, and possible anaphylaxis
<i>Psoroptes ovis</i> (sheep) <i>Psoroptes cervinus</i> (elk) <i>Psoroptes cuniculi</i> (rabbits) <i>Psoroptes natalensis</i> (cattle) <i>Psoroptes equi</i> (horses)	Scab mite	Psoroptic mange	Erythematous pruritic papules without burrows, excoriations, and vesicles on exposed areas of contact including hands and face
<i>Sarcoptes scabiei</i> var. <i>canis</i> (dogs)	Canine scabies mite	Canine scabies, zoonotic scabies	Erythematous pruritic papules with burrows, excoriations, and vesicles on exposed areas of contact including hands and face

^aPathogen has been isolated although the vectorial capacity is not well established.

have come in contact with an affected animal. In the canine host, *S. scabiei* var. *canis* causes pruritus and hair loss secondary to inflammation and scratching. Typically, human manifestations present as a pruritic papulovesicular eruption lasting 1–3 months, unlike human scabies, which may last several years if left untreated (181). Infestation generally manifests in humans on areas of contact with the affected pet, such as the forearms, thighs, chest, and abdomen. In children, the distribution resembles that of *S. scabiei* var. *hominis*, which involves the palms, finger webs spaces, head, and neck (182,183). Depending on the strain of mite, the lapse between infestation and development of skin lesions varies. *S. scabiei* var. *canis* may penetrate human skin in less than 30 minutes following contact, and the rash may develop within 24–96 hours of contact with the affected animal (181,184). In contrast, the pruritic erythematous papular eruption of *S. scabiei* var. *hominis* usually presents strain 4–6 weeks following contact because of the development of a delayed type IV hypersensitivity reaction (181).

Microscopic examination of skin scrapings may reveal mites, eggs, or feces (scybala). On clinical exam, mite burrows may be accentuated by applying India ink to the skin followed by removal with alcohol. The burrows will be highlighted by the residual ink staining. Epiluminescence stereomicroscopy may identify the presence of air bubbles with a “jet-with-contrail” appearance that may represent the mite and burrow (185). Skin biopsy may demonstrate a dense, perivascular infiltrate of mononuclear cells with numerous eosinophils around papillary and reticular dermal vessels. The presence of a mite may or may not be noted. The “pink pigtail” sign has been recently identified as a possible marker of scabies infestation. Review of several cases suggested that “pigtail-like structures” in the stratum corneum are egg shell fragments attached to the epidermis and are left behind after the mite hatches (186).

Canine scabies causes a self-limited infestation in humans and treatment of the affected animal will often prevent reinfestation. Higher relative humidity and lower temperatures prolong survival, and *S. scabiei* var. *canis* mites can survive between 1 and 2 days off the host (184). Despite being self-limited, the skin eruption may be uncomfortable, and topical treatment with a scabicide such as permethrin 5% cream will hasten recovery. If further contact with the infested animal is prevented, treatment may only require one application of the topical acaricide. Previously, topical lindane 1% and topical crotamiton 10% have been used suc-

cessfully for patients with canine scabies (182). In refractory cases, a single dose of oral ivermectin 200 mcg/kg has been suggested (181). Similar as in other mite infestations, topical corticosteroids, menthol preparations, and oral antihistamines may be of benefit. In both Psoroptic and Scabetic infestation, successful eradication requires removal or treatment of the infested animal from which the mites were transferred.

Conclusions

Mite infestations are important in dermatology, and the present study reviewed etiology, clinical manifestation, and treatment of mite infestations. Dermatologic manifestations of mite infestation range from papulosquamous eruptions to urticarial lesions to bullous eruptions. Mite-associated diseases may have systemic complications and may be confused with other dermatologic conditions. Treatment options include topical preparations, such as crotamiton and permethrin, topical and intralesional corticosteroids, and oral antibiotics. The treatment depends on the etiology of the infestation. Prevention of infestation may be accomplished by pre-treating clothing with permethrin, by using insect repellent DEET on clothing and skin, and treating animals infected with mites (Table 1).

References

1. Potts J. Eradication of ectoparasites in children. How to treat infestations of lice, scabies, and chiggers. *Postgrad Med* 2001; **110**: 57–59, 63–64.
2. Gasser R, Wyniger R. [Distribution and control of Trombiculidae with special reference to *Trombicula autumnalis*.]. *Acta Trop* 1955; **12**: 308–326.
3. Hase T, Roberts LW, Hildebrandt PK, Cavanaugh DC. Stylostome formation by *Leptotrombidium* mites (Acari: Trombiculidae). *J Parasitol* 1978; **64**: 712–718.
4. Elston DM. What's eating you? Chiggers. *Cutis* 2006; **77**: 350–352.
5. Millikan LE. Mite infestations other than scabies. *Semin Dermatol* 1993; **12**: 46–52.
6. Smith GA, Sharma V, Knapp JF, Shields BJ. The summer penile syndrome: seasonal acute hypersensitivity reaction caused by chigger bites on the penis. *Pediatr Emerg Care* 1998; **14**: 116–118.
7. Rivers JK, Martin J, Pukay B. Walking dandruff and Cheyletiella dermatitis. *J Am Acad Dermatol* 1986; **15**: 1130–1133.
8. Platt RE, Wiemers J. A survey of fur mites in domestic rabbits. *Lab Anim Sci* 1976; **26**: 758–761.
9. Kunkle GA, Miller WH Jr. Cheyletiella infestation in humans. *Arch Dermatol* 1980; **116**: 1345.

10. Bronswijk JE, De Kreek EJ. Cheyletiella (Acari: Cheyletiellidae) of dog, cat and domesticated rabbit, a review. *J Med Entomol* 1976; **13**: 315–327.
11. Cohen SR. Cheyletiella dermatitis. A mite infestation of rabbit, cat, dog, and man. *Arch Dermatol* 1980; **116**: 435–437.
12. Shelley ED, Shelley WB, Pula JF, McDonald SG. The diagnostic challenge of nonburrowing mite bites. Cheyletiella yasguri. *JAMA* 1984; **251**: 2690–2691.
13. van Bronswijk JE, Jansen LH, Ophof AJ. Invasion of a house by the dog parasite Cheyletiella yasguri (Smiley 1965), a mite causing prurigo in man. *Dermatologica* 1972; **145**: 338–343.
14. Scott D, Miller W, Griffin C. Parasitic skin diseases. In: Small animal dermatology, 5th ed. Philadelphia: WB Sanders, 1995: 412–417.
15. Lee BW. Cheyletiella dermatitis: a report of fourteen cases. *Cutis* 1991; **47**: 111–114.
16. Ottenschot TR, Gil D. Cheyletiellosis in long-haired cats. *Tijdschr Diergeneesk* 1978; **103**: 1104–1108.
17. Klayman E, Schillhorn van Veen TW. Diagnosis of ectoparasitism. *Mod Vet Pract* 1981; **62**: 767–771.
18. Chadwick AJ. Use of a 0.25 per cent fipronil pump spray formulation to treat canine cheyletiellosis. *J Small Anim Pract* 1997; **38**: 261–262.
19. Folz SD, Kakuk TJ, Henke CL, Rector DL, Tesar FB. Clinical evaluation of amitraz for treatment of canine scabies. *Mod Vet Pract* 1984; **65**: 597–600.
20. Elston DM. What's eating you? Cheyletiella mites. *Cutis* 2004; **74**: 23–24.
21. Paterson PY, Taylor W. Rickettsialpox. *Bull N Y Acad Med* 1966; **42**: 579–587.
22. Nichols E, Rindge ME, Russell GG. The relationship of the habits of the house mouse and the mouse mite (Allodemnyssus sanguineus) to the spread of rickettsialpox. *Trans Am Clin Climatol Assoc* 1952; **64**: 169–184.
23. Levine JF, Lage AL. House mouse mites infesting laboratory rodents. *Lab Anim Sci* 1984; **34**: 393–394.
24. Greenberg M, Pellitteri OJ, Jellison WL. Rickettsialpox – a newly recognized rickettsial disease: III. Epidemiology. *Am J Public Health Nations Health* 1947; **37**: 860–868.
25. Kass EM, Szaniawski WK, Levy H, Leach J, Srinivasan K, Rives C. Rickettsialpox in a New York City hospital, 1980 to 1989. *N Engl J Med* 1994; **331**: 1612–1617.
26. Brettman LR, Lewin S, Holzman RS, et al. Rickettsialpox: report of an outbreak and a contemporary review. *Medicine (Baltimore)* 1981; **60**: 363–372.
27. Greenberg M, Pellitteri OJ. Rickettsialpox. *Bull N Y Acad Med* 1947; **23**: 338.
28. Radulovic S, Feng HM, Morovic M, et al. Isolation of Rickettsia akari from a patient in a region where Mediterranean spotted fever is endemic. *Clin Infect Dis* 1996; **22**: 216–220.
29. Rose HM. The clinical manifestations and laboratory diagnosis of rickettsialpox. *Ann Intern Med* 1949; **31**: 871–883, illust.
30. Tamura A, Ohashi N, Urakami H, Miyamura S. Classification of Rickettsia tsutsugamushi in a new genus, Orientia gen. nov., as Orientia tsutsugamushi comb. nov. *Int J Syst Bacteriol* 1995; **45**: 589–591.
31. Saah A. Orientia tsutsugamushi (scrub typhus). In: Mandell G, Bennett J, Dolin R, eds. Principles and practice of infectious disease, 5th ed. Philadelphia: Livingstone, C, 2000: 2056–2057.
32. Watt G, Parola P. Scrub typhus and tropical rickettsioses. *Curr Opin Infect Dis* 2003; **16**: 429–436.
33. Joklik W, Willet H, Amos D, Wilford C. Zinsser microbiology, 20th ed. Norwalk, CT: Appleton & Lange, 1992.
34. Berman SJ, Kundin WD. Scrub typhus in South Vietnam. A study of 87 cases. *Ann Intern Med* 1973; **79**: 26–30.
35. Settle E, Pinkerton H, Corbett A. A pathologic study of Tsutsugamushi disease (scrub typhus) with notes on clinicopathologic correlation. *J Lab Clin Med* 1945; **30**: 639–661.
36. Brown GW, Shirai A, Rogers C, Groves MG. Diagnostic criteria for scrub typhus: probability values for immunofluorescent antibody and Proteus OXK agglutinin titers. *Am J Trop Med Hyg* 1983; **32**: 1101–1107.
37. Brown GW, Robinson DM, Huxsoll DL. Serological evidence for a high incidence of transmission of Rickettsia tsutsugamushi in two Orang Asli settlements in Peninsular Malaysia. *Am J Trop Med Hyg* 1978; **27**: 121–123.
38. Johnson DE, Crum JW, Hanchalay S, Saengruchi C. Seroepidemiological survey of Rickettsia tsutsugamushi infection in a rural Thai village. *Trans R Soc Trop Med Hyg* 1982; **76**: 1–3.
39. Kawamura A Jr, Murata M, Osono M, et al. Studies on inapparent infection of tsutsugamushi disease in Izu Shichito Islands: seroepidemiology and demonstration of an avirulent Rickettsia strain for mice. *Jpn J Exp Med* 1980; **50**: 91–105.
40. Panpanich R, Garner P. Antibiotics for treating scrub typhus. *Cochrane Database Syst Rev* 2000; CD002150.
41. Song JH, Lee C, Chang WH, et al. Short-course doxycycline treatment versus conventional tetracycline therapy for scrub typhus: a multicenter randomized trial. *Clin Infect Dis* 1995; **21**: 506–510.
42. Kim YS, Yun HJ, Shim SK, Koo SH, Kim SY, Kim S. A comparative trial of a single dose of azithromycin versus doxycycline for the treatment of mild scrub typhus. *Clin Infect Dis* 2004; **39**: 1329–1335.
43. Kim YS, Lee HJ, Chang M, Son SK, Rhee YE, Shim SK. Scrub typhus during pregnancy and its treatment: a case series and review of the literature. *Am J Trop Med Hyg* 2006; **75**: 955–959.
44. Strickman D, Sheer T, Salata K, et al. In vitro effectiveness of azithromycin against doxycycline-resistant and -susceptible strains of Rickettsia tsutsugamushi, etiologic agent of scrub typhus. *Antimicrob Agents Chemother* 1995; **39**: 2406–2410.
45. Watt G, Kantipong P, Jongsakul K, Watcharapichat P, Phulsuksombati D, Strickman D. Doxycycline and rifampicin for mild scrub-typhus infections in northern Thailand: a randomised trial. *Lancet* 2000; **356**: 1057–1061.
46. Sheehy TW, Hazlett D, Turk RE. Scrub typhus. A comparison of chloramphenicol and tetracycline in its treatment. *Arch Intern Med* 1973; **132**: 77–80.
47. Andrew R, Bonin JM, Williams S. Tick typhus in North Queensland. *Med J Aust* 1946; **2**: 253.
48. Naltsas S, Hodge SJ, Gataky GJ Jr, Owen LG. Eczematous dermatitis caused by Dermanyssus americanus. *Cutis* 1980; **25**: 429–431.
49. Braun M III. Avian mite dermatitis. *Cutis* 1979; **23**: 680–682.
50. Lucky AW, Sayers C, Argus JD, Lucky A. Avian mite bites acquired from a new source – pet gerbils: report of 2 cases and review of the literature. *Arch Dermatol* 2001; **137**: 167–170.

51. Crystal MM. Rapid evaluation of candidate materials preliminary to in vivo testing for systemic control of northern fowl mites, *Ornithonyssus sylviarum* (Acari: Macronyssidae). *J Med Entomol* 1987; **24**: 584–586.
52. Sulzberger MB, Kaminstein I. Avian itch mites as a cause of human dermatoses: canary birds' mites responsible for 2 groups of cases in New York. *Arch Dermatol Syphilol* 1936; **33**: 60.
53. Lutsky I, Bar-Sela S. Northern fowl mite (*Ornithonyssus sylviarum*) in occupational asthma of poultry workers. *Lancet* 1982; **2**: 874–875.
54. DeVane JA. A survey of poultry ectoparasite problems and their research in the United States. *Poultry Sci* 1978; **57**: 1217–1220.
55. Deoreo GA. Pigeons acting as vector in acariasis caused by *Dermanyssus gallinae* (DeGeer, 1778). *AMA Arch Derm* 1958; **77**: 422–429.
56. Auger P, Nantel J, Meunier N, Harrison RJ, Loisele R, Gyorkos TW. Skin acariasis caused by *Dermanyssus gallinae* (de Geer): an in-hospital outbreak. *Can Med Assoc J* 1979; **120**: 700–703.
57. Uesugi Y, Aiba S, Suetake T, Tagami H. Multiple infestations with avian mites within a family. *Int J Dermatol* 1994; **33**: 566–567.
58. Dietrich M, Horstmann RD. [Urticarial dermatitis in acariasis caused by *Dermanyssus hirundinis* – pseudoscabies]. *Med Welt* 1983; **34**: 595–597.
59. Orton DI, Warren LJ, Wilkinson JD. Avian mite dermatitis. *Clin Exp Dermatol* 2000; **25**: 129–131.
60. Bellanger AP, Bories C, Foulet F, Bretagne S, Botterel F. Nosocomial dermatitis caused by *Dermanyssus gallinae*. *Infect Control Hosp Epidemiol* 2008; **29**: 282–283.
61. Thind BB, Ford HL. Assessment of susceptibility of the poultry red mite *Dermanyssus gallinae* (Acari: Dermanyssidae) to some acaricides using an adapted filter paper based bioassay. *Vet Parasitol* 2007; **144**: 344–348.
62. Freeman E, Kataria MS. Poultry mite infestation. *Br Med J* 1969; **4**: 53.
63. Kong TK, To WK. Images in clinical medicine. Bird-mite infestation. *N Engl J Med* 2006; **354**: 1728.
64. Congly H. Northern fowl mite dermatitis. *Can Med Assoc J* 1985; **132**: 738.
65. Chamberlain RW, Sikes RK. Laboratory investigations on the role of bird mites in the transmission of eastern and western equine encephalitis. *Am J Trop Med Hyg* 1955; **4**: 106–118.
66. Durden LA, Linthicum KJ, Monath TP. Laboratory transmission of eastern equine encephalomyelitis virus to chickens by chicken mites (Acari: Dermanyssidae). *J Med Entomol* 1993; **30**: 281–285.
67. Garvin MC, Scheidler LC, Cantor DG, Bell KE. Abundance and temporal distribution of *Ornithonyssus sylviarum* Canestrini and Fanzago (Acarina: Mesostigmata) in gray catbird (*Dumetella carolinensis*) nests. *J Vector Ecol* 2004; **29**: 62–65.
68. Fox JG. Outbreak of tropical rat mite dermatitis in laboratory personnel. *Arch Dermatol* 1982; **118**: 676–678.
69. Hetherington GW, Holder WR, Smith EB. Rat mite dermatitis. *JAMA* 1971; **215**: 1499–1500.
70. Durden LA, Best TL, Wilson N, Hilton CD. Ectoparasitic mites (Acari) of sympatric Brazilian free-tailed bats and big brown bats in Alabama. *J Med Entomol* 1992; **29**: 507–511.
71. Spears RE, Durden LA, Hagan DV. Ectoparasites of Brazilian free-tailed bats with emphasis on anatomical site preferences for *Chiroptonyssus robustipes* (Acari: Macronyssidae). *J Med Entomol* 1999; **36**: 481–485.
72. Chung SL, Hwang SJ, Kwon SB, Kim DW, Jun JB, Cho BK. Outbreak of rat mite dermatitis in medical students. *Int J Dermatol* 1998; **37**: 591–594.
73. Engel PM, Welzel J, Maass M, Schramm U, Wolff HH. Tropical rat mite dermatitis: case report and review. *Clin Infect Dis* 1998; **27**: 1465–1469.
74. Theis J, Lavoipierre MM, LaPerriere R, Kroese H. Tropical rat mite dermatitis. Report of six cases and review of mite infestations. *Arch Dermatol* 1981; **117**: 341–343.
75. Schultz H. Human infestation by *Ophionyssus natricis* snake mite. *Br J Dermatol* 1975; **93**: 695–697.
76. Keh B. Dermatitis caused by the bat mite, *Chiroptonyssus robustipes* (Ewing), in California. *J Med Entomol* 1974; **11**: 498.
77. Creel NB, Crowe MA, Mullen GR. Pet hamsters as a source of rat mite dermatitis. *Cutis* 2003; **71**: 457–461.
78. Baumstark J, Beck W, Hofmann H. Outbreak of tropical rat mite (*Ornithonyssus bacoti*) dermatitis in a home for disabled persons. *Dermatology* 2007; **215**: 66–68.
79. Fairburn EA, Frain-Bell W. *Bdellonyssus bacoti* as a casual agent of cutaneous disease. *Br J Dermatol* 1956; **68**: 350–354.
80. Watson J. New building, old parasite: Mesostigmatid mites – an ever-present threat to barrier facilities. *Ilar J* 2008; **49**: 303–309.
81. Kelaher J, Jogi R, Katta R. An outbreak of rat mite dermatitis in an animal research facility. *Cutis* 2005; **75**: 282–286.
82. Schwab M, Allen R, Sulkin SE. The tropical rat mite (*Liponyssus bacoti*) as an experimental vector of Coxsackie virus. *Am J Trop Med Hyg* 1952; **1**: 982–986.
83. Ram SM, Satija KC, Kaushik RK. *Ornithonyssus bacoti* infestation in laboratory personnel and veterinary students. *Int J Zoonoses* 1986; **13**: 138–140.
84. Durden LA, Turell MJ. Inefficient mechanical transmission of Langat (tick-borne encephalitis virus complex) virus by blood-feeding mites (Acari) to laboratory mice. *J Med Entomol* 1993; **30**: 639–641.
85. Shelmire B, Dove W. The tropical rat mite *Liponyssus bacoti* Hirst, 1914: cause of a skin eruption of man, and a possible vector of endemic typhus fever. *JAMA* 1931; **96**: 579–584.
86. Andrews JR, Ramsay GW. A case of papular dermatosis in man attributed to an ascid mite (Acari). *J Med Entomol* 1982; **19**: 111–112.
87. Halliday RB, Walter DE, Lindquist EE. Revision of the Australian Ascidae (Acarina: Mesostigmata). *Invertebr Taxon* 1998; **12**: 1–54.
88. Letchford J, Strungs I, Farrell D. Pyemotes species strongly implicated in an outbreak of dermatitis in a Queensland country hospital. *Pathology* 1994; **26**: 330–332.
89. Rawles LT. Grain itch. *J Med Indiana Ass* 1909; **3**: 351.
90. Rycroft RJ, Kennedy C. Pyemotes dermatitis in display artists. *Clin Exp Dermatol* 1981; **6**: 629–634.
91. Hewitt M, Barrow GI, Miller DC, Turk SM. A case of Pyemotes dermatitis. With a note on the role of these mites in skin disease. *Br J Dermatol* 1976; **94**: 423–430.
92. Kunkle GA, Greiner EC. Dermatitis in horses and man caused by the straw itch mite. *J Am Vet Med Assoc* 1982; **181**: 467–469.
93. Betz TG, Davis BL, Fournier PV, Rawlings JA, Elliot LB, Baggett DA. Occupational dermatitis associated with straw itch mites (*Pyemotes ventricosus*). *JAMA* 1982; **247**: 2821–2823.

94. Booth BH, Jones RW. Epidemiological and clinical study of grain itch. *J Am Med Assoc* 1952; **150**: 1575–1579.
95. Fine RM, Scott HG. Straw itch mite dermatitis caused by *Pyemotes ventricosus*: comparative aspects. *South Med J* 1965; **58**: 416–420.
96. Samsinak K, Chmela J, Vobrazkova E. *Pyemotes herfsi* (Oudemans, 1936) as causative agent of another mass dermatitis in Europe (Acari, Pyemotidae). *Folia Parasitol (Praha)* 1979; **26**: 51–54.
97. Outbreak of pruritic rashes associated with mites – Kansas, 2004. *MMWR Morb Mortal Wkly Rep* 2005; **54**: 952–955.
98. Broce AB, Zurek L, Kalisch JA, et al. *Pyemotes herfsi* (Acari: Pyemotidae), a mite new to North America as the cause of bite outbreaks. *J Med Entomol* 2006; **43**: 610–613.
99. Vissian L, Barety M, Motte P, Le Fichoux Y. [Epidemic aspect of parasite pruriginous dermatitis of *Pyemotes zwoelferi* type (author's transl)]. *Ann Dermatol Venereol* 1980; **107**: 21–24.
100. Le Fichoux Y, Rack G, Motte P, Dellamonica P, Marty P. Pruriginous dermatitis due to *Pyemotes zwoelferi* Krczal, 1963. About several cases in the Alpes-Maritimes (France). *Acta Trop* 1980; **37**: 83–89.
101. Tomalski MD, Kutney R, Bruce WA, Brown MR, Blum MS, Travis J. Purification and characterization of insect toxins derived from the mite, *Pyemotes tritici*. *Toxicon* 1989; **27**: 1151–1167.
102. Tomalski MD, Bruce WA, Travis J, Blum MS. Preliminary characterization of toxins from the straw itch mite, *Pyemotes tritici*, which induce paralysis in the larvae of a moth. *Toxicon* 1988; **26**: 127–132.
103. Tomalski MD, Miller LK. Insect paralysis by baculovirus-mediated expression of a mite neurotoxin gene. *Nature* 1991; **352**: 82–85.
104. Uenotsuchi T, Satoh E, Kiryu H, Yano Y. *Pyemotes* dermatitis caused by indirect contact with husk rice. *Br J Dermatol* 2000; **143**: 680–682.
105. Liguori G, Ceccarelli MT, Mellino M, Marinelli P. [Recent experience with mites in stored products]. *Ann Ig* 1989; **1**: 237–246.
106. Swan DC. The hay itch mite *Pediculoides ventricosus* (Newport) (Acarina, Pediculoididae) in South Australia. *J Agric Ind S Aust* 1934; **37**: 1289–1299.
107. Hanks LM, McCelfresh JS, Millar JG, Paine TD. Control of the straw itch mite (Acari: Pyemotidae) with sulfur in an insect rearing facility. *J Econ Entomol* 1992; **85**: 683–686.
108. Newell IM. Feeding habits in the genus *Balaustium* (Acarina, Erythraeidae), with special reference to attacks on man. *J Parasitol* 1963; **49**: 498–502.
109. Ido T, Kumakiri M, Lao LM, Yano Y, Takada N. Dermatitis caused by *Balaustium murorum*. *Acta Derm Venereol* 2004; **84**: 80–81.
110. Baima B, Sticherling M. Demodicidosis revisited. *Acta Derm Venereol* 2002; **82**: 3–6.
111. Purcell SM, Hayes TJ, Dixon SL. Pustular folliculitis associated with *Demodex folliculorum*. *J Am Acad Dermatol* 1986; **15**: 1159–1162.
112. Karıncaoglu Y, Tepe B, Kalayci B, Seyhan M. Pseudozoster clinical presentation of *Demodex* infestation after prolonged topical steroid use. *Clin Exp Dermatol* 2008; **33**: 740–742.
113. Karıncaoglu Y, Bayram N, Aycan O, Esrefoglu M. The clinical importance of *demodex folliculorum* presenting with nonspecific facial signs and symptoms. *J Dermatol* 2004; **31**: 618–626.
114. Ecker RI, Winkelmann RK. *Demodex granuloma*. *Arch Dermatol* 1979; **115**: 343–344.
115. Farina MC, Requena L, Sarasa JL, et al. Spinulosis of the face as a manifestation of demodicidosis. *Br J Dermatol* 1998; **138**: 901–903.
116. De Dulanto F, Camacho-Martinez F. [Demodicidosis gravis]. *Ann Dermatol Venereol* 1979; **106**: 699–704.
117. English FP, Nutting WB. Demodicosis of ophthalmic concern. *Am J Ophthalmol* 1981; **91**: 362–372.
118. Gao YY, Di Pascuale MA, Li W, et al. High prevalence of *Demodex* in eyelashes with cylindrical dandruff. *Invest Ophthalmol Vis Sci* 2005; **46**: 3089–3094.
119. Heacock CE. Clinical manifestations of demodicosis. *J Am Optom Assoc* 1986; **57**: 914–919.
120. Gao YY, Di Pascuale MA, Elizondo A, Tseng SC. Clinical treatment of ocular demodicosis by lid scrub with tea tree oil. *Cornea* 2007; **26**: 136–143.
121. Garcia-Vargas A, Mayorga-Rodriguez JA, Sandoval-Tress C. Scalp demodicidosis mimicking favus in a 6-year-old boy. *J Am Acad Dermatol* 2007; **57**: S19–21.
122. Damian D, Rogers M. *Demodex* infestation in a child with leukaemia: treatment with ivermectin and permethrin. *Int J Dermatol* 2003; **42**: 724–726.
123. Castanet J, Monpoux F, Mariani R, Ortonne JP, Lacour JP. Demodicidosis in an immunodeficient child. *Pediatr Dermatol* 1997; **14**: 219–220.
124. Sahn EE, Sheridan DM. Demodicidosis in a child with leukemia. *J Am Acad Dermatol* 1992; **27**: 799–801.
125. Herron MD, O'Reilly MA, Vanderhooft SL. Refractory *Demodex* folliculitis in five children with acute lymphoblastic leukemia. *Pediatr Dermatol* 2005; **22**: 407–411.
126. Seyhan ME, Karıncaoglu Y, Bayram N, Aycan O, Kuku I. Density of *Demodex folliculorum* in haematological malignancies. *J Int Med Res* 2004; **32**: 411–415.
127. Ayres S Jr. Rosacea and rosacea-like demodicidosis. *Int J Dermatol* 1987; **26**: 198–199.
128. Forton F, Seys B, Marchal JL, Song AM. *Demodex folliculorum* and topical treatment: acaricidal action evaluated by standardized skin surface biopsy. *Br J Dermatol* 1998; **138**: 461–466.
129. Hoekzema R, Hulsebosch HJ, Bos JD. Demodicidosis or rosacea: what did we treat? *Br J Dermatol* 1995; **133**: 294–299.
130. Nakagawa T, Sasaki M, Fujita K, Nishimoto M, Takaiwa T. *Demodex* folliculitis on the trunk of a patient with mycosis fungoides. *Clin Exp Dermatol* 1996; **21**: 148–150.
131. Morsy TA, el Okbi MM, el-Said AM, Arafa MA, Sabry AH. *Demodex* (follicular mite) infesting a boy and his pet dog. *J Egypt Soc Parasitol* 1995; **25**: 509–512.
132. Elston DM. *Demodex* mites as a cause of human disease. *Cutis* 2005; **76**: 294–296.
133. Elston DM, Lawler KB, Iddins BO. What's eating you? *Demodex folliculorum*. *Cutis* 2001; **68**: 93–94.
134. Sanfilippo AM, English JC III. Resistant scalp folliculitis secondary to *Demodex* infestation. *Cutis* 2005; **76**: 321–324.
135. Schaller M, Sander CA, Plewig G. *Demodex* abscesses: clinical and therapeutic challenges. *J Am Acad Dermatol* 2003; **49**: S272–274.
136. El-Shazly AM, Hassan AA, Soliman M, Morsy GH, Morsy TA. Treatment of human *Demodex folliculorum* by camphor oil and metronidazole. *J Egypt Soc Parasitol* 2004; **34**: 107–116.
137. Clyti E, Nacher M, Sainte-Marie D, Pradinaud R, Couppie P. Ivermectin treatment of three cases of demodicidosis

- during human immunodeficiency virus infection. *Int J Dermatol* 2006; **45**: 1066–1068.
138. Castellani A. Note on copra itch. *J Trop Med Hyg* 1912; **15**: 374.
 139. Armentia A, Fernandez A, Perez-Santos C, et al. Occupational allergy to mites in salty ham, chorizo and cheese. *Allergol Immunopathol (Madr)* 1994; **22**: 152–154.
 140. Estevez MD. Occupational contact urticaria-dermatitis by *Tyrophagus putrescentiae*. *Contact Dermatitis* 2006; **55**: 308–309.
 141. Prosser Thomas EW. Dermatitis due to *Tyroglyphus longior*, gerv., var. *castellanii*, Hirst, in cheese dust. *Br J Dermatol Syphilis* 1942; **52**: 313–319.
 142. Revsbech P, Andersen G. Storage mite allergy among grain elevator workers. *Allergy* 1987; **42**: 423–429.
 143. Revsbech P, Dueholm M. Storage mite allergy among bakers. *Allergy* 1990; **45**: 204–208.
 144. Terho EO, Husman K, Vohlonen I, Rautalahti M, Tukiainen H. Allergy to storage mites or cow dander as a cause of rhinitis among Finnish dairy farmers. *Allergy* 1985; **40**: 23–26.
 145. Vidal C, Rial A. Airborne contact dermatitis from *Tyrophagus putrescentiae*. *Contact Dermatitis* 1998; **38**: 181.
 146. Athanassiou CG, Kavallieratos NG, Palyvos NE, Sciarretta A, Trematerra P. Spatiotemporal distribution of insects and mites in horizontally stored wheat. *J Econ Entomol* 2005; **98**: 1058–1069.
 147. Mullen GR, Durden LA. Medical and veterinary entomology, 1st ed. San Diego, CA: Elsevier Science & Technology, 2002.
 148. van Hage-Hamsten M, Johansson E. Clinical and immunologic aspects of storage mite allergy. *Allergy* 1998; **53**: 49–53.
 149. Griffiths DA. Experimental studies on the systematics of the genus *Acarus* Linnaeus, 1758 Sarcoptiformes, Acarina. *Acarologia* 1964; **6**: 101–116.
 150. Griffiths DA. A revision of the genus *Acarus* (Acaridae, Acarina). *Bull Br Mus Nat Hist* 1964; **11**: 413–464.
 151. Eales NB. The life history and economy of the cheese mites. *Ann Appl Biol* 1917; **4**: 28–35.
 152. Robertson PL. Cheese mite infestation: an important storage problem. *Int J Dairy Technol* 1952; **5**: 86–95.
 153. Oppenheim M, Taglicht H. Two cases of *Acarus* disease in date packers. *Wiener Medizinische Wochenschrift* 1925; **75**: 363–367.
 154. O'Donovan WJ. Dermatitis due to *Carpoglyphus passulorum*. *Br J Dermatol* 1920; **32**: 297–298.
 155. Rollier R. [Vesicular Dermatoses Due to *Glyciphagus Domesticus*]. *Bull Soc Fr Dermatol Syphiligr* 1963; **70**: 676–677.
 156. Pigatto PD, Bigardi A, Riboldi A, Legori A, Altomare GF, Ottoboni F. Occupational atopic contact dermatitis from *Rhizoglyphus*. *Contact Dermatitis* 1991; **25**: 193–194.
 157. Collins DA. A review of alternatives to organophosphorus compounds for the control of storage mites. *J Stored Prod Res* 2006; **42**: 395–426.
 158. Golob P. Current status and future perspectives for inert dusts for control of stored product insects. *J Stored Prod Res* 1997; **33**: 69–79.
 159. Wilkin DR. Resistance to lindane in *Acarus siro* from an English cheese store. *J Stored Prod Res* 1973; **9**: 101–104.
 160. Arlian LG, Bernstein D, Bernstein IL, et al. Prevalence of dust mites in the homes of people with asthma living in eight different geographic areas of the United States. *J Allergy Clin Immunol* 1992; **90**: 292–300.
 161. Arlian LG, Platts-Mills TA. The biology of dust mites and the remediation of mite allergens in allergic disease. *J Allergy Clin Immunol* 2001; **107**: S406–413.
 162. Aylesworth R, Baldrige D. Feather pillow dermatitis caused by an unusual mite, *Dermatophagoides schermenetskyi*. *J Am Acad Dermatol* 1985; **13**: 680–681.
 163. Aylesworth R, Baldrige D. *Dermatophagoides schermenetskyi* and feather pillow dermatitis. *Minn Med* 1983; **66**: 43.
 164. Adinoff AD, Tellez P, Clark RA. Atopic dermatitis and aeroallergen contact sensitivity. *J Allergy Clin Immunol* 1988; **81**: 736–742.
 165. Ricci G, Patrizi A, Specchia F, et al. Effect of house dust mite avoidance measures in children with atopic dermatitis. *Br J Dermatol* 2000; **143**: 379–384.
 166. Sanchez-Borges M, Capriles-Hulett A, Caballero-Fonseca F. Oral mite anaphylaxis (pancake syndrome) also observed in children. *Ann Allergy Asthma Immunol* 2006; **96**: 755–756.
 167. Hannaway PJ, Miller JD. The pancake syndrome (oral mite anaphylaxis) by ingestion and inhalation in a 52-year-old woman in the northeastern United States. *Ann Allergy Asthma Immunol* 2008; **100**: 397–398.
 168. Matsumoto T, Hisano T, Hamaguchi M, Miike T. Systemic anaphylaxis after eating storage-mite-contaminated food. *Int Arch Allergy Immunol* 1996; **109**: 197–200.
 169. Sanchez-Borges M, Suarez-Chacon R, Capriles-Hulett A, Caballero-Fonseca F. An update on oral anaphylaxis from mite ingestion. *Ann Allergy Asthma Immunol* 2005; **94**: 216–220, quiz 20–22, 306.
 170. Wen DC, Shyur SD, Ho CM, et al. Systemic anaphylaxis after the ingestion of pancake contaminated with the storage mite *Blomia freemani*. *Ann Allergy Asthma Immunol* 2005; **95**: 612–614.
 171. Erben AM, Rodriguez JL, McCullough J, Ownby DR. Anaphylaxis after ingestion of beignets contaminated with *Dermatophagoides farinae*. *J Allergy Clin Immunol* 1993; **92**: 846–849.
 172. Sanchez-Borges M, Capriles-Hulett A, Fernandez-Caldas E, et al. Mite-contaminated foods as a cause of anaphylaxis. *J Allergy Clin Immunol* 1997; **99**: 738–743.
 173. Zahler M, Essig A, Gothe R, Rinder H. Genetic evidence suggests that *Psoroptes* isolates of different phenotypes, hosts and geographic origins are conspecific. *Int J Parasitol* 1998; **28**: 1713–1719.
 174. Wall R, Kolbe K. Taxonomic priority in *Psoroptes* mange mites: *P. ovis* or *P. equi*? *Exp Appl Acarol* 2006; **39**: 159–162.
 175. Pegler KR, Evans L, Stevens JR, Wall R. Morphological and molecular comparison of host-derived populations of parasitic *Psoroptes* mites. *Med Vet Entomol* 2005; **19**: 392–403.
 176. Elston DM. What's eating you? *Psoroptes* mites. *Cutis* 2006; **77**: 283–284.
 177. Mazyad SA, Sanad EM, Morsy TA. Two types of scab mites infesting man and sheep in North Sinai. *J Egypt Soc Parasitol* 2001; **31**: 213–222.
 178. Forbes AB, Pitt SR, Baggott DG, et al. A review of the use of a controlled-release formulation of ivermectin in the treatment and prophylaxis of *Psoroptes ovis* infestations in sheep. *Vet Parasitol* 1999; **83**: 319–326.
 179. Kurtdebe A, Karaer Z, Acar A, et al. Use of selamectin for the treatment of psoroptic and sarcoptic mite infestation in rabbits. *Vet Dermatol* 2007; **18**: 18–22.

180. Andrews JR. The origin and evolution of host associations of *Sarcoptes scabiei* and the subfamily Sarcoptinae Murray. *Acarologia* 1983; **24**: 85–94.
181. Burroughs RF, Elston DM. What's eating you? Canine scabies. *Cutis* 2003; **72**: 107–109.
182. Norins AL. Canine scabies in children. "Puppy dog" dermatitis. *Am J Dis Child* 1969; **117**: 239–342.
183. Smith EB, Claypoole TF. Canine scabies in dogs and in humans. *JAMA* 1967; **199**: 59–64.
184. Arlian LG, Runyan RA, Achar S, Estes SA. Survival and infectivity of *Sarcoptes scabiei* var. *canis* and var. *hominis*. *J Am Acad Dermatol* 1984; **11**: 210–215.
185. Argenziano G, Fabbrocini G, Delfino M. Epiluminescence microscopy. A new approach to in vivo detection of *Sarcoptes scabiei*. *Arch Dermatol* 1997; **133**: 751–753.
186. Kristjansson AK, Smith MK, Gould JW, Gilliam AC. Pink pigtails are a clue for the diagnosis of scabies. *J Am Acad Dermatol* 2007; **57**: 174–175.