FIG. 292.1 Dorsal view of a female body louse, *Pediculus humanus* var. *corporis.* *(From Public Health Image Library. Image 9204. Atlanta, GA: Centers for Disease Control and Prevention.)*

FIG. 292.3 Enlarged ventral image of a pubic or crab louse, *Phthirus pubis.* *(From Public Health Image Library. Image 4077. Atlanta, GA: Centers for Disease Control and Prevention.)*

FIG. 292.2 Dorsal view of a female head louse, *Pediculus humanus* var. *capitis,* containing eggs or nits. *(From Public Health Image Library. Image 377. Atlanta, GA: Centers for Disease Control and Prevention.)*

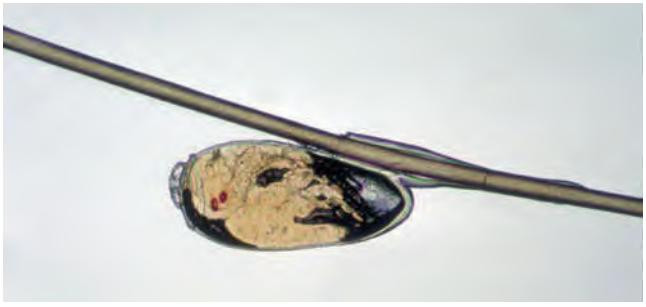


FIG. 292.4 The unhatched nit or egg of the head louse, *Pediculus humanus* var. *capitis,* attached to a hair shaft. Note the red eye spots of the developing nymph embryo. *(From Public Health Image Library. Image 378. Atlanta, GA: Centers for Disease Control and Prevention.)*

B. quintana has now been isolated in head lice as well as body lice from homeless persons in the United States, establishing the potential for transmission of trench fever by blood-feeding head lice in addition to body lice.[6] During a 2011 outbreak of relapsing fever in Ethiopia, investigators demonstrated *B. recurrentis* DNA by quantitative real-time PCR in 23% of head lice from patients with positive blood smears, establishing the potential for transmission of relapsing fever by head lice in addition to body lice.[7]

In addition to serving as vectors for epidemic typhus (*R. prowazekii*), trench fever (*B. quintana*), and relapsing fever (*B. recurrentis*), body lice have been suspected in the transmission of a fourth pathogen, *Yersinia pestis,* the causative agent of plague. Collectively, these pathogens have killed millions of persons, especially homeless people and displaced war refugees without access to proper sanitation, personal hygiene, and clean clothing. In 2010, Drali and coinvestigators collected body and head lice from 37 infected individuals in plague-hyperendemic areas of the Democratic Republic of the Congo. Using multiplex, real-time PCR to rapidly differentiate head and body lice, the investigators detected evidence of *B. quintana* in 33.5% of body lice and 19% of head lice and *Y. pestis* in one head louse PCR-negative for other pathogens and two body lice PCR-positive for *B. quintana*.[2] Although these results will require independent confirmation in larger numbers of study subject samples, plague should probably be added to the list of dangerous pathogens that potentially can be transmitted by body lice and head lice.

DIAGNOSIS

Lice infestations are diagnosed by demonstrating live adult lice, nymphs, and viable eggs, or nits, in their precise human ecologic niches. Adult lice are flattened dorsoventrally and are 1 mm (pubic lice) to 3 mm (head and body lice) in length, have three pairs of legs ending in powerful claws that can grip hair shafts, and exhibit a reddish-brown hue after blood-feeding (see Figs. 292.2 and 292.3). Females can live on their hosts for up to 3 months, lay up to 300 nits in a lifetime (see Fig. 292.2), and die within 24 hours when separated from hosts. Nits are oval, less than 1 mm in diameter, and grayish white (Fig. 292.4); they fluoresce in ultraviolet or Wood light when viable. Nits are deposited on hair shafts at the skin surface and hatch nymphs within 6 to 10 days (see Fig. 292.4). Nymphs resemble miniature adults and grow to adulthood within 10 days. Empty egg cases remain attached to hair shafts after hatching and are not diagnostic of active infection.[8] Head lice and their viable nits are often attached to hairs close to the scalp, especially in occipital and postauricular locations. Because body lice visit their human hosts only to feed on blood, adults, nymphs, and nits are found in clothing, usually aligned along inner seams (Fig. 292.5). Pubic lice and their nits may be found in the pubic, perianal, and inguinal areas; in axillary and chest hair; and even in the eyelashes (phthiriasis palpebrum), especially in children who acquire pubic lice infestations from their parents (Fig. 292.6).[9]

CLINICAL MANIFESTATIONS AND DIFFERENTIAL DIAGNOSIS

Head Lice

The clinical manifestations of pediculosis capitis range from asymptomatic infestations to severe pruritus, with self-inflicted, often secondarily infected, excoriations with impetigo and postoccipital lymphadenopathy. The differential diagnosis of pediculosis capitis includes eczema, lichen

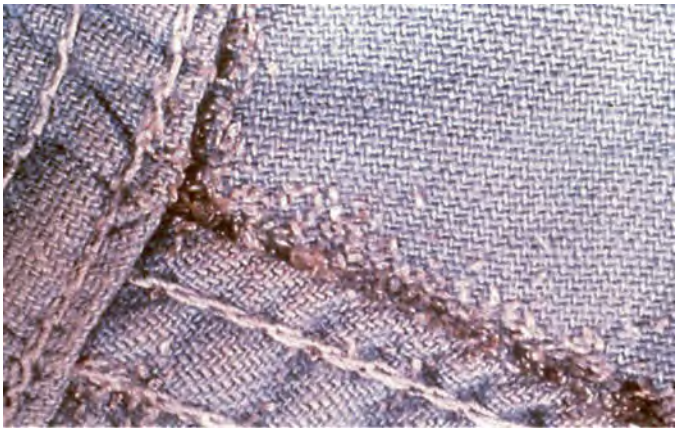


FIG. 292.5 Live eggs from the body louse, *Pediculus humanus* var. *corporis,* lining the seams of clothing. *(From Public Health Image Library. Image 5270. Atlanta, GA: Centers for Disease Control and Prevention.)*

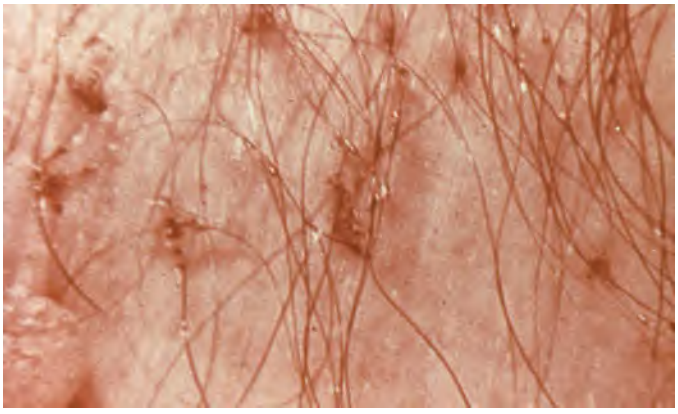


FIG. 292.6 Erythematous lesions seen in the pubic region of a patient in response to the bites of the blood-feeding crab or pubic louse, *Phthirus pubis.* *(From Public Health Image Library. Image 4078. Atlanta, GA: Centers for Disease Control and Prevention.)*

simplex chronicus, dandruff, seborrheic dermatitis, and bacterial impetigo.

Body Lice

Despite the fact that body lice reside in clothing, often along seams and not on the body, like head lice infestation, pediculosis corporis is often much more symptomatic than pediculosis capitis and causes severe pruritus, with extensive self-inflicted excoriations (see Fig. 292.5). The sites of blood-feeding are often present as erythematous macules, papules, or areas of papular urticaria with a central hemorrhagic punctum. The differential diagnosis includes eczematous dermatitis, lichen simplex chronicus, and scabies.

Crab Lice (Pediculosis Pubis)

Infestation with crab lice, or pediculosis pubis (phthiriasis), is less symptomatic compared with pediculosis capitis. These organisms affect all hair-bearing regions, most commonly the pubic and perianal areas, but also the upper eyelashes and the hairy areas of the axillae, chest, and abdomen. More extensive infestations usually occur in males with more body hair–bearing regions than females. Pubic lice may appear as 1- to 2-mm brownish-gray specks in infested hairy areas, where they remain stationary for days with claws grasping hair shafts and mouth parts embedded in the skin (see Fig. 292.3). The average life span for *Phthirus pubis* is 17 days for females and 22 days for males. Females deposit grayish-white eggs or nits at the skin-hair junctions. The egg incubation period is 7 to 8 days, and the life cycle from egg to adult is 22 to 27 days. Clinical manifestations include papular urticaria and self-inflicted, often infected, excoriations at blood-feeding sites, as well as regional, usually inguinal, lymphadenopathy (see Fig. 292.6). Pathognomonic findings may include maculae ceruleae *(taches bleues),* bluish-gray irregularly shaped macules, 0.5 to 1 cm in diameter, scattered over the lower abdominal wall, buttocks, and upper thighs. Maculae ceruleae may be caused by subcutaneous tissue staining from heme pigments altered by louse saliva and digestion. The differential diagnosis of crab lice includes eczematous dermatitis, seborrheic dermatitis, tinea cruris, folliculitis, molluscum contagiosum, and scabies, which frequently coexists with phthiriasis. Management includes initial bathing with soap and water, followed by two topical or systemic treatments with pediculicides, 7 to 10 days apart (Table 292.1).

THERAPY

Both *Pediculus humanus* species (head and body lice) and *Phthirus pubis* (the crab or pubic louse) have now demonstrated high levels of resistance worldwide to the safest topical pediculicides, specifically the natural pyrethrins and synthetic pyrethroids (permethrin, phenothrin).[8,10–14] In addition, resistance to lindane, an organochlorine insecticide, and malathion, an organophosphate insecticide, both alone and combined with pyrethroids, has been reported in the United Kingdom and elsewhere.[8,10] In a randomized comparison of wet combing versus 0.5% malathion shampoos for head lice in the United Kingdom, Roberts and colleagues[10] reported a 78% cure rate for malathion shampoo versus 38% for wet combing. In an in vitro pediculicidal efficacy comparison of five pediculicides available in the United States, Meinking and associates[11] reported the following results:

1. There were significant differences in the pediculicidal efficacies of the five pesticides tested.
2. Malathion was the only tested pesticide in their study that had not become less effective as a pediculicide.
3. The ranked order of therapeutic effectiveness from most to least effective was 0.5% malathion, undiluted natural pyrethrins with piperonyl butoxide, 1% permethrin, diluted natural pyrethrins with piperonyl butoxide, and 1% lindane.
4. Some head lice in the United States had become resistant to most pediculicides.

The increasing resistance of head lice to the pyrethrins and pyrethroids has led to the increasing use of more toxic pesticides, specifically lindane, malathion, and carbaryl (not approved by the US Food and Drug Administration [FDA] in the United States) in treating pyrethroid-resistant pediculosis capitis worldwide.[10,13,14] Lindane is being inappropriately overprescribed, especially for recurrent infestations with lindane-resistant head lice.[8,15] Lindane is an organochlorine insecticide that bioaccumulates in adipose and nerve tissue with overapplication or, if ingested, can cause seizures, especially in children.[8,11] Although malathion, an organophosphate pesticide, has demonstrated the greatest therapeutic efficacy against head lice in the United States, it is an irreversible acetylcholinesterase inhibitor that can cause a cholinergic toxidrome and fatal neuromuscular paralysis after overapplication or ingestion.[13] Carbaryl, a carbamate pesticide highly effective against both head lice and scabies, is being increasingly prescribed for pediculosis capitis outside the United States, especially in the United Kingdom and Europe.[14] Carbaryl is a reversible (nonaging) acetylcholinesterase inhibitor that is closely related to the organophosphate pesticides and can also cause a cholinergic (muscarinic and nicotinic) toxidrome after overapplication or ingestion.

Unfortunately, all the topical pesticides used to treat ectoparasitic infections share the same three characteristics as the three most commonly ingested childhood poisons: (1) prescribed, or often over-the-counter, medications; (2) household products; and (3) pesticides.[14] As the prevalence of ectoparasitic infections with pesticide-resistant ectoparasites increases, alternative pesticides, more toxic than pyrethrins and pyrethroids, will be prescribed for ectoparasitic infestations. In addition, medications will continue to be administered in households, and household accidental overapplication or ingestion of more toxic pesticide formulations for pediculosis may increase without enhanced public health education measures.[14,16]

TABLE 292.1 Recommended Pediculicide Treatments for Pediculosis Capitis

PEDICULICIDE	TRADE NAMES	THERAPEUTIC EFFICACY (OVICIDAL, PEDICULICIDAL)	SAFETY PROFILE	CONTRAINDICATIONS
0.33% Pyrethrins + 4% piperonyl butoxide shampoo	A-200 (OTC) RID (OTC)	95% ovicidal; no residual activity; increasing drug resistance	Excellent	Chrysanthemum and daisy (plant family Compositae) allergies possible contraindications
1%–5% Permethrin cream rinse	Acticin (OTC, Rx) Nix (OTC)	2-wk residual activity; increasing drug resistance	Excellent	Prior allergic reactions
0.5% Malathion lotion, 1% malathion shampoo	Ovide (Rx)	95% ovicidal; rapid (5-min) killing; good residual activity; increasing drug resistance, but not in the United States	Flammable 78% isopropyl alcohol vehicle stings eyes, skin, mucosa; increasing drug resistance; organophosphate poisoning risks with overapplication and ingestion	Infants and children <6 mo of age; pregnancy; breastfeeding
1% Lindane lotion and shampoo	Generic (Rx)	95% ovicidal; no residual activity; increasing drug resistance	Potential for CNS toxicity from organochlorine poisoning, usually manifesting as seizures, with overapplication and ingestion	Preexisting seizure disorder; infants and children <6 mo of age; pregnancy; breastfeeding; not recommended for use due to toxicity
0.9% Spinosad suspension	Natroba (Rx)	New to market; no reports of resistance; not ovicidal	Excellent	Infants and children age 4 yr and younger; presumed safe in pregnancy based on animal studies
5% Benzyl alcohol lotion	Ulesfia (Rx)	No resistance reported; not ovicidal	Excellent	Infants and children age 6 mo and younger; presumed safe in pregnancy based on animal studies
0.5% Ivermectin lotion	Sklice (Rx)	No resistance; single 10-min application; not ovicidal but nymphs die when they emerge from nits	Excellent	Infants and children age 6 mo and younger; safety in pregnancy uncertain
Ivermectin, 200–400 µg/kg tablet	Stromectol (Rx)	Excellent; not ovicidal; single PO dose, second dose in 7–10 days recommended	Excellent, but not in widespread use; nausea and vomiting possible; take on empty stomach with water only	Safety in pregnancy uncertain; not recommended for children weighing <5 kg; not FDA approved for pediculosis in United States

[a]Carbaryl (Sevin), a carbamate pesticide, is not currently approved or available as a human topical preparation for use for pediculosis in the United States. Carbaryl is, however, prescribed for pediculosis in Europe and elsewhere. Ectoparasite resistance to carbaryl has not been reported.
CNS, Central nervous system; *FDA,* US Food and Drug Administration; *OTC,* over-the-counter availability; *Rx,* available by prescription only.

A descriptive meta-analysis of pesticide poisonings in children in the United States over the period 1966–2008 demonstrated that malathion pediculicide ingestions were increasing, possibly as a result of increasing pyrethroid-resistant head lice infestations.[14] Subsequently, a state health department analysis of pesticide exposures in children younger than 7 years of age over the period 2003–2007 reported that lice shampoo exposures by ingestions, intraocular instillations, and prolonged topical applications were second only to mosquito-repellent exposures and resulted in more medical visits than all other pesticide exposures.[16]

Therapy for Pediculosis Capitis

Management of pediculosis capitis includes two topical or systemic treatments with pediculicides, 7 to 10 days apart, and removal of all viable nits by carefully combing wet hair. Olive oil, petroleum jelly, and HairClean 1-2-3 are preferred hair-wetting agents, and plastic combs are preferred over metal combs. Unfortunately, the ideal pediculicide with 100% killing activity against lice and nits does not exist. Table 292.1 presents the most commonly used pediculicides for lice infestations. As noted, drug resistance is increasing against the safest pediculicides, the pyrethrins and synthetic pyrethroids, and even against lindane and malathion, an effective ovicidal insecticide with 95% efficacy against viable nits.[8,10,11]

A randomized controlled trial has now demonstrated that a single oral dose of ivermectin, 400 µg/kg of body weight, repeated at 7 days, established higher louse-free rates by day 15 than two applications of 0.5% malathion lotion in patients with pyrethroid-resistant head lice infestations.[17] In 2012, the FDA approved the use of topical 0.5% ivermectin lotion for head lice infestations after two multisite, randomized, double-blind studies comparing single applications of 0.5% ivermectin lotion with vehicle control that demonstrated significantly greater louse-free days at 1, 7, and 14 days in the ivermectin group than in the vehicle control group.[18] Today, both oral ivermectin (although not FDA approved) and topical ivermectin lotion for head lice offer convenient, single-dose treatments that kill nymphs when they emerge from nits and can be reserved for drug-resistant head lice cases to limit the potential for ivermectin resistance.

Therapy for Body Lice

Management includes initial bathing with soap and water, followed by two topical or systemic treatments with pediculicides, 7 to 10 days apart (see Table 292.1). Topical medications should be applied to clean affected areas, allowed to dry, and not rinsed for 8 (malathion) to 24 (pyrethrins, pyrethroids) hours.

PREVENTION

Prevention strategies for head lice include combinations of sanitizing the environment and, more important, eliminating all human reservoirs of carriage of head lice in households, apartments, housing complexes, homeless shelters, classrooms, and schools. Some common preventive interventions include the following: (1) avoiding contact with potentially contaminated items, such as hats, head sets, clothing, towels, combs, brushes, bedding, and upholstery; (2) soaking all combs and brushes in isopropyl alcohol or 2% Lysol solution; (3) sanitizing the household environment by hot-cycle washing and drying of all bedding, clothing, and headgear; and (4) inspecting high-risk schoolchildren for active head lice, viable nits, and nymphs. Lebwohl and colleagues[8] have recommended that "no-nit policies" in schools be abandoned. As noted, nonviable nits on hair shafts are simply empty egg cases and do not indicate active louse infestation. Dermoscopy can now clearly distinguish viable nits from hatched, empty nits and pseudonits immediately and reliably and offers a more sensitive screening tool for head lice infestations than inspection alone.[19]

Prevention and control strategies for pediculosis corporis should include the following:

1. Hot-cycle washing and drying of all clothing and bedding
2. Clothing and body delousing with 1% permethrin dusting powder, especially in outbreak situations with potential for bacterial disease transmission

3. Institution of basic personal hygiene and sanitation measures, including showering, body washing, and clean clothing changes

Prevention strategies for pubic lice are similar to the prevention strategies for body lice and should include the following:

1. Hot-cycle washing and drying of all clothing and bedding
2. Institution of basic personal hygiene and sanitation measures
3. Treatment of sexual contacts with active infestations
4. Examination and laboratory testing of patients and their sexual contacts for other sexually transmitted diseases, especially crusted scabies and AIDS.

Key References

The complete reference list is available online at Expert Consult.

2. Drali R, Shako J-C, Davoust B, et al. A new clade of African body and head lice infected by *Bartonella quintana* and *Yersinia pestis*—Democratic Republic of the Congo. A. *J Trop Med Hyg*. 2015;93:990–993.
7. Boutellis A, Mediannikov O, Bilcha KD, et al. *Borrelia recurrentis* in head lice, Ethiopia. *Emerg Infect Dis*. 2013;19:796–798.
8. Lebwohl M, Clark L, Levitt J. Therapy for head lice based on life cycle, resistance, and safety considerations. *Pediatrics*. 2007;119:965–974.
9. Downs AM, Stafford KA, Harvey I, et al. Evidence for double resistance to permethrin and malathion in head lice. *Br J Dermatol*. 1999;141:508–511.
17. Chosidow O, Giraudeau B, Cottrell J, et al. Oral ivermectin versus malathion lotion for difficult-to-treat head lice. *N Engl J Med*. 2010;363:896–905.
18. Pariser DM, Meinking TL, Bell M, et al. Topical 0.5% ivermectin lotion for treatment of head lice. *N Engl J Med*. 2012;367:1687–1693.

References

1. Drali R, Mumcuoglu KY, Yesilyurt G, et al. Studies of ancient lice reveal unsuspected past migrations of vectors. *Am J Trop Med Hyg*. 2015;93:623–625.
2. Drali R, Shako J-C, Davoust B, et al. A new clade of African body and head lice infected by *Bartonella quintana* and *Yersinia pestis*—Democratic Republic of the Congo. A. *J Trop Med Hyg*. 2015;93:990–993.
3. Pape M, Kollaras P, Mandraveli K, et al. Occurrence of *Bartonella henselae* and *Bartonella quintana* among human immunodeficiency virus-infected patients. *Ann N Y Acad Sci*. 2005;1063:299–301.
4. Loutit JS. *Bartonella* infections: diverse and elusive. *Hosp Pract (Minneap)*. 1998;33:37–38.
5. Maurin M, Birtles R, Raoult D. Current knowledge of *Bartonella* species. *Eur J Clin Microbiol*. 1997;16:487–506.
6. Bonilla DL, Kabeya H, Henn J, et al. *Bartonella quintana* in body lice and head lice from homeless persons, San Francisco, California, USA. *Emerg Infect Dis*. 2009;15:912–915.
7. Boutellis A, Mediannikov O, Bilcha KD, et al. *Borrelia recurrentis* in head lice, Ethiopia. *Emerg Infect Dis*. 2013;19:796–798.
8. Lebwohl M, Clark L, Levitt J. Therapy for head lice based on life cycle, resistance, and safety considerations. *Pediatrics*. 2007;119:965–974.
9. Downs AM, Stafford KA, Harvey I, et al. Evidence for double resistance to permethrin and malathion in head lice. *Br J Dermatol*. 1999;141:508–511.
10. Roberts RJ, Casey D, Morgan DA, et al. Comparison of wet combing with malathion for treatment of head lice in the UK: a pragmatic randomized controlled trial. *Lancet*. 2000;356:540–544.
11. Meinking TL, Serrano L, Hard B, et al. Comparative in vitro pediculicidal efficacy of treatments in a resistant head lice population in the United States. *Arch Dermatol*. 2002;138:220–224.
12. Hill N. Control of head lice: past, present and future. *Exp Rev Anti Infect Ther*. 2006;4:887–894.
13. Uziel Y, Adler A, Aharonowitz G, et al. Unintentional childhood poisonings in the Sharon area in Israel: a prospective 5-year study. *Pediatr Emerg Care*. 2005;21:248–251.
14. Diaz JH. Increasing pesticide-resistant ectoparasitic infections may increase pesticide poisoning risks in children. *J la State Med Soc*. 2008;160:210–220.
15. Burkhart CG. Relationship of treatment-resistant head lice to the safety and efficacy of pediculicides. *Mayo Clin Proc*. 2004;79:661–666.
16. Badakhsh R, Lackovic M. Pesticide exposures among children less than seven years of age—Louisiana pesticide surveillance data, 2003-2007. *La Morb Rep*. 2009; 125(March–April):4–6.
17. Chosidow O, Giraudeau B, Cottrell J, et al. Oral ivermectin versus malathion lotion for difficult-to-treat head lice. *N Engl J Med*. 2010;363:896–905.
18. Pariser DM, Meinking TL, Bell M, et al. Topical 0.5% ivermectin lotion for treatment of head lice. *N Engl J Med*. 2012;367:1687–1693.
19. Zalaudek I, Argenziano G. Dermoscopy of nits and pseudonits. *N Engl J Med*. 2012;367:1741.

293 Scabies

James H. Diaz

SHORT VIEW SUMMARY

Definition
- Scabies is an infestation by the itch or scabies mite *Sarcoptes scabiei* var. *hominis* and has become a significant reemerging ectoparasitosis in its most severe form, as crusted or Norwegian scabies, among homeless people, institutionalized older adults, individuals with intellectual disability, and immunocompromised individuals.

Epidemiology
- The worldwide annual prevalence of scabies has been estimated to be about 300 million cases.
- Scabies occurs worldwide in both sexes, at all ages, and among all ethnic and socioeconomic groups. Scabies is hyperendemic throughout the developing world, especially in sub-Saharan Africa, India, the Aboriginal regions of northern Australia, and the South Pacific Islands, especially the Solomon Islands.
- Crusted or Norwegian scabies is highly transmissible in the hospital environment.

Microbiology
- The human scabies mite is an obligate parasite and completes its entire life cycle on its human hosts, as females burrow intradermally to lay eggs and larvae emerge and mature to reinfest the same or new hosts. The entire incubation period from eggs to full-grown mites lasts 14 to 15 days.
- The human incubation period from initial infestation to symptom development is 3 to 6 weeks in initial infestations and 1 to 3 days in reinfestations as a result of prior sensitization to mite antigens.

Diagnosis
- The diagnosis of scabies is made by epidemiologic considerations and clinical observations.
- A clinical diagnosis may be confirmed by low-power microscopic examination of a burrow skin scraping that excavates the female mite.
- Skin biopsy may help confirm the diagnosis in atypical cases.
- Newer diagnostic methods for scabies include enhanced microscopic techniques (dermoscopy), immunologic detection of specific scabies antibodies, and molecular identification of scabies DNA.

Therapy
- Both topical 5% permethrin and oral ivermectin appear most effective for individual classic scabies.
- The management of crusted (Norwegian) scabies may require combined, intense scabicidal therapies with both topical 5% permethrin and oral ivermectin, especially in high-risk community and institutional outbreaks.

Prevention
- Aggressive treatment of infested patients and all close household, institutional, and sexual contacts, especially in cases of highly infectious crusted (Norwegian) scabies, is required.
- Disposal or hot wash-dry sterilization (by machine washing and drying at 60°C [140°F] or higher) of all contaminated clothing and bedding of index cases is essential.
- Provision of improved access for personal hygiene and health care for all displaced, homeless, or institutionalized people should be implemented.

Scabies, an infection by the itch or scabies mite, *Sarcoptes scabiei* var. *hominis,* remains a major public health problem throughout the developing world (Fig. 293.1). Scabies in its most severe form, crusted or Norwegian scabies (Fig. 293.2), has now become a significant reemerging ectoparasitosis in the developed world, especially among homeless people, institutionalized older adults, individuals with intellectual disability, and immunocompromised individuals.[1]

EPIDEMIOLOGY

The worldwide annual prevalence of scabies has been estimated to be about 300 million cases.[2] Although more often associated with crowding, homelessness, and institutionalization, scabies occurs worldwide in both sexes, at all ages, and among all ethnic and socioeconomic groups. In the United Kingdom, scabies is more prevalent in women and children living in urban areas and occurs more often in winter than summer.[3] In a prospective survey in Belgium, Lapeere and colleagues[4] reported a crude incidence for scabies of 28 cases/100,000 inhabitants per year. The highest annual incidence of scabies was noted in immigrants (88/100,000) and in people older than age 75 years (51/100,000). Scabies is hyperendemic throughout the developing world, especially in sub-Saharan Africa (13% annual prevalence rate), India, the Aboriginal regions of northern Australia, and the South Pacific Islands, especially the Solomon Islands.[2,5,6]

Scabies infections with crusted (Norwegian) scabies are more prevalent among several specific high-risk groups including men who have sex with men, patients treated in sexually transmitted disease clinics, homeless individuals with the acquired immunodeficiency syndrome (AIDS), and patients with human T-cell lymphotropic virus type 1 (HTLV-1) infection.[1,7,8] Many experts now recommend evaluating all high-risk patients with crusted scabies for human immunodeficiency virus (HIV) and HTLV-1 infection.[9] In a prospective study of 23 patients with crusted scabies in Peru, HTLV-1 infection was diagnosed in 16 patients (69.6%) by enzyme-linked immunosorbent assay and confirmed by Western immunoblot analysis.[8] In addition to HTLV-1 infection, other significant comorbid features for crusted scabies in the Peruvian study included corticosteroid therapy (8.6%), malnutrition (8.6%), and Down syndrome (4.3%).

TRANSMISSION

In contrast to ectoparasitic fleas and flies, scabies mites cannot jump or fly, but they can crawl at a rate of 2.5 cm/min on warm, moist skin (see Fig. 293.1).[2] They can survive for 24 to 36 hours at room temperature and average humidity and remain capable of infesting humans.[10] Scabies is most easily transmitted by skin-to-skin contact, as with sex partners and children playing, as well as health care providers examining highly infectious patients with crusted scabies. High-risk sexual behaviors for contracting scabies include sporadic sexual contacts and men who have sex with men.[4] Scabies mites have not been demonstrated to transmit HIV, HTLV-1, or any other infectious agent. The more mites there are on a human host, the greater is the risk for transmission by close direct contact, more so than by indirect contact with fomites, such as shared bedding and clothing. Although rare, indirect transmission of scabies

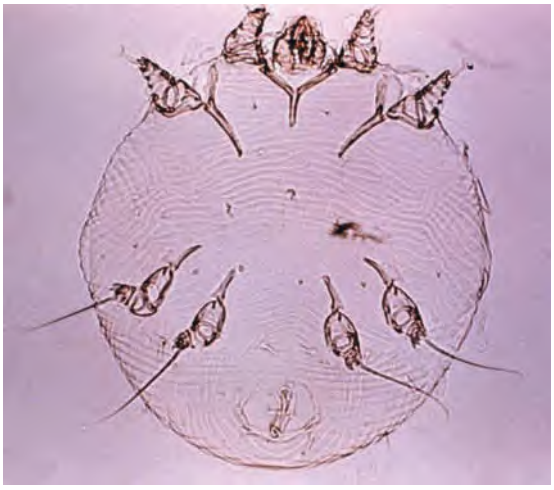


FIG. 293.1 Micrograph of a cleared and slide-mounted scabies mite, *Sarcoptes scabiei* (ventral view). *(From Centers for Disease Control and Prevention [CDC], Atlanta, GA. CDC Public Health Image Library, image 6301.)*

FIG. 293.2 Crusted (Norwegian) scabies on the extensor surface of the elbow secondarily infected with *Staphylococcus aureus.* Note the confluence of the crusts and pustules and the similarity of the lesion to psoriasis, both in its hyperkeratotic appearance and in its location on an extensor surface. Risk factors for crusted scabies include immunocompromise secondary to advanced age, prolonged glucocorticoid therapy, cancer chemotherapy, and human immunodeficiency virus or human T-cell lymphotropic virus type 1 infection. *(From Fitzpatrick TB, Johnson RA, Wolff K, Suurmond D.* Color Atlas and Synopsis of Clinical Dermatology. *4th ed. New York: McGraw-Hill; 2001:841.)*

occurs and is more common in immunocompromised hosts with AIDS, in family members of an index atypical (crusted) case, and within the institutional settings described.[1]

Several nonhuman species of sarcoptic mites can cause animal scabies with itching, inflammation, and hair loss. Animal scabies occurs commonly in domestic pets and animals, especially in cats, dogs, pigs, horses, and camels. Immunocompromised individuals may also contract animal scabies from domestic animals, usually dogs, with sarcoptic mange. Animal scabies mites are facultative ectoparasites in humans and cannot effectively complete their life cycles in human (dead-end) hosts. Infections are usually self-limited in humans but can be treated successfully, if indicated, with 5% permethrin lotion, 10% crotamiton cream or lotion, or oral ivermectin.

FIG. 293.3 Dorsal view of an older patient's hand demonstrating a crusted scabies infestation by the scabies mite, *Sarcoptes scabiei.* Note the localized crusting in the interdigital web spaces. *(From Centers for Disease Control and Prevention [CDC], Atlanta, GA. CDC Public Health Image Library, image 4800.)*

CLINICAL MANIFESTATIONS

The human scabies mite is an obligate parasite and completes its entire life cycle on its human hosts, as females burrow intradermally to lay eggs and larvae emerge and mature to reinfest the same or new hosts. Female mites burrow preferentially into the thinner areas of the epidermis by dissolving the stratum corneum with proteolytic secretions. Burrows are usually no deeper than the stratum granulosum. Female mites then lay their eggs at the end of tunneled burrows 5 to 10 mm long, and larvae hatch 2 to 3 days after eggs are laid. The entire incubation period from eggs to full-grown mites lasts 14 to 15 days.[11] The human incubation period from initial infection to symptom development is 3 to 6 weeks in initial infections and 1 to 3 days in reinfections as a result of prior sensitization to mite antigens.[2]

Classic or typical scabies manifests as generalized, intense nocturnal itching in a characteristic topographic distribution as 10 to 15 fertile female mites are transferred from infected patients to new hosts. The skin eruptions in reinfections and atypical forms of scabies are considered consequences of infection and hypersensitivity reactions to mite antigens.[2]

In classic scabies, the preferred distribution of skin eruptions includes hairless areas with a thin stratum corneum, such as the sides and interdigital web spaces of fingers and toes, popliteal fossae, flexor surfaces of the wrists, buttocks, and female breasts (Fig. 293.3).[2] Although inflammatory pruritic papules are present at most infested sites, the pathognomonic linear to serpiginous intradermal burrows, 5 to 10 mm long, dotted with fecal lithes (pellets) or scybala, and terminating in raised papules hiding ovipositing females, may be absent. Nonspecific secondary lesions occur commonly as the result of scratching and secondary infection and include self-inflicted excoriations, eczematization, lichenification, and impetigo.

Bullous scabies is a rare subtype of classic scabies, with only 44 cases reported to date.[12] The highly pruritic bullous lesions resemble bullous pemphigoid and most commonly cluster on the trunk and extremities but may involve genitals, feet, thighs, inguinal folds, and neck.[12] Facial and mucosal involvement has not been reported.[12] Risk factors for bullous scabies include male sex and advanced age.[12] To date, all cases have been successfully treated with available antiscabietic therapies.[12]

In addition to classic and bullous scabies, scabies may also manifest in three atypical forms, especially in high-risk institutionalized or immunocompromised individuals with HIV or HTLV-1 infections. These atypical forms of scabies include scalp scabies in infants, crusted (Norwegian) scabies in institutionalized and immunocompromised individuals and in high-risk populations such as Aboriginal populations in remote areas of tropical northern Australia, and sexually transmitted nodular scabies. Scabietic nodules develop in 7% to 10% of patients with scabies infections, usually in men on the penis and scrotum, and

appear as darkened, tender nodules 5 to 20 mm in diameter, often with a raised female mite burrow on top (Fig. 293.4). The atypical forms of scabies are compared with classic scabies and stratified by high-risk human host populations, clinical manifestations, and differential diagnoses in Table 293.1.

FIG. 293.4 Sexually transmitted nodular scabies infestation in a man caused by the scabies mite, *Sarcoptes scabiei.* Note the nodular pustular lesions clustered around the umbilicus and inner thighs. *(From Centers for Disease Control and Prevention [CDC], Atlanta, GA. CDC Public Health Image Library, image 6538.)*

DIAGNOSIS

Although newer diagnostic methods are under investigation, the diagnosis of scabies is made predominantly by epidemiologic considerations and clinical observations. A clinical diagnosis may be confirmed by low-power microscopic examination of a burrow skin scraping that excavates the female mite, 0.2 to 0.5 mm in length, translucent with brown legs, and too small to be seen with the unassisted eye. Eggs (0.02–0.03 mm in diameter), smaller eggshell fragments, and fecal lithes or pellets may also be identified in microscopic specimens of burrow scrapings (see Fig. 293.1).[2] Potassium hydroxide should not be used to mount the burrow scrapings because it can dissolve mite fecal lithes. As noted, failure to identify pathognomonic burrows and to find mites is common, particularly in initial cases with low ectoparasite burdens, and does not rule out scabies. One of the simplest and most often overlooked bedside tests to identify scabies mite burrows is the burrow ink test (BIT), which relies on ink to highlight burrows. In the BIT, fountain pen ink or any liquid ink is gently rubbed on skin surfaces with suspected scabies and then wiped off with an alcohol pad to reveal the ink-highlighted wavy tunnels made by the burrowing female mite in the stratum corneum.

In atypical scabies cases, skin biopsy may help confirm the diagnosis.[2] Although untested in controlled trials in large study populations, newer diagnostic methods for scabies now under investigation include enhanced microscopic techniques (e.g., epiluminescence microscopy, computed or digital dermoscopy, or videodermoscopy), immunologic detection of specific scabies antibodies by enzyme-linked immunosorbent assay, and molecular identification of scabies DNA by polymerase chain reaction assay.[13,14-16] Handheld dermoscopy (also known as dermatoscopy) is an accurate test that requires a dermatoscope and a trained microscopist.[16] The simplest dermatoscopes consist of a low-power (10×) magnifier, a nonpolarized light source, and a liquid medium between the light source and a transparent lens to limit skin reflections. More sophisticated dermatoscopes, such as digital epiluminescence dermatoscopes, rely on polarized light rather than a liquid medium to cancel out skin surface reflections and can be wirelessly linked to computers for videodermoscopy and display screens for real-time imaging and video capturing of scabies lesions. Low-cost videomicroscopy was compared with videodermoscopy in a small trial that showed noninferiority of videomicroscopy, which may have implications in diagnostic testing in settings with scarce resources.[17] Serologic and molecular methods for diagnosing scabies are under development and testing and are not universally available.[16] At the present time, epidemiologic considerations and clinical observations,

TABLE 293.1 Different Presenting Forms of Scabies

PRESENTING FORMS OF SCABIES	SPECIFIC HIGH-RISK POPULATIONS	CLINICAL MANIFESTATIONS	LIMITED DIFFERENTIAL DIAGNOSES
Classic scabies (scabies vulgaris)	Infants and children; sexually active adults; men who have sex with men	Intense generalized pruritus, worse at night; inflammatory pruritic papules localized to finger webs, flexor aspects of wrists, elbows, axillae, buttocks, genitalia, female breasts; lesions and pruritus spare the face, head, and neck; secondary lesions include eczematization, excoriation, impetigo	Dermatitis herpetiformis, drug reactions, eczema, pediculosis corporis, lichen planus, pityriasis rosea
Scalp scabies	Infants and children; institutionalized older adults; AIDS patients; patients with preexisting crusted scabies	Atypical crusted papular lesions of the scalp, face, palms, and soles	Dermatomyositis, ringworm, seborrheic dermatitis
Crusted scabies (Norwegian scabies, scabies norvegica, scabies crustosa)	Institutionalized older adults; institutionalized developmentally disabled (Down syndrome) individuals; homeless people, especially HIV-positive; all immunocompromised patients, particularly those with AIDS or positive for HIV or HTLV-1; transplant recipients; patients on prolonged systemic corticosteroid therapy and chemotherapy	Psoriasiform hyperkeratotic papular lesions of the scalp, face, neck, hands, feet, with extensive nail involvement; eczematization and impetigo common	Contact dermatitis, drug reactions, eczema, erythroderma, ichthyosis, psoriasis
Nodular scabies	Sexually active adults; men who have sex with men; HIV-positive men > HIV-positive women	Violaceous pruritic nodules localized to male genitalia, groin, axillae, representing hypersensitivity reaction to mite antigens	Acropustulosis, atopic dermatitis, Darier disease, lupus erythematosus, lymphomatoid papulosis, papular urticaria, necrotizing vasculitis, secondary syphilis

AIDS, Acquired immunodeficiency syndrome; *HIV,* human immunodeficiency virus; *HTLV-1,* human T-cell lymphotropic virus type 1.

TABLE 293.2 Currently Recommended Treatment for Scabies

SCABICIDES	FDA APPROVED?	PREGNANCY CATEGORY[A]	DOSING SCHEDULE	SAFETY PROFILE	CONTRAINDICATIONS
5% Permethrin cream (Actin, Nix, Elimite)	Yes	B	Apply from neck down; wash off after 8–14 h; good residual activity, but second application recommended after 1 wk	Excellent; itching and stinging on application	Prior allergic reactions; infants <2 mo of age; breastfeeding
1% Lindane lotion or cream	Yes	B	Apply 30–60 mL from neck down; wash off after 8–12 h; no residual activity; increasing drug resistance	Potential for central nervous system toxicity from organochloride poisoning, usually manifesting as seizures, with overapplication and ingestions	Preexisting seizure disorder; infants and children <6 mo of age; pregnancy; breastfeeding
10% Crotamiton cream or lotion (Eurax)	Yes	C	Apply from neck down on 2 consecutive nights; wash off 24 h after second application	Excellent; not very effective; exacerbates pruritus	None
2%–10% Sulfur in petrolatum ointments	No	C	Apply for 2–3 days, then wash	Excellent; not very effective	Preexisting sulfur allergy
10%–25% Benzoyl benzoate lotion	No	None	Two applications for 24 h with 1-day to 1-wk interval	Irritant; exacerbates pruritus; can induce contact irritant dermatitis and pruritic cutaneous xerosis	Preexisting eczema
0.5% Malathion lotion (Ovide), 1% malathion shampoo (unavailable in United States)	No	B	95% Ovicidal; rapid (5 min) killing; good residual activity; increasing drug resistance	Flammable 78% isopropyl alcohol vehicle stings eyes, skin, mucosa; increasing drug resistance; organophosphate poisoning risk with overapplication and ingestions	Infants and children <6 mo of age; pregnancy; breastfeeding
Ivermectin (Stromectol)	Yes	C	200-μg/kg single PO dose, may be repeated in 14–15 days; not ovicidal, second dose on day 14 or 15 highly recommended; recommended for endemic or epidemic scabies in institutions and refugee camps	Excellent; may cause nausea and vomiting; take on empty stomach with water	Safety in pregnancy uncertain; probably safe during breastfeeding; not recommended for children <5 yr of age or weighing <15 kg

[a]US Food and Drug Administration safety in pregnancy categories: A, safety established; B, presumed safe; C, uncertain safety; D, unsafe; X, highly unsafe.
PO, Per os (orally).

often aided by bedside techniques such as the BIT and dermoscopy, remain the most rapid and practical methods for diagnosing scabies.[16]

THERAPY

Topical or oral scabicides should be used to treat infested persons and their close personal contacts simultaneously, regardless of the presence of symptoms.[2] Currently recommended treatment options for scabies are listed in Table 293.2. In a review on the treatment of scabies, Strong and Johnstone[18] noted that both topical 5% permethrin and oral ivermectin appear most effective for individual infections, more research would be needed to compare the effectiveness of malathion with permethrin for individual infections, and there was insufficient evidence at the present time to recommend specific miticides to control community and institutional outbreaks of scabies. The most effective topical agents for scabies are 5% permethrin cream and 1% lindane cream or lotion, with permethrin safer and slightly more effective than lindane, which is an organochlorine pesticide capable of causing seizures and sudden death caused by overapplication or accidental ingestions.[19,20] The other topical agents for scabies include 10% to 25% benzoyl benzoate lotions (not available in the United States), 10% crotamiton cream or lotion, 2% to 10% sulfur in petrolatum ointments, and 0.8% ivermectin lotion (not available in the United States) (see Table 293.2).

The topical agents for scabies may not be well accepted or tolerated by some patients for many reasons, including severe burning and stinging (with 25% benzyl benzoate and 5% permethrin) in cases of secondarily excoriated or eczematous infections and inability of demented or disabled patients to comply with application regimens. In such cases, two doses of oral ivermectin, 200 μg/kg/dose, taken with food, one dose on day 1 and a second dose between days 8 and 15, may offer a more acceptable and equally effective alternative. In a 2007 prospective trial, Sule and Thacher[21] compared the effectiveness of oral ivermectin, 200 μg/kg/dose, with topical 25% benzoyl benzoate and monosulfiram soap in 210 Nigerian patients 5 to 65 years of age with scabies. Subjects with persistent lesions received a second course of therapy after 2 weeks. The investigators observed resolution of all lesions in 77 of 98 subjects (79%) treated with ivermectin and in 60 of 102 subjects (59%) treated topically ($P = .003$). The scabies cure rate at 4 weeks was 95% in the ivermectin group and 86% in the topical treatment group ($P = .04$). It was concluded that oral ivermectin is as effective as topical treatment with benzyl benzoate and monosulfiram in scabies and leads to more rapid improvement. Ivermectin, however, is not ovicidal, and a second course of oral treatment at adult mite maturation time of 14 to 15 days is now recommended.[2,22]

In a prospective trial comparing oral ivermectin with topical 5% permethrin in scabies, Usha and Gopalakrishnan Nair[22] reported a 70% cure rate with a single dose of ivermectin, compared with a 95% cure rate with topical 5% permethrin ($P < .003$), but a second dose of ivermectin, 200 μg/kg, taken 2 weeks later, increased the cure rate to 95%. Nevertheless, the US Centers for Disease Control and Prevention (CDC) recommends topical 5% permethrin cream or lotion as first-line therapy for scabies, especially in initial classic infections.[23]

Scabies mite drug resistance to both topical 5% permethrin preparations and oral ivermectin has emerged in severe outbreaks of crusted scabies in nursing homes and in the hyperendemic regions noted.[24,25] As a result, the management of crusted scabies may require combined, intense scabicidal therapies in high-risk community and institutional outbreaks. Currie and McCarthy[26] have recommended both 5% topical permethrin every 2 to 3 days for 1 to 2 weeks and oral ivermectin, 200 μg/kg/dose, taken with food and administered as three doses (days 1, 2, and 8), five doses (days 1, 2, 8, 9, and 15), or seven doses, depending on the severity of the infection. For refractory institutional and community outbreaks, the authors recommended combined therapy with topical permethrin and oral ivermectin for all symptomatic cases with classic or crusted scabies and a single oral dose of ivermectin, 200 μg/kg, for all exposed, asymptomatic residents, visitors, and staff.[26]

The emerging resistance of the scabies mite to commonly used standard treatments, specifically topical permethrin and oral ivermectin, will limit the future therapeutic usefulness of these agents and encourage

the development of new and effective acaricidal agents.[27] Tea tree oil (TTO) has already demonstrated acaricidal effects against scabies mites in vitro and has been used as an effective topical adjunct for treating crusted scabies not responding to standard therapies.[28] Thomas and coworkers[28] reviewed the therapeutic potential of TTO for scabies and recommended larger scale randomized controlled trials comparing the therapeutic efficacy of TTO with standard treatments for scabies, especially crusted scabies and resistant scabies.

PREVENTION

Prevention and control strategies for scabies include the following: (1) aggressive treatment of infested patients and all close household, institutional, and sexual contacts, especially in cases of highly infectious crusted scabies; (2) disposal or hot wash-dry sterilization (by machine washing and drying at 60°C [140°F] or higher) of all contaminated clothing and bedding of index cases; (3) provision of improved access for personal hygiene and health care for all displaced, homeless, or institutionalized persons; and (4) aggressive control of outbreaks of zoonotic scabies with the potential for human transmission caused by the sarcoptic mites of various domestic animals, especially cats, dogs, camels, pigs, and horses.[2,25]

As noted, epidemic outbreaks of scabies that are often associated with impetigo and are difficult to control frequently plague isolated, impoverished communities in the Aboriginal regions of northwestern Australia and throughout the South Pacific islands. In a prospective study designed to control endemic scabies in isolated island communities, Romani and coworkers[28] randomly assigned three island communities in Fiji to one of three different drug treatment interventions for mass scabies control: (1) topical permethrin to affected persons and their contacts (standard care group, $n = 803$); (2) mass topical permethrin administration (permethrin group, $n = 532$); and (3) mass administration of oral ivermectin (ivermectin group, $n = 716$).[28] Although the prevalence of scabies declined significantly in all three groups, the greatest decline was observed in the ivermectin group (relative reduction 94% from a prevalence rate of 32.1% to 1.9%). The prevalence of impetigo also declined significantly in all three groups, with the greatest decline again in the ivermectin group (relative reduction 67%).[28] The authors concluded that in addition to improvements in living standards, the mass administration of scabicides, especially oral ivermectin, was efficacious for the population-based control of endemic scabies and associated impetigo.[28]

CONCLUSION

All patients with scabies and their close household, institutional, and sexual contacts should be informed that scabies is a transmissible ectoparasitic infection and that several topical treatments and an effective oral treatment are now readily available and highly effective (see Table 293.2). Precise diagnosis should be confirmed, if possible, by microscopic, immunologic, or molecular methods. Topical 5% permethrin preparations remain reasonable first-line therapies for classic scabies and are recommended by the CDC. Topical 25% benzoyl benzoate preparations are equally effective alternative choices, especially for crusted scabies.[26] Oral ivermectin, 200 μg/kg initially and repeated in 2 weeks, may be preferred for patients who cannot tolerate topical therapies, who are unable to adhere to topical application schedules, or who have atypical or topical drug–resistant scabies. Sexually active patients with nodular scabies should be screened for other sexually transmitted diseases. Serious consideration should be given to screening patients with crusted scabies for HIV and HTLV-1 infections, particularly in scabies and HTLV-1 hyperendemic regions of the world and in homeless shelter outbreaks of crusted scabies. Future molecular investigations of scabies mite biology and genetic drug resistance are needed to permit the development of better diagnostic tools and treatment strategies for human scabies, especially atypical scabies.

Key References

The complete reference list is available online at Expert Consult.

8. Blas M, Bravo F, Wenceslao C, et al. Norwegian scabies in Peru: the impact of human T cell lymphotropic virus type 1 infection. *Am J Trop Med Hyg*. 2005;72:855–857.
14. Prins C, Stucki L, French L, et al. Dermoscopy for the in vivo detection of *Sarcoptes scabiei*. *Dermatology*. 2004;208:241–243.
17. Micali G, Lacarrubba F, Verzì AE, et al. Low-cost equipment for diagnosis and management of endemic scabies outbreaks in underserved populations. *Clin Infect Dis*. 2015;60:327–329.
18. Strong M, Johnstone PW. Interventions for treating scabies. *Cochrane Database Syst Rev*. 2007;(3):CD000320.
21. Sule HM, Thacher TD. Comparison of ivermectin and benzyl benzoate lotion for scabies in Nigerian patients. *Am J Trop Med Hyg*. 2007;76:392–395.
22. Usha V, Gopalakrishnan Nair TV. A comparative study of oral ivermectin and topical permethrin cream in the treatment of scabies. *J Am Acad Dermatol*. 2000;42:236–240.
25. Arlian LG, Estes SA, Vyszenski-Moher DL. Prevalence of *Sarcoptes scabiei* in the homes and nursing homes of scabietic patients. *J Am Acad Dermatol*. 2002;46:794–796.
26. Currie BJ, McCarthy JS. Permethrin and ivermectin for scabies. *N Engl J Med*. 2010;362:717–725.

References

1. Tijoe M, Vissers WH. Scabies outbreaks in nursing homes for the elderly: recognition, treatment options and control of reinfestation. *Drugs Aging*. 2008;25:299–306.
2. Chosidow O. Scabies. *N Engl J Med*. 2006;354:1718–1727.
3. Downs AMR, Harvey I, Kennedy CTC. The epidemiology of head lice and scabies in the UK. *Epidemiol Infect*. 1999;122:471–477.
4. Lapeere H, Naeyaert JM, DeWeert J, et al. Incidence of scabies in Belgium. *Epidemiol Infect*. 2008;136:395–398.
5. Mahé A, Faye O, N'Diaye HT, et al. Definition of an algorithm for the management of common skin diseases at primary health care level in sub-Saharan Africa. *Trans R Soc Trop Med Hyg*. 2005;99:39–47.
6. Lawrence G, Leafasia J, Sheridan J, et al. Control of scabies, skin sores and haematuria in children in the Solomon Islands: another role for ivermectin. *Bull World Health Organ*. 2005;83:34–42.
7. Otero L, Varela JA, Espinosa E, et al. *Sarcoptes scabiei* in a sexually transmitted infections unit: a 15-year study. *Sex Transm Dis*. 2004;31:761–765.
8. Blas M, Bravo F, Wenceslao C, et al. Norwegian scabies in Peru: the impact of human T cell lymphotropic virus type 1 infection. *Am J Trop Med Hyg*. 2005;72:855–857.
9. Del Guidice P, Sainte Marie D, Gerard Y, et al. Is crusted (Norwegian) scabies a marker of adult T cell leukemia/lymphoma in human T-lymphotropic virus type 1-seropositive patients? *J Infect Dis*. 1997;176:1090–1092.
10. Arlian LG, Runyan RA, Achar S, et al. Survival and infectivity of *Sarcoptes scabiei* var. *canis* and var. *hominis*. *J Am Acad Dermatol*. 1984;11:210–215.
11. Walton SF, Holt DC, Currie BJ, et al. Scabies: new future for a neglected disease. *Adv Parasitol*. 2004;57:309–376.
12. Luo D-Q, Huang M-X, Liu J-H, et al. Bullous scabies. *Am J Trop Med Hyg*. 2016;95:689–693. Case report.
13. Argenziano G, Fabbrocini G, Delfino M. Epiluminescence microscopy: a new approach to in vivo detection of *Sarcoptes scabiei*. *Arch Dermatol*. 1997;133:751–753.
14. Prins C, Stucki L, French L, et al. Dermoscopy for the in vivo detection of *Sarcoptes scabiei*. *Dermatology*. 2004;208:241–243.
15. Walton SF, McBroom J, Mathews JD, et al. Crusted scabies: a molecular analysis of *Sarcoptes scabiei* variety *hominis* populations from patients with repeated infestations. *Clin Infect Dis*. 1999;29:1226–1230.
16. Leung V, Miller M. Detection of scabies: a systematic review of diagnostic methods. *Can J Infect Dis Med Microbiol*. 2011;22:143–146.
17. Micali G, Lacarrubba F, Verzì AE, et al. Low-cost equipment for diagnosis and management of endemic scabies outbreaks in underserved populations. *Clin Infect Dis*. 2015;60:327–329.
18. Strong M, Johnstone PW. Interventions for treating scabies. *Cochrane Database Syst Rev*. 2007;(3):CD000320.
19. Singal A, Thami GP. Lindane neurotoxicity in childhood. *Am J Ther*. 2006;13:277–280.
20. Sudakin DL. Fatality after a single dermal application of lindane lotion. *Arch Environ Occup Health*. 2007;62:201–203.
21. Sule HM, Thacher TD. Comparison of ivermectin and benzyl benzoate lotion for scabies in Nigerian patients. *Am J Trop Med Hyg*. 2007;76:392–395.
22. Usha V, Gopalakrishnan Nair TV. A comparative study of oral ivermectin and topical permethrin cream in the treatment of scabies. *J Am Acad Dermatol*. 2000;42:236–240.
23. Centers for Disease Control and Prevention. Scabies; 2008. http://www.cdc.gov/scabies.
24. Mounsey KE, Holt DC, McCarthy J, et al. Scabies: molecular perspectives and therapeutic implications in the face of emerging drug resistance. *Future Microbiol*. 2008;3:57–66.
25. Arlian LG, Estes SA, Vyszenski-Moher DL. Prevalence of *Sarcoptes scabiei* in the homes and nursing homes of scabietic patients. *J Am Acad Dermatol*. 2002;46:794–796.
26. Currie BJ, McCarthy JS. Permethrin and ivermectin for scabies. *N Engl J Med*. 2010;362:717–725.
27. Thomas J, Carson CF, Peterson GM, et al. Therapeutic potential of tea tree oil for scabies. *Am J Trop Med Hyg*. 2016;94:258–266. Review article.
28. Romani L, Whitfeld MJ, Koroivueta J, et al. Mass drug administration for scabies control in a population with endemic disease. *N Engl J Med*. 2015;373:2305–2313.

294 Myiasis and Tungiasis

James H. Diaz

SHORT VIEW SUMMARY

MYIASIS

Definition

- Myiasis is an ectoparasitic infestation of viable or necrotic tissues by the dipterous larvae of higher flies. Furuncular myiasis is the most common clinical manifestation of myiasis and occurs when one or more fly larvae penetrate the skin, causing pustular lesions that resemble boils or furuncles.

Epidemiology

- Myiasis is an opportunistic infestation that occurs in travelers returning from tropical jungles and in vulnerable populations living in endemic areas of the tropics.

Microbiology

- In human botfly furuncular myiasis, botfly larvae rapidly burrow into the skin with sharp mandibles to begin their developmental instar stages, which can last 6 to 12 weeks and cause draining, boil-like lesions, or furuncles.

Diagnosis

- The diagnosis of myiasis is usually by clinical inspection and examination, often with microscopy. Some immunodiagnostic tests have been developed to detect the antibodies to the antigens of specific fly species causing myiasis.

Therapy

- Furuncular myiasis may be treated conservatively by coaxing embedded larvae from furuncles by smothering their respiratory spiracles with occlusive coatings of petrolatum (Vaseline), clear fingernail polish, tobacco tar, pork fat, raw beefsteak, or bacon strips. However, unsuccessful occlusive therapy may asphyxiate larvae and necessitate their surgical or vacuum extraction. Along with larval removal, myiasis wounds should be cleansed and conservatively débrided, tetanus prophylaxis administered, and bacterial secondary infections treated with antibiotics.

Prevention

- Methods include control of domestic and livestock animal larval infestations; sanitary disposal of animal carcasses and offal to deny flies their preferred breeding grounds; proper management of any open human wounds or cutaneous infections; cementing floors to deny floor maggot flies their preferred egg-laying surfaces; sleeping on raised beds or cots in screened huts or tents; wearing long-sleeved shirts and pants, which can be pyrethrin- or pyrethroid-impregnated; spraying exposed skin with diethyl toluamide (*N,N*-diethyl-meta-toluamide [DEET])–containing repellents; and ironing both sides of all clothes and diapers left outside to dry in tumbu fly habitats.

TUNGIASIS

Definition

- Tungiasis is a painful, cutaneous infestation with the gravid female jigger flea that usually occurs on the feet but may occur anywhere where bare skin touches soil containing gravid female fleas.

Epidemiology

- In travelers returning to accessible health care infrastructures in developed nations, tungiasis is an exotic infestation with a minimal parasite burden and a simple surgical cure, but in the impoverished and underserved communities of developing tropical nations, tungiasis is a recurrent infestation of the feet with a high parasite burden, causing significant morbidity, including autoamputation.

Microbiology

- In tungiasis the gravid female jigger (chigoe) flea penetrates bare skin, usually on the feet (or heels), under or near the toenails or in the interdigital web spaces, to feed on blood and tissue juices and to incubate hundreds of developing eggs within days; it swells to 2000 times its size and then expels eggs over a period of 3 weeks or less before dying and leaving its shriveled carcass in a contaminated wound tract.

Diagnosis

- The diagnosis of tungiasis is usually by clinical inspection and examination, often with microscopy.

Therapy

- Tungiasis is treated by extracting all embedded fleas immediately with sterile needles or curets, administering tetanus prophylaxis, and treating secondary wound infections with appropriate topical or oral antibiotics.

Prevention

- Methods include wearing shoes, which can be sprayed with pyrethroid or DEET-containing solutions; not sitting naked on bare ground; insecticide treatment of flea-infested domestic and stray animals and pets with 10% pyrethrin or pyrethroid sprays, or 1% to 4% malathion powder; bathing the feet of domestic and stray dogs and pigs with insecticide solutions, such as 2% trichlorfon (Neguvon); and spraying or dusting households, especially those with dirt floors, with 1% to 4% malathion.

Flies and fleas are mostly bothersome biting nuisances of humans and animals that can also transmit infectious diseases and deeply invade living tissues, causing amputation, disfigurement, and, rarely, death. Flies can serve as mechanical vectors of shigellosis, and rat fleas can transmit bubonic plague and murine typhus. Flies may lay their eggs on human flesh, and their developing larvae, or maggots, can invade subcutaneous tissues and penetrate external body cavities, such as the orbits, ears, and nares. Flea larvae can also burrow into subcutaneous tissues to feed, complete their developmental stages or instars, and promote secondary infections with incapacitating sequelae, including autoamputation of toes and fingers, especially in impoverished tropical communities plagued by endemic jigger fleas *(Tunga penetrans)*.

MYIASIS

Myiasis is an ectoparasitic infestation of viable or necrotic tissues by the dipterous larvae of higher flies and may be broadly classified as obligatory or facultative myiasis. In obligatory myiasis, maggots must live and feed on human or animal hosts as part of their life cycle. In facultative myiasis, normally free-living maggots that preferentially feed on carrion and decaying matter attack and feed on the necrotic sores and wounds of living human and animal hosts. Maggot therapy with blowfly larvae is still used today to débride necrotic wounds.

Myiasis may be further stratified clinically as furuncular (subcutaneous) myiasis, wound (superficial cutaneous) myiasis, cavitary (atrial or invasive) myiasis, intestinal myiasis, urinary myiasis, and vaginal myiasis.

Furuncular myiasis is the most common clinical manifestation of myiasis and occurs when one or more larvae penetrate the skin, causing pustular lesions that resemble boils or furuncles. Larval maggots can also infest external orifices, sores, or open wounds, causing cavitary and wound myiasis. Cavitary myiasis is usually caused by screwworm larvae that can penetrate festering wounds or invade the orbits, nostrils, or external ear canals (Figs. 294.1 and 294.2). Intestinal myiasis is uncommon, usually caused by the accidental ingestion of maggot-contaminated food, and characterized by self-limited nausea, vomiting, and diarrhea. Genitourinary myiasis is also uncommon and may present as dysuria, hematuria, and pyuria, after larval invasion of the urethra (urinary myiasis) or vagina (vaginal myiasis).

Although there are many families of dipterous flies (order Diptera), flies from three families cause most human and animal myiasis: Oestridae, or botflies; Calliphoridae, screwworms and blowflies; and Sarcophagidae, carrion-feeding flies. The most common myiasis-causing fly species are classified taxonomically and stratified by clinical type of myiasis infestation in Table 294.1.

Epidemiology

In a retrospective epidemiologic study in Rio de Janeiro, Marquez and colleagues[1] described 71 patients with furuncular and cavitary myiasis during the period 1999–2003. Myiasis was more prevalent among adults older than 51 years (42%) and children younger than 10 years (34%). Most of the population was male (61%) and impoverished (62%). The predominant causative agent of furuncular myiasis was *Dermatobia hominis,* the New World human botfly, and the predominant causative agent of cavitary myiasis was *Cochliomyia macellaria,* an indigenous species of New World screwworm. The authors concluded that myiasis is an opportunistic infestation of disadvantaged vulnerable populations living in nonhygienic conditions. In a similar retrospective collective analysis, Jiang[2] described 54 cases of human myiasis in China from 1995–2001. Although the Chinese cases were equally distributed between genders, most cases occurred in infants and children (72%) and were described as either hypodermic-invasive ($n = 31$) or ocular ($n = 12$). In another collective review, Schwartz and Gur[3] reported 12 cases of furuncular myiasis caused by *D. hominis,* the human botfly, in 12 Israeli travelers returning from four South American countries in the Amazon Basin. Clyti and coworkers[4] described an epidemic of human botfly furuncular myiasis in 30 patients living in coastal urban communities of French Guiana between January and March 2000 after heavy rainfall that accelerated soil maturation of pupae into adults.

Tamir and associates[5] reported two cases of furuncular myiasis caused by *Cordylobia rodhaini,* the Lund fly, in Israeli travelers returning from Ghana. In addition to *D. hominis* and *Cordylobia* spp., *Cuterebra* spp. of botflies can also cause furuncular myiasis in North America and throughout Africa and Asia (Fig. 294.3). Shorter and coworkers[6] reported two cases of *Cuterebra* spp. botfly-induced furuncular myiasis in children in New England (see Fig. 294.3). *Cuterebra* or rabbit botflies are endemic in the northeastern United States and southeastern Canada, where they have established a zoonotic reservoir in lagomorphs (rabbits and hares) and cause periodic cluster outbreaks of furuncular myiasis in domestic animals and humans.[7]

FIG. 294.1 Forearm exit site wound of a third-stage (instar) New World screwworm fly larva or maggot, *Cochliomyia hominivorax.* *((From Seppänen M, Virolainen-Julkunen A, Kakko I, et al. Myiasis during adventure sports race.* Emerg Infect Dis. *2004;10:137–139.)*

FIG. 294.2 16-mm–long third-stage (instar) New World screwworm fly larva or maggot, *Cochliomyia hominivorax.* This had just emerged from a forearm exit wound (see Fig. 294.1) after tissue-feeding in a traumatic wound for 9 days. *(From Seppänen M, Virolainen-Julkunen A, Kakko I, et al. Myiasis during adventure sports race.* Emerg Infect Dis. *2004;10: 137–139.)*

TABLE 294.1 Myiasis-Causing Flies

FAMILY (COMMON FAMILY NAME)	TAXONOMIC CLASSIFICATION	COMMON NAME	GEOGRAPHIC DISTRIBUTION	TYPE OF MYIASIS INFESTATION
Oestridae (botflies)	*Dermatobia hominis*	New World botfly	Caribbean, Central and South America	Furuncular
	Cuterebra spp.	Rodent and rabbit botflies	North America, northern Central America	Furuncular
	Hypoderma sinense	Indian botfly	India	Furuncular
Calliphoridae (screwworm flies, blowflies)	*Cordylobia anthropophaga*	Tumbu fly	Africa	Furuncular
	Cordylobia rodhaini	Lund fly	Africa	Furuncular
	Auchmeromyia senegalensis	Congo floor mat fly	Africa	Superficial cutaneous (no tissue invasion)
	Cochliomyia hominivorax	New World screwworm	Southern North America, Central and South America	Wound, cavitary
	Chrysomyia bezziana			Wound, cavitary
	Lucilia spp.	Old World screwworm	Africa, Asia	Wound, cavitary
	Calliphora spp.	Greenbottle blowflies	Worldwide	Wound (used for maggot therapy)
		Bluebottle blowflies	Worldwide	Wound (used for maggot therapy)
Sarcophagidae (carrion flies)	*Sarcophaga carnaria*		Africa	Wound, cavitary, gastrointestinal
	Wohlfahrtia magnifica		Africa	Wound, cavitary, gastrointestinal

FIG. 294.3 First-stage (instar) larva of a *Cuterebra* spp. botfly native to North America. Note the rows of anterior hooklets that can anchor the feeding larva to the dermis. *(From Centers for Disease Control and Prevention. Public health image library: ID #1427; page last reviewed December 20, 2017. https://phil.cdc.gov/details_linked.aspx?pid=1427. Accessed June 4, 2019.)*

Although dermatoses are among the leading medical complaints in returning travelers, myiasis and tungiasis remain among the least frequently diagnosed dermatologic conditions. However, recently reported case series of myiasis and tungiasis in returning travelers have provided new epidemiologic analyses of the frequencies, distributions, and risk factors for these infections.[8,9] In a retrospective observational study of 90 returning Israeli travelers with myiasis over the reporting period 1999–2014, most cases were caused by *D. hominis* and acquired in Latin America ($n = 72$, 80%), and fewer cases were caused by *Cordylobia* spp. and acquired in Africa ($n = 18$, 20%).[8] Manual extraction was sufficient to remove the embedded larvae in most cases (76%), with fewer cases (24%) requiring surgical extraction.[8] Most patients did not receive antibiotic prophylaxis, and only one patient developed a secondary infection after partial removal of the larva.[8] The authors concluded that myiasis is not a rare dermatoses in returning travelers, with most cases acquired in Latin America not Africa, and treatment should include full larval extraction without antibiotic prophylaxis.[8]

In an outbreak of tungiasis in 13 travelers returning from Madagascar, walking barefoot (100% of cases) and/or wearing open sandals (62% of cases) were the highest risk factors for tungiasis, with a mean clinical incubation period of 15 days or longer than observed in prior case series (7–12 days).[9] Observations such as these in returning travelers have provided clinicians with a better understanding of the causes, clinical incubation periods, and treatments of these infections, which are less rare than generally suspected.[10]

Clinical Manifestations

The most common forms of human myiasis worldwide are furuncular myiasis and cavitary (invasive) myiasis. Furuncular myiasis is most often caused by subcutaneous larval invasion by the tumbu fly, *Cordylobia anthropophaga,* in Africa, and the New World human botfly, *D. hominis,* in the subtropical and tropical areas of the Americas (see Table 294.1). Cavitary myiasis is usually caused by zoonotic screwworm larval deposition in open wounds or external orifices, such as the nares, ears, and orbits, and may be characterized by deep tissue larval invasion, with secondary infection and extensive tissue necrosis. *Cochliomyia hominivorax,* the New World screwworm, is a common cause of cavitary myiasis in the Americas, and *Chrysomyia bezziana,* the Old World screwworm, is a common cause of cavitary myiasis in Africa, Asia, and Indonesia.[11] Cavitary myiasis must be managed aggressively with surgical débridement and antibiotic therapy for secondary infections to limit tissue damage and disfigurement (see Figs. 294.1 and 294.2).[11]

Although the clinical manifestations, treatments, and prevention strategies are similar in furuncular myiasis, the mechanisms of larval fly invasion are often different. The gravid female tumbu fly deposits its eggs on moist sandy soil or on wet clothing (e.g., cloth diapers) hung outside to dry. When the human victim dons egg-infested clothing, larvae emerge and rapidly burrow into the skin with sharp mandibles for further development. On the other hand, the female botfly captures blood-feeding insects, usually mosquitoes, in midflight and attaches her eggs to the undersurface of the insect.[12] The intermediate biting vector then delivers the botfly eggs to its blood meal victims, where the eggs hatch immediately and release their larvae to feed on warm-blooded hosts. Human botfly larvae then rapidly burrow into the skin with sharp mandibles to begin their developmental instar stages, which can last 6 to 12 weeks.[12]

In addition to travel history in endemic regions, the mechanisms of larval fly invasion assist in differentiating the cause of furuncular myiasis. In tumbu fly (*C. anthropophaga)* myiasis, lesions are usually located on body regions covered by clothing, such as the buttocks and trunk. In New World human botfly *(D. hominis)* myiasis, lesions are usually located on exposed areas, such as the scalp, face, and extremities.

After completing three instar stages, the final larval forms of the tumbu fly and human botfly wriggle out of their draining, boil-like, 1- to 2-cm furuncular swellings; drop to the ground; and pupate in warm, moist soil into adult flies within 9 to 14 days. Victims may recall a flying insect bite that preceded human botfly-induced furuncular myiasis. While developing in their furuncles, larvae are active, protrude intermittently through draining wounds, and maintain surface contact for respiration with their posterior, paired spiracles.[13] Anterior hooklets anchor the maggots in place subcutaneously, making manual removal, even with forceps, difficult (see Fig. 294.3).[13]

Therapy

Management strategies for furuncular myiasis include coaxing embedded larvae from furuncles by smothering their respiratory spiracles, often visible in lesions, with occlusive coatings of petrolatum (Vaseline), clear fingernail polish, tobacco tar, pork fat, raw beefsteak, or bacon strips.[13] The injection of lidocaine into draining lesions has also been recommended as a successful extraction technique.[14] Nevertheless, unsuccessful occlusive therapy may asphyxiate larvae and necessitate their surgical or vacuum extraction.[15] Along with larval removal, myiasis wounds should be cleansed and conservatively débrided, tetanus prophylaxis administered, and bacterial secondary infections treated with antibiotics. Although *Clostridium tetani* infection of penetrating wounds does occur, tetanus has not been reported in myiasis but has been reported after ectoparasitic infestations with *T. penetrans,* the chigoe (jigger) flea, in Africa and South America.[16]

Prevention and Control

Prevention and control strategies for myiasis include the following: (1) control of domestic and livestock animal larval infestations; (2) sanitary disposal of animal carcasses and offal to deny flies their preferred breeding grounds; (3) proper management of any open human wounds or cutaneous infections; (4) cementing floors to deny floor maggot flies their preferred egg-laying surfaces; (5) sleeping on raised beds or cots in screened huts or tents; (6) wearing long-sleeved shirts and pants, which can be pyrethrin or pyrethroid impregnated; (7) spraying exposed skin with diethyl toluamide (*N,N*-diethyl-meta-toluamide [DEET])–containing repellents; and (8) ironing both sides of all clothes and diapers left outside to dry in tumbu fly habitats.

FLEA INFESTATIONS

Fleas of the insect order Siphonaptera are a small group of morphologically similar wingless ectoparasites of warm-blooded animals, including humans. They are not only biting nuisances but also competent vectors of infectious diseases, most notably *Yersinia pestis* and murine typhus (Table 294.2). Although fleas are often classified by host specificity (or presence of head combs), all fleas can rapidly adapt from animal to nearby human hosts, especially if preferred hosts are exterminated by disease or pesticides. Fleas undergo complete metamorphosis from egg to adult stages, with larvae, pupae, and adults exhibiting different morphologies and preferred habitats. Signaled by vibrations and locally rising carbon dioxide levels, adult fleas emerge from egg cases within weeks, leap onto the closest mammalian hosts, and begin blood-feeding and reproducing.

TABLE 294.2 Flea-Transmitted Infectious Diseases

INFECTIOUS DISEASE	CAUSATIVE AGENT	FLEA VECTOR (COMMON NAME)	ANIMAL RESERVOIR	MAJOR CLINICAL MANIFESTATIONS	THERAPY
Bubonic plague	*Yersinia pestis*	*Xenopsylla cheopis* (rat flea); *Oropsylla montana* (squirrel flea)	Rodents (rats, prairie dogs, squirrels); domestic animals (cats > dogs)	Headache, fever, chills, regional lymphadenopathy—draining buboes, pneumonitis (secondary plague pneumonia), septicemia, meningitis; case-fatality rate, 14%	Antibiotic therapy recommended within 24 h with any of the following effective antibiotics: tetracyclines, gentamicin, streptomycin, chloramphenicol
Murine typhus	*Rickettsia typhi*	*Xenopsylla cheopis* (rat flea); *Nosopsyllus fasciatus* (northern rat flea); *Oropsylla montana* (squirrel flea)	Rodents (rats and mice)	Fever, headache, maculopapular rash, thrombocytopenia, rarely pneumonitis and encephalitis	Doxycycline, 100 mg bid × 7–10 days
Flea-borne spotted fever	*Rickettsia felis*	*Ctenocephalides felis* (cat flea)	Rodents (rats, mice, opossums)	Nonspecific fever, headache, maculopapular rash	Tetracyclines (doxycycline)
Cat-scratch fever (disease)	*Bartonella henselae*	*Ctenocephalides felis* (cat flea)	Feral cats (kittens)	Low-grade fever, malaise, regional and rarely multifocal lymphadenopathy, endocarditis; complications more common in HIV infection and include bacillary angiomatosis, peliosis hepatis, neuroretinitis, and encephalopathy	Doxycycline and macrolides (azithromycin) effective; add rifampin for complications
Tungiasis—portal of entry for *Clostridium tetani*	Ectoparasite	*Tunga penetrans* (chigoe or jigger flea)	Humans; domestic animals (dogs > cats and pigs)	Painful white papules with central black pits discharging eggs and feces with lateral pressure, especially on dorsal aspects of toes under toenails and on heels	Surgical extraction of gravid female fleas; ivermectin ineffective in humans and only partially effective in dogs for jigger flea management
		Tunga trimamillata (only in Ecuador and Peru)		Same	Same

HIV, Human immunodeficiency virus.

TUNGIASIS

A currently reemerging, combless, ectoparasitic flea, *T. penetrans,* the chigoe or jigger sand flea, is endemic in the Caribbean and South America, where it originated, and in sub-Saharan Africa, where it was introduced. Tungiasis, a painful, cutaneous infestation with the gravid female jigger flea, is now hyperendemic in underprivileged communities in Africa, South America, and the Caribbean; has successfully reemerged in Mexico and Central America; and has been increasingly reported in travelers returning from subtropical and tropical areas worldwide.[17–23]

Epidemiology

In travelers returning to accessible health care infrastructures in developed nations, tungiasis is an exotic infestation, with a minimal parasite burden and a simple surgical cure. However, in the impoverished and underserved communities of developing tropical nations, tungiasis is a recurrent infestation with a high parasite burden, causing significant morbidity. In a descriptive study of greater than 90% of a population of a poor fishing village in Brazil, Muehlen and colleagues[20] found a 51.3% point prevalence of tungiasis, with more males (54%) infested than females, and prevalence peaks in children aged 5 to 9 years and adults aged 60 years and older. In a point prevalence study in another poor community in northeastern Brazil, Ariza and associates[21] examined 142 persons with jigger flea superinfestations and counted a total of 3445 lesions on feet (median, 17 lesions; maximum, 18 lesions). Greater than 70% of patients presented with foot pain, 59% complained of difficulty walking, 46% had toenail loss, 42% had foot abscesses, and 25% had deformed toes. In a regional prevalence study of five towns in southwestern Trinidad, Chadee[22] found the prevalence of tungiasis to range from 15.7% to 17.9%, with feet more often infected than other anatomic regions, males more likely infested than females, and higher parasite burdens in males (5.44 ± 2.54 fleas) than females (2.38 ± 2.00 fleas). In a 2007 cross-sectional study of 142 households in a rural community in western Nigeria, Ugbomoiko and associates[23] reported a 45.2% point prevalence of tungiasis, with 95% of the lesions on the feet, no gender difference in prevalence, but prevalence peaks between ages 6 and 14 years and at age 60 years and older. Ectopic lesions of the elbows and hands occurred in 10% of the population. In a study of the ectopic localization of tungiasis among 1184 residents of a poor community in northeastern Brazil with a 33.6% point prevalence of tungiasis, Heukelbach and coworkers[24] reported that 6% of all lesions presented at sites other than feet, most commonly on the hands (5.5%) but also on the buttocks, elbows, and thighs (Fig. 294.4).

FIG. 294.4 Three periungual lesions of tungiasis on the index finger of a 6-year-old girl. These were caused by tissue-feeding gravid female jigger fleas, *Tunga penetrans. (From Feldmeier H, Eisele M, Sabóia-Moura RC, et al. Severe tungiasis in underprivileged communities: case series from Brazil.* Emerg Infect Dis. *2003;9:949–955.)*

FIG. 294.5 Periungual lesion of tungiasis on the fourth toe of a 50-year-old woman. This was caused by a tissue-feeding gravid female jigger flea, *Tunga penetrans*. Note the elevation of the nail bed by the lesion. *(From Feldmeier H, Eisele M, Sabóia-Moura RC, et al. Severe tungiasis in underprivileged communities: case series from Brazil.* Emerg Infect Dis. *2003;9:949–955.)*

FIG. 294.6 The right foot of a 50-year-old man suffering from recurrent tungiasis. Note that all nails have been lost in the recurrent infestations and nonhealing wounds remain on all toes. The patient had chronic pain and could only wear slippers and walk with difficulty. *(From Feldmeier H, Eisele M, Sabóia-Moura RC, et al. Severe tungiasis in underprivileged communities: case series from Brazil.* Emerg Infect Dis. *2003;9:949–955.)*

In French Guiana and Brazil the most important zoonotic reservoir of flea-transmitted tungiasis was in domestic and stray dogs.[25,26] In a comparison of the prevalence of tungiasis in animals and humans in an impoverished Brazilian community, Pilger and colleagues[26] reported a human prevalence of 39%, a combined domestic cat and dog prevalence of 59%, and a higher prevalence (42%) of tungiasis in households with infested dogs and cats. By comparison, Sanushi and associates,[27] in a review of 14 cases of tungiasis in travelers returning to the United States, reported that patients manifested at most two lesions and complained only of local pain and itching.

Clinical Manifestations

Tungiasis is caused by the dermal penetration of the gravid female jigger (chigoe) flea to feed on blood and tissue juices, usually on the feet (or heels), under or near the toenails, or in the interdigital web spaces (Fig. 294.5).[17] Although the smallest of flea species (1 mm long or shorter), within days the gravid female swells with hundreds of developing eggs to 2000 times its size, expelling eggs over a period of 3 weeks or less, and then dying and leaving its shriveled carcass in a contaminated wound tract.[17] Initially, the embedded jigger flea produces a subcutaneous papule or vesicle 6 to 8 mm in diameter, with a central black dot pinpointing the exteriorized segments, including the anus, genital opening, and breathing spiracles (see Figs. 294.4 and 294.5). The papule darkens with intralesional hemorrhage and, if squeezed, extrudes eggs, feces, and internal organs through exteriorized posterior abdominal segments. The differential diagnosis of tungiasis includes bacterial skin infections (impetigo), bacterial and fungal paronychia, cercarial dermatitis, fire ant bites, folliculitis, and scabies. The complications of tungiasis include septicemia, abscesses, fissures, toenail (fingernail) loss, necrotic ulcers, osteomyelitis, and eventual autoamputation of toes and, less often, fingers (Fig. 294.6).[17] Tungiasis has been associated with lethal tetanus in nonvaccinated individuals and was identified as the place of entry for 10% of tetanus cases in São Paulo in a 1991 study.[16,28]

Therapy

Management strategies for tungiasis include extracting all embedded fleas immediately with sterile needles or curets, administering tetanus prophylaxis, and treating secondary wound infections with appropriate topical or oral antibiotics. Other than surgical extraction, there are no therapeutic options for tungiasis. There remains a definite need for an effective antiparasitic drug treatment option for tungiasis, especially in superinfestations.[29] In a double-blinded, randomized, placebo-controlled trial, Heukelbach and coworkers[29] have shown that a single dose of oral ivermectin, 300 µg/kg repeated at 24 hours, has no clinical efficacy compared with placebo as measured by parasite signs of viability or death.

Prevention and Control

In addition to wearing shoes, which can be sprayed with pyrethroid or DEET-containing solutions, and not sitting naked on bare ground, preventive strategies for tungiasis include the following: (1) insecticide treatment of flea-infested domestic and stray animals and pets with 10% pyrethrin or pyrethroid sprays or 1% to 4% malathion powder; (2) bathing the feet of domestic and stray dogs and pigs with insecticide solutions, such as 2% trichlorfon; and (3) spraying or dusting households, especially those with dirt floors, with 1% to 4% malathion. Other strategies for the environmental control of jigger fleas include improved stray animal control, especially for cats and dogs; providing cement foundation or slab flooring for dirt-floored homes or building raised homes with solid floors; discouraging stray dogs and cats and other domestic animals, especially pigs, as indoor pets; and spraying rodent and stray animal runways and paths, household unpaved walkways, and dirt floors with solutions containing kerosene, fuel oil, 1% lindane, 1% to 4% malathion, or 2% trichlorfon.

Key References

The complete reference list is available online at Expert Consult.

1. Marquez AT, Mattos Mda S, Nascimento SB. Myiasis associated with some socioeconomic factors in five urban areas of the State of Rio de Janeiro. *Rev Soc Bras Med Trop*. 2007;40:175–180, [in Portuguese].
4. Clyti E, Deligny C, Nacher M, et al. Short report: an urban epidemic of human myiasis caused by *Dermatobia hominis* in French Guiana. *Am J Trop Med Hyg*. 2008;79:797–798.
7. Plotinsky RN, Talbot EA, Davis H. Short report: *Cuterebra* cutaneous myiasis, New Hampshire, 2004. *Am J Trop Med Hyg*. 2007;76:596–597.
17. Feldmeier H, Eisele M, Sabóia-Moura RC, et al. Severe tungiasis in underprivileged communities: case series from Brazil. *Emerg Infect Dis*. 2003;9:949–955.
19. Heukelbach J, Gomide M, Araújo F Jr, et al. Cutaneous larva migrans and tungiasis in international travelers exiting Brazil: an airport survey. *J Travel Med*. 2007;14:374–380.
21. Ariza L, Seidenschwang M, Buckendahl J, et al. Tungiasis: a neglected disease causing severe morbidity in a shantytown in Fortaleza, State of Ceara. *Rev Soc Bras Med Trop*. 2007;40:63–67.
26. Pilger D, Schwalfenberg S, Heukelbach J, et al. Investigations on the biology, epidemiology, pathology and control of *Tunga penetrans* in Brazil, II. The importance of animal reservoirs for human infestation. *Parasitol Res*. 2008;102:875–880.
29. Heukelbach J, Franck S, Feldmeier H. Therapy of tungiasis: a double-blinded randomized controlled trial with oral ivermectin. *Mem Inst Oswaldo Cruz*. 2004;99:873–876.

References

1. Marquez AT, Mattos Mda S, Nascimento SB. Myiasis associated with some socioeconomic factors in five urban areas of the State of Rio de Janeiro. *Rev Soc Bras Med Trop*. 2007;40:175–180, [in Portuguese].
2. Jiang C. A collective analysis of 54 cases of human myiasis in China from 1995-2001. *Chin Med J*. 2002;115:1445–1447.
3. Schwartz E, Gur H. *Dermatobia hominis* myiasis: an emerging disease among travelers to the Amazon Basin of Bolivia. *J Travel Med*. 2002;9:97–99.
4. Clyti E, Deligny C, Nacher M, et al. Short report: an urban epidemic of human myiasis caused by *Dermatobia hominis* in French Guiana. *Am J Trop Med Hyg*. 2008;79:797–798.
5. Tamir J, Haik J, Schwartz E. Myiasis with Lund's fly *(Cordylobia rodhaini)* in travelers. *J Travel Med*. 2003;10: 293–295.
6. Shorter N, Werninghaus K, Mooney D, et al. Furuncular cuterebrid myiasis. *J Pediatr Surg*. 1997;32:1511–1513.
7. Plotinsky RN, Talbot EA, Davis H. Short report: *Cuterebra* cutaneous myiasis, New Hampshire, 2004. *Am J Trop Med Hyg*. 2007;76:596–597.
8. Lachish T, Marhoom E, Mumcuoglu KY, et al. Myiasis in travelers. *J Travel Med*. 2015;22:232–236.
9. Belaz S, Gay E, Beaucournu JC, et al. Tungiasis outbreak in travelers from Madagascar. *J Travel Med*. 2015;22:263–266.
10. Monsel G, Caumes E. What's new in travel-associated dermatology? Editorial. *J Travel Med*. 2015;22:221–224.
11. Seppannen M, Virolainen-Julkunen A, Kakko I, et al. Myiasis during adventure sports race. *Emerg Infect Dis*. 2004;10:137–139.
12. Sloop GD, Lopez FA. Furuncular myiasis. *J la State Med Soc*. 2006;158:14–16.
13. Liebert PS, Madden RC. Human botfly larva in a child's scalp. *J Pediatr Surg*. 2004;39:629–630.
14. Lui H, Buck HW. Cutaneous myiasis: a simple and effective technique for extraction of *Dermatobia hominis* larvae. *Int J Dermatol*. 1992;31:657–679.
15. Boggild AK, Keystone JS, Kain KC. Furuncular myiasis: a simple and rapid method for extraction of intact *Dermatobia hominis* larvae. *Clin Infect Dis*. 2002;35:336–338.
16. Obengui P. Tungiasis and tetanus at the University Hospital Center in Brazzaville. *Dakar Med*. 1989;34:44–48, [in French].
17. Feldmeier H, Eisele M, Sabóia-Moura RC, et al. Severe tungiasis in underprivileged communities: case series from Brazil. *Emerg Infect Dis*. 2003;9:949–955.
18. Ibanez-Bernal S, Velasco-Castrejon O. New records of tungiasis in Mexico (Siphonaptera: Tungidae). *J Med Entomol*. 1996;33:988–989.
19. Heukelbach J, Gomide M, Araújo F Jr, et al. Cutaneous larva migrans and tungiasis in international travelers exiting Brazil: an airport survey. *J Travel Med*. 2007;14: 374–380.
20. Muehlen M, Heukelbach J, Wilcke T, et al. Investigations on the biology, epidemiology, pathology and control of *Tunga penetrans* in Brazil, II. Prevalence, parasite load and topographic distribution of lesions in a population of a traditional fishing village. *Parasitol Res*. 2003;90:449–453.
21. Ariza L, Seidenschwang M, Buckendahl J, et al. Tungiasis: a neglected disease causing severe morbidity in a shantytown in Fortaleza, State of Ceara. *Rev Soc Bras Med Trop*. 2007;40:63–67.
22. Chadee DD. Tungiasis among five communities in south-western Trinidad, West Indies. *Ann Trop Med Parasitol*. 1998;92:107–113.
23. Ugbomoiko US, Ofoezie IE, Heukelbach J. Tungiasis: high prevalence, parasite load, and morbidity in a rural community in Lagos State, Nigeria. *Int J Dermatol*. 2007;46:475–481.
24. Heukelbach J, Wilcke T, Eisele M, et al. Ectopic localization of tungiasis. *Am J Trop Med Hyg*. 2002;67: 214–216.
25. Rietschel W. Observations of the sand flea *(Tunga penetrans)* in humans and dogs in French Guiana. *Tierarztl Prax*. 1989;17:189–193, [in German].
26. Pilger D, Schwalfenberg S, Heukelbach J, et al. Investigations on the biology, epidemiology, pathology and control of *Tunga penetrans* in Brazil, II. The importance of animal reservoirs for human infestation. *Parasitol Res*. 2008;102:875–880.
27. Sanushi ID, Brown EB, Shepard TG, et al. Tungiasis: report of one case and review of the 14 reported cases in the United States. *J Am Acad Dermatol*. 1989;20:941–944.
28. Litvoc J, Leite RM, Katz G. Epidemiology of tetanus in São Paulo State (Brazil). *Rev Inst Med Trop São Paulo*. 1991;33:447–484, [in Portuguese].
29. Heukelbach J, Franck S, Feldmeier H. Therapy of tungiasis: a double-blinded randomized controlled trial with oral ivermectin. *Mem Inst Oswaldo Cruz*. 2004;99:873–876.

295 Mites, Including Chiggers

James H. Diaz

SHORT VIEW SUMMARY

Definition
- Mites are among the smallest arthropods, with more than 3000 species, only about 25 of which, mostly chigger, animal, plant, and scabies mites, are of any medical importance.

Epidemiology
- Most mites are simply biting nuisances that can cause highly symptomatic maculopapular eruptions and do not transmit infectious diseases.

Microbiology
- Only biting larvae of Asian scrub typhus chiggers (*Leptotrombidium* spp.) can transmit scrub typhus caused by *Orientia tsutsugamushi* (formerly *Rickettsia tsutsugamushi*), and only biting house mouse mites *(Liponyssoides sanguineus)* can transmit rickettsialpox caused by *Rickettsia akari.*

Diagnosis
- Clinical diagnoses are highly sensitive and specific in the presence of a mite-bite outbreak.
- Mite-transmitted scrub typhus and rickettsialpox manifest clinically in a similar fashion with fever, bite eschar, regional lymphadenopathy, conjunctival injection, central nervous system symptoms in severe scrub typhus (e.g., confusion, delirium, coma, or transient hearing loss), and centrifugal rash in scrub typhus (see Chapter 193).

Therapy
- Treatment of chigger bites is supportive with soap and water cleansing, warm water soaks, and topical and local anesthetics and antihistamines. Impetigo and secondary infections are potential complications that would necessitate antibiotic treatment.
- Both scrub typhus and rickettsialpox respond to treatment with oral doxycycline.

Prevention
- Chigger bites can be prevented with campsite spraying of pyrethrin or pyrethroid-containing insecticides; by spraying or impregnating pyrethrin and pyrethroid-containing repellents on clothing and sleeping bags; and by applying diethyltoluamide–containing insect repellents (*N,N*-diethyl-meta-toluamide [DEET]) to exposed skin.
- Rickettsialpox can be prevented by improving rodent reservoir control in campgrounds, homes, apartments, barns, sheds, and, especially, crowded public housing.
- There are no vaccines for scrub typhus or rickettsialpox.
- Weekly doses of 200 mg of doxycycline can prevent *O. tsutsugamushi* infections in endemic regions.

Mites, including chigger and scabies mites, are among the smallest arthropods, with most barely visible without magnification. Only about 20 species of the more than 3000 species of chigger, animal, plant, and scabies mites are of any medical importance, and most of these are simply biting nuisances and do not transmit infectious diseases.[1] Mites are closely related to ticks but not as prodigious at blood-feeding. They also do not transmit as broad a range of infectious microbial diseases as ticks. The most serious diseases transmitted by mites are scrub typhus and rickettsialpox.

Only biting larvae of Asian scrub typhus chiggers (*Leptotrombidium* spp.) can transmit scrub typhus caused by *Orientia tsutsugamushi* (formerly *Rickettsia tsutsugamushi*), and only biting house mouse mites *(Liponyssoides sanguineus)* can transmit rickettsialpox caused by *Rickettsia akari*. Both scrub typhus mites and house mouse mites are, like ticks, capable of inheriting bacterial infections by transovarial transmission and maintaining infections in several mite generations as bacteria are passed from adult to juvenile stages (nymphs and larvae) by transstadial transmission. Originally considered vectors of a rodent zoonosis, scrub typhus chiggers are the main environmental reservoirs of *O. tsutsugamushi* in endemic regions, with much smaller secondary reservoirs in wild rodents.[1] Common house mice are the zoonotic reservoirs of *R. akari,* not only in crowded urban apartment buildings in the United States but also in mice-infested buildings, such as sheds and barns, in more rural locations worldwide.[2]

MITE TAXONOMY AND ECOLOGY

Mites may be commonly classified as scabies mites (see Chapter 293), trombiculid or chigger mites (also called chiggers, red bugs, and itch mites), human follicle mites, dust mites, and a variety of animal, plant, and wood mites (Table 295.1). All mite species develop close generational associations with their ecosystems and zoonotic reservoirs, often referred to as mite islands.[1] Mite islands usually border cleared land and scrub bush and have several habitat requirements, including grassy vegetation with warm soil temperatures and high humidity, frequently visiting rodent hosts to feed larvae, and sufficient small insect fauna to feed nymphs and reproducing adults. Humans stumbling onto mite islands are at significantly higher risk for multiple larval chigger bites or trombidiosis worldwide or scrub typhus in the endemic regions of Eurasia and Asia.

EPIDEMIOLOGY AND OUTCOMES OF MITE INFESTATIONS

Among the trombiculid chiggers (the Trombiculidae), including the scrub typhus–transmitting *Leptotrombidium* spp., only the larvae are human and animal ectoparasites. The larger chigger nymphs and adults are free living and feed on small insects and their eggs. All trombiculid larvae exhibit a unique method of feeding on their human hosts and transmitting salivary secretions, which may contain *O. tsutsugamushi* in endemic regions. When larval mites have selected a human host, they will congregate where the skin is soft, warm, and moist, particularly where clothing is tight against the skin, such as under waistbands, undergarment elastic bands, and socks. Initially painless, chigger bites cluster in these regions on the genitalia, perineum, thighs, buttocks, waist, and ankles and become symptomatic in 3 to 6 hours (Fig. 295.1). Larvae pierce the skin with sharp mouthparts and inject tissue-dissolving saliva to create a pool of lymph, other body fluids, and dissolved epithelial cells to drink from (see Fig. 295.1). Unlike ticks, mites are not blood feeders, but tissue juice feeders. The repeated injection of saliva into the bite wound induces a host reaction that forms a strawlike hollow tube, known as a hypostome or stylostome, which extends downward into the host's skin, anchoring the mite firmly.[1,3] Some trombiculid larvae remain attached to and feeding on human hosts for up to a month, but the larval vectors of scrub typhus feed only for 2 to 10 days before dropping to the ground engorged and ready to mature into free-ranging nymphs.

TABLE 295.1 Mites of Medical Importance

FAMILY, GENUS, SPECIES	COMMON NAMES (PLANT OR ANIMAL MITE)	GEOGRAPHIC DISTRIBUTION	MAINTENANCE IN NATURE	CLINICAL MANIFESTATIONS	INFECTIOUS DISEASE TRANSMISSION
Sarcoptidae					
Sarcoptes scabiei var. *hominis*	Scabies (itch) mite (human mite)	Worldwide	Obligate ectoparasite of humans, human reservoir	Classic scabies Atypical scabies	None
Trombiculidae					
Neotrombicula autumnalis	European harvest mite (animal mite)	Europe	Free-living ectoparasites of small mammals and birds	Scrub itch (trombidiosis)	None
Eutrombicula alfreddugesi	American chigger mite (animal mite)	Western Hemisphere	Free-living ectoparasites of small mammals and birds	Scrub itch (trombidiosis)	None
Eutrombicula sarcina	Asian chigger mite (animal mite)	Asia, Australia	Free-living ectoparasites of small mammals and birds	Scrub itch (trombidiosis)	None
Leptotrombidium deliense	Asian rodent chigger (animal mite)	Southeast Asia, Japan, Philippines, South Pacific, Australia	Free-living ectoparasites of rodents and insectivores, transovarial/transstadial passage of infectious disease agent	Scrub typhus (tsutsugamushi disease)	Orientia tsutsugamushi (formerly Rickettsia tsutsugamushi), causative agent of scrub typhus
Leptotrombidium akamushi, *Leptotrombidium pallidum*, and *Leptotrombidium scutellaris*	Japanese rodent chiggers (animal mites)	Japan	Same	Same	Same
Leptotrombidium arenicola and *Leptotrombidium fletcheri*	Malaysian rodent chiggers (animal mites)	Malaysia	Same	Same	Same
Leptotrombidium pavlovskyi	Russian rodent chigger (animal mite)	Far east of former Soviet Union	Same	Same	Same
Demodicidae					
Demodex folliculorum	Hair follicle mite	Worldwide	Obligate ectoparasite of man, human host reservoir in hair follicles	Benign follicular (scaling) dermatitis Chronic blepharitis (demodicidosis)	None
Demodex brevis	Sebaceous gland mite	Worldwide	Obligate ectoparasite of man, human host reservoir in sebaceous glands	May potentiate granulomatous acne	None
Pyroglyphidae					
Dermatophagoides pteronyssinus	European house dust mite (human mite)	Worldwide	Free-living ectoparasites of man; live in human bedrooms, especially in mattresses; feed on human skin detritus	House dust mite allergies and asthma	None
Dermatophagoides farinae	American house dust mite (human mite)	Worldwide	Same	Same	None
Dermanyssidae					
Liponyssoides sanguineus (formerly *Allodermanyssus sanguineus*)	House mouse mite	North America, Northern Europe, Asia, and Africa	Free-living ectoparasites of field mice, transovarial/transstadial passage of infectious disease agent	Rickettsialpox	Yes (Rickettsia akari)
Dermanyssus gallinae	Red poultry (chicken) mite	Worldwide	Free-living ectoparasites of domestic and wild birds	Poultry workers' dermatitis of hands	None
Macronyssidae					
Ornithonyssus bacoti	Tropical rat mite	Temperate and tropical regions worldwide	Free-living ectoparasites of large rodents: *Rattus rattus, Rattus norvegicus*	Urticarial papulovesicular to pustular dermatitis	None
Ornithonyssus bursa	Tropical fowl mite	Same	Free-living ectoparasites of domestic and wild birds	Pruritic papules in a scabietic distribution: finger webs, axillae, groin, buttocks	None
Laelapidae					
Laelaps echidnina	Spiny rat mite	Worldwide, the most prevalent rodent mite species in the United States	Free-living ectoparasites of large rodents: *Rattus rattus, Rattus norvegicus*	Nonspecific mite-bite dermatitis	None

TABLE 295.1 Mites of Medical Importance—cont'd

FAMILY, GENUS, SPECIES	COMMON NAMES (PLANT OR ANIMAL MITE)	GEOGRAPHIC DISTRIBUTION	MAINTENANCE IN NATURE	CLINICAL MANIFESTATIONS	INFECTIOUS DISEASE TRANSMISSION
Pyemotidae					
Pyemotes tritici ventricosus	Grain (hay) itch mite	Worldwide	Free-living ectoparasites of straw-, hay-, grain-, and rice-eating moths, beetles, weevils	Grain workers' pruritic vesicular eruption	None
Pediculoides ventricosus Newport	European wood beetle itch mite	Worldwide	Free-living ectoparasites of straw-, hay-, grain-, and rice-eating moths, beetles, weevils	Solitary to multiple highly erythematous pruritic macules, some of which have attached macular tracts resembling comet tails	None
Pyemotes herfsi	Oak leaf gall mite	Europe, introduced into the United States	Free-living ectoparasites of gall-making larvae of oak trees	Pruritic, erythematous, vesicular eruptions of limbs, face, and neck	None
Acaridae[a]					
Carpoglyphus lactis	Cheese and dried fruit mites	Worldwide	Free-living ectoparasites of cheeses and dried fruits	Cheese and fruit workers' dermatitis	None
Tyrophagus putrescentiae	Copra (dried coconut meat or kernel) mite	Copra (dried coconut meat or kernel) growing areas	Free-living ectoparasites of coconut copra	Copra itch	None
Glycyphagidae[a]					
Glycyphagus domesticus	Grocer's mite	Worldwide	Free-living ectoparasites of fruits and vegetables	Grocer's itch	None
Glycyphagus destructor	Hay mite	Worldwide	Free-living ectoparasites of cut hay	Hay workers' and hay wagon riders' allergy, asthma, rhinitis, conjunctivitis	None

[a]The acarid and glycyphagid plant mites may rarely enter the gastrointestinal tract if swallowed with food and cause intestinal distress (gastrointestinal acariasis). They may also be inhaled in aerosols and cause bronchial irritation and respiratory distress (respiratory acariasis). They may crawl into the urethra to cause dysuria (urinary acariasis). The mites can be recovered from feces, sputum, and urine. Treatment is supportive. Although they may rarely infest humans, plant mites are all free living and do not reproduce in human dead-end hosts.

FIG. 295.1 An intensely pruritic red bleb 24 hours after a bite inflicted by a larval trombiculid species chigger mite. Although these species of lymph-sucking mites do not transmit infectious diseases in the United States, many species of larval trombiculids transmit scrub typhus or tsutsugamushi disease, caused by the rickettsial microorganism *Orientia* (formerly *Rickettsia*) *tsutsugamushi,* throughout Southeast Asia and the western Pacific region. *(From Centers for Disease Control and Prevention [CDC], Atlanta, GA. CDC Public Health Image Library, image 3806.)*

All of the noninfectious chigger larvae can cause trombidiosis or trombiculiasis (trombiculidiasis), with the American chigger mite *(Eutrombicula alfreddugesi)* being the most common culprit in the United States; the European harvest mite, *Neotrombicula autumnalis,* the most common culprit in Europe; and the Asian chigger, *Eutrombicula sarcina,* the most common culprit in Asia. In the United States, clusters of trombiculid larval bites are often referred to as "chiggers," a colloquial term for the intensely pruritic groupings of erythematous welts inflicted by American chigger mites. In Asia and Australia, similar clusters of larval mite bites may be referred to as "scrub itch," especially in Australia, where scrub itch mites, *Trombicula hirsti,* commonly cause trombidiosis. Among the scrub typhus–carrying *Leptotrombidium* larval chigger mites, *Leptotrombidium deliense,* the Asian rodent chigger, is a principal vector throughout eastern Asia and Eurasia.

Unlike North American "chiggers," scrub typhus is a serious and potentially fatal zoonotic infectious disease caused by *O. tsutsugamushi* organisms transmitted by bites from infected *Leptotrombidium* species chigger larvae. As a result of experiences with scrub typhus among US troops in the Pacific during World War II and later during wars in Korea, Vietnam, and Afghanistan, scrub typhus was originally considered a regional zoonosis confined to the "tsutsugamushi triangle," which extended from Pakistan in the west to the Pacific coast of Russia in the east to northern Australia in the south.[4] In 2006, additional cases were detected in the Middle East caused by a new species, *Orientia chuto*, and in Chile caused by *O. tsutsugamushi.*[4] In 2016, three additional cases of scrub typhus caused by *O. tsutsugamushi* were confirmed serologically (with enzyme-linked immunosorbent assay [ELISA] and indirect immunofluorescent antibody assay [IFA]) and molecularly (with polymerase chain reaction [PCR] detection of DNA in the 47-kD and 56-kD genes) in Chile, which is 12,000 km away from the tsutsugamushi triangle.[4] Although the zoonotic reservoirs and larval mite vectors of scrub typhus outside of the tsutsugamushi triangle remain unidentified, there is now a wider global distribution of the disease than initially assumed.[4]

After *O. tsutsugamushi*–infected *Leptotrombidium* chigger bites, an 8- to 10-day incubation period precedes the onset of classic clinical manifestations of scrub typhus with bite eschar, regional lymphadenopathy, conjunctival injection, central nervous system (CNS) manifestations (e.g., headache, confusion, delirium, transient hearing loss), and centrifugal rash (Fig. 295.2).[5] Many cases are nonclassic with nonspecific

clinical manifestations and go undiagnosed, especially when serologic tests are unavailable. In the temperate regions of Eurasia, there is a definite scrub typhus seasonal transmission cycle determined by peaking temperatures and humidity during weeks of marked seasonal change in late spring to early summer and again in late fall to early winter. In the tropics, scrub typhus transmission occurs year-round. Fatal complications may include adult respiratory distress syndrome (especially in older patients), hypotensive shock, acute renal failure, encephalomyelitis, and disseminated intravascular coagulation.[5]

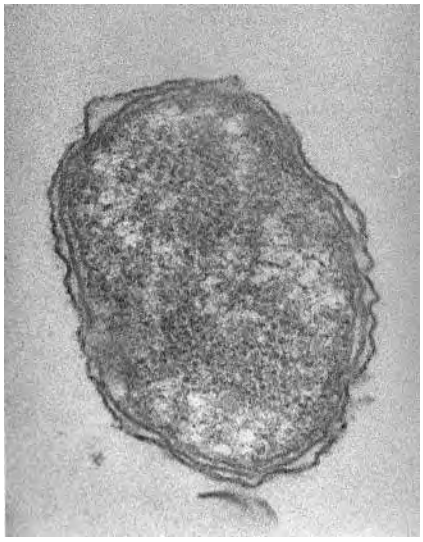


FIG. 295.2 A transmission electron micrograph of an extracellular *Orientia tsutsugamushi* rickettsial microorganism, the causative agent of scrub typhus or tsutsugamushi disease throughout Southeast Asia and the Western Pacific region. During World War II, scrub typhus was second only to malaria as a cause of hospitalization among US troops in the Pacific theater of operations.[1] *(From Centers for Disease Control and Prevention [CDC], Atlanta, GA. CDC Public Health Image Library, image 8730.)*

Although the most common CNS manifestations of scrub typhus are meningitis or meningoencephalitis, Yun and coauthors recently reported a case of acute transverse myelitis in a 67-year-old man with dysuria and lower-leg weakness associated with serologically confirmed scrub typhus in Korea and successfully treated with doxycycline and corticosteroids.[5]

In 2016, Thipmontree and coauthors reported another very rare complication of scrub typhus in a 74-year-old Thai woman with acute abdominal symptoms requiring surgery, due to a ruptured spleen.[6] In this case, scrub typhus was confirmed by a fourfold increase in immunoglobulin M (IgM) titer by ELISA and positive PCR targeting the 47- and 56-kD genes of *O. tsutsugamushi*.[6] The patient underwent splenectomy and was treated successfully with intravenous chloramphenicol and oral doxycycline over 10 days.[6]

The house mouse mite, *Liponyssoides sanguineus,* maintains a rickettsial zoonosis in its preferred house mouse *(Mus musculus)* reservoir and can transmit rickettsialpox caused by *R. akari* through its bites.[1,3] Although initially described in clusters in crowded apartment complexes in large US cities, including New York, Boston, Cleveland, Philadelphia, and Pittsburgh, rickettsialpox has now been reported in rural areas of the eastern United States and eastern Europe Many experts believe that rickettsialpox is underreported and more widely distributed in silent sylvan cycles worldwide. The incubation period and initial clinical manifestations of rickettsialpox mirror those of scrub typhus, with eschar formation at the bite site within 10 to 12 days followed by fever, chills, severe headache, conjunctival injection, and truncal maculopapular then vesicular rash.[2,7,8] Illness is typically mild, and regional lymphadenopathy is uncommon (Table 295.2). Unlike with scrub typhus, complications are rare, but they may include thrombocytopenia and interstitial pneumonia.[2,8]

TABLE 295.2 Presentation, Diagnosis, Differential Diagnosis, and Management of Mite-Transmitted Scrub Typhus Versus Rickettsialpox

	SCRUB TYPHUS	RICKETTSIALPOX
Bacterial agent	*Orientia* (formerly *Rickettsia*) *tsutsugamushi*[a]	*Rickettsia akari*
Mite vector	Larvae of *Leptotrombidium* species of Asian rodent chigger mites	Common house mouse mites, *Liponyssoides sanguineus*
Incubation period	8–10 days (range, 8–20 days)	10–12 days
Mite-bite site	Painless initial bite, eschar at bite site (50%)	Painless initial bite, eschar at bite site
Presenting constitutional symptoms	Fever, chills, headaches, myalgia, pathognomonic hearing loss (30%)	Fever, chills, severe headache, myalgia
Conjunctival injection	Present	May be present
Regional lymphadenopathy	Present regionally and tender	Usually absent
Associated rash exanthema	Delayed truncal onset, erythematous macules, then maculopapules that spread peripherally	Abrupt truncal onset, erythematous macules that develop central vesicles in crops
Pulmonary findings	Cough, tachypnea, dyspnea, bibasilar rales	Asymptomatic bibasilar rales
Radiographic findings	Infiltrates common	Usually normal
Potential complications	Adult respiratory distress syndromes, acute renal failure, disseminated intravascular coagulation, encephalomyelitis	Thrombocytopenia
Differential diagnosis	Infectious mononucleosis, leptospirosis, tularemia, anthrax, spotted fever rickettsioses	Chickenpox, tick-bite eschar
Diagnostic methods	*Screening:* rapid dipstick recombinant 56-kDa protein antigen test *Serodiagnostic:* indirect immunofluorescent antibody tests, immunoperoxidase assays *Confirmatory:* microscopic isolation of agent from blood or tissues, polymerase chain reaction for agent DNA (or RNA)	*Serodiagnostic:* immunofluorescent antibody assay for IgG to both *R. akari* and *R. rickettsii,* with follow-up cross-adsorption testing for predominant antibodies *Confirmatory:* isolation from skin biopsy specimen
Recommended treatment	Tetracycline 500 mg orally 4 × daily × 1 wk, or doxycycline 100 mg twice daily × 1 wk, or IV chloramphenicol, 50–75 mg/kg/day × 1 wk (only for complicated cases) In childhood and pregnancy, consider the macrolides: azithromycin, clarithromycin, or roxithromycin	Doxycycline 100 mg orally twice daily × 7–10 days
Outcomes/case-fatality rates (%)	1%–15%	<1%

[a]Although initially classified among the *Rickettsia,* the causative agent of scrub typhus, *Orientia tsutsugamushi* (originally *Rickettsia tsutsugamushi*) has now been reclassified into a separate genus, *Orientia,* based on molecular evidence that its cell wall differs significantly from that of *Rickettsia* both ultrastructurally and in its component proteins.[4]

Dermatophagoides spp. dust mites have highly allergenic exoskeletons, body fragments, and feces, all of which can be easily aerosolized during bed making and pillow fluffing. Allergens from living and dead dust mites frequently cause allergic rhinitis and asthmatic bronchitis in predisposed, atopic persons. The American house dust mite, *Dermatophagoides farinae,* is now distributed worldwide, as is the European house dust mite, *Dermatophagoides pteronyssinus.*[3] House dust mites prefer to live in bedrooms year-round, especially in mattresses and carpets in warm, humid homes. They exhibit maximum growth and reproduction during seasonal warming cycles at ambient temperatures at or above 25°C and relative humidity at or above 75%.[3]

Scabies and follicle mites are the only exclusively human ectoparasitic mites and cannot transmit infectious diseases. Less serious but more common than scabies is infection with the human follicle mites: *Demodex folliculorum* inhabits hair follicles, and *Demodex brevis* inhabits sebaceous glands. These diminutive (0.1–0.4 mm) human mites feed on sebum and exfoliated skin while lodged in human hair follicles or sebaceous gland pores.

Demodex mites or face mites are commensal ectoparasites that cluster in hair follicles and sebaceous glands on the nose, eyelids, and nasolabial folds and have even been found living in earwax *(D. folliculorum).* All of the developmental stages of *Demodex* mites occur over an egg-to-egg cycle of 13 to 15 days entirely within hair follicles or sebaceous glands, especially in females overusing cream-based facial cosmetics and adolescents with increased sebaceous gland activity. Other than causing comedones or "blackheads," *Demodex* infections cause few adverse symptoms and rarely need treatment, unless infections are associated with acne, blepharitis, impetigo, rosacea, or seborrheic dermatitis.[1,3] In some cases, infection or demodicosis may be precipitated by immunosuppression from stress, advancing age, or coexisting illness.

Although of limited clinical significance, a number of animal, plant, and wood mite species can cause bothersome erythematous papulovesicular eruptions if encountered. Bites from the red chicken or poultry mite, *Dermanyssus gallinae,* can cause a pruritic dermatitis, usually on the backs of the hands and forearms in poultry workers (Fig. 295.3).[9] Both St. Louis encephalitis virus and western equine encephalitis virus have been isolated from naturally infected red chicken mites, but they are not preferred vectors for these mosquito-borne arboviruses (see Fig. 295.3).[9] Bites from the rat mite *Ornithonyssus bacoti,* which is ubiquitous in the temperate areas of Europe and the Americas, can cause a papulovesicular dermatitis in stockyard and warehouse workers.[9] The rat mite can also transmit *Rickettsia typhi,* the agent of murine typhus from rat to rat, maintaining the rodent zoonosis, but is incapable of human transmission.[9] The bird mite, *Ornithonyssus bursa,* is a common ectoparasite of pigeons worldwide and a frequent cause of mite infections with maculopapular dermatitis of the finger webs and axillae in pigeon breeders and fanciers.[10]

Like chigger mites, the plant and wood mites do not transmit infectious diseases but are common causes of annoying infections with pruritic, erythematous maculopapular rashes on the limbs and face of arborists, landscapers, and campers.

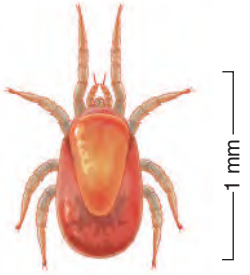
The North American hay itch mite, *Pyemotes tritici ventricosus,* feeds preferentially on the larvae of insects that infest cane, hay, straw, and some grains, especially rice (Fig. 295.4).[11] In 1965, Fine and Scott[11] were the first investigators to describe *P. tritici ventricosus* dermatitis in the southern United States, where hayrides, cane furniture, and straw rugs are popular.[11] Such exposures frequently place patients in skin contact with infested hay, straw, or cane furniture during peak mite feeding and breeding seasons in the spring and summer.[11] Hay itch mite dermatitis is characterized by pruritic, maculopapulovesicular eruptions on the limbs and trunk, which resolve rapidly with topical corticosteroid therapy.[11]

A close relative of the North American hay itch mite, *P. tritici ventricosus,* the oak leaf gall mite *(Pyemotes herfsi),* which preferentially feeds on insect larvae in oak trees, caused an outbreak of plant insect mite dermatitis in the United States in 2004.[12] Over 300 residents of Pittsburg, Kansas, sought immediate medical attention for an intensely pruritic, erythematous maculopapular rash clustering on the face, neck, and limbs (Fig. 295.5).[12] All lesions healed within days after topical treatment with antihistamines and corticosteroids.[12]

Three separate outbreaks of dermatitis affecting over 100 patients caused by the European hay itch mite *(Pyemotes ventricosus)* were reported from Spain in 2000.[13] A similar outbreak, also suggestive of mite bite–induced dermatitis, was reported in southeastern France in 2006.[14] The dermatitis was characterized by solitary to multiple, highly erythematous pruritic macules; some were accompanied by contiguous, linear erythematous macular tracts that resembled comet tails (Fig. 295.6).[14] In a 2007 outbreak investigation of an additional 42 cases of dermatitis with comet tail signs in the same region, Del Giudice and coworkers[15] identified *P. ventricosus* mites as causative agents and described the epidemiology and outcomes of *P. ventricosus* infections in homes and humans.

Most residences of index case patients with *P. ventricosus* dermatitis were infested with live furniture beetles, *Anobium punctatum,* which do not bite or infest humans.[15] However, adult *P. ventricosus* mites, common ectoparasites of furniture beetles, were present in stereomicroscopic examination of wood dust beneath beetle-infested furniture.[15] Confocal laser scanning microscopy (CLSM) of a central microvesicle in a maculopapular lesion on an experimentally infested coinvestigator demonstrated an ovoid foreign body consistent with a *P. ventricosus* mite.[15] Both naturally occurring and experimental infections caused the characteristic maculopapular rash of *P. ventricosus* dermatitis, again associated with comet signs (see Fig. 295.6).[15] Although oral prednisone

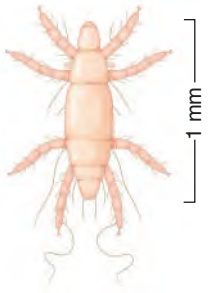
ACARINA > DERMANYSSIDAE



Dermanyssus gallinae (DeGeer)
(female, dorsal aspect)

FIG. 295.3 An illustration of the dorsal aspect of the female red chicken (poultry) mite, *Dermanyssus gallinae,* which is a common cause of poultry workers' and pigeon breeders' dermatitis worldwide. *(From Centers for Disease Control and Prevention [CDC], Atlanta, GA. CDC Public Health Image Library, image 5482.)*

ACARINA > PEDICULOIDIDAE



Pyemotes ventricosus
(Newport)
(female, dorsal aspect)

FIG. 295.4 An illustration of the dorsal aspect of the female grain (hay) itch mite, *Pyemotes tritici ventricosus* (formerly *Pyemotes ventricosus*), which is a common cause of highly pruritic and papulovesicular rashes among hay threshers, hay wagon riders, outdoor yard workers, and campers during late summers worldwide. A similar plant mite, *Pyemotes herfsi,* the European oak leaf gall mite, caused an outbreak of pruritic, erythematous papulovesicular rashes, inducing 300 residents of Pittsburg, Kansas, to seek medical care in late August 2004.[8] *(From Centers for Disease Control and Prevention [CDC], Atlanta, GA. CDC Public Health Image Library, image 5482.)*

(0.5 mg/kg) rapidly relieved pruritus, *P. ventricosus* dermatitis would persist or recur in index case patients until beetle-infested furniture was removed from households or patients permanently vacated their infested residences, which were often in resort regions.[15]

DIAGNOSIS AND MANAGEMENT OF MITE INFECTIONS

All larval chigger bites cluster where clothing is tight against the skin, especially the genitalia, thighs, buttocks, waist, and ankles, and generally do not create itching and discomfort until the larvae have withdrawn their mouthparts and departed. Forcibly removing feeding chiggers often decapitates larvae, leaving mouthparts embedded to cause further inflammation.[1] Untested strategies for removing feeding, engorged chiggers intact have included painting chigger bite sites with collodion, clear fingernail polish, or Liquid Skin and then drying the sites with a hair dryer and peeling the coated and dried chiggers off the skin intact. Intense itching commences within 3 to 6 hours after bites, which is then followed by intensely pruritic, erythematous papules (10–12 hours) and crusting and healing (24–48 hours; see Fig. 295.1).[1,3]

Several noninvasive techniques, such as surface microscopy, dermoscopy or dermatoscopy, epiluminescence microscopy, and videodermatoscopy, can provide rapid high-power magnification (10× to 1000×) and in vivo observation of the skin, and these techniques have been used to differentially diagnose several chronic ectoparasitic disorders, including scabies, pediculosis, phthiriasis, tungiasis, and myiasis.[16] In 2014, Italian investigators used videodermatoscopy to diagnose a case of chronic, intensely pruritic trombiculosis caused by the larval European harvest or autumn mite, *N. autumnalis*, in a man with multiple self-induced excoriations on the extremities previously treated as scabies (Fig. 295.7).[16]

Treatment is supportive with soap and water cleansing, warm water soaks, and topical local anesthetics and antihistamines. Impetigo and secondary infections are potential complications that would necessitate antibiotic treatment.

Follicle mite infections on the face usually require no treatment other than soap and water washing to reduce infections.[1,3,9] Scalp and eyelash infections respond to washes with 0.5% selenium or 10% sulfur-containing creams, lotions, or shampoos, with care to avoid ocular instillation. Chronic follicle mite infections (demodicidosis) with blepharitis and rosacea-like dermatitis may require treatment with a single oral dose of ivermectin, 200 µg/kg, especially in immunocompromised patients. Most animal and plant mite bites also can be managed symptomatically with topical agents unless active infections are present, which can be controlled with topical 10% crotamiton, 25% benzyl benzoate, or topical 1% lindane or 1% malathion preparations. House dust mite allergies may be managed with immunotherapy with house dust mite extracts.

Although initially classified among the *Rickettsia,* the causative agent of scrub typhus, *O. tsutsugamushi* (originally *R. tsutsugamushi*), has now been reclassified into a separate genus, *Orientia,* based on molecular evidence that its cell wall differs significantly from that of *Rickettsia*

FIG. 295.5 Close-up photograph of the pruritic, erythematous papulovesicular bite lesions inflicted by the European oak leaf gall mite on a resident of Pittsburg, Kansas, in late August 2004.[12] *(From Centers for Disease Control and Prevention [CDC]. Outbreak of pruritic rashes associated with mites—Kansas, 2004.* MMWR Morb Mortal Wkly Rep. *2005;54;952–955. Photograph courtesy A. Broce, L. Zurek, Kansas State University.* http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5438a3.htm.*)*

FIG. 295.6 (A–F) Photographs of six persons with skin lesions of *Pyemotes ventricosus* dermatitis. Note the central microvesicles, ulcerations or crusts, and many lesions with the pathognomonic comet signs. (D) Lymphangitis-like dermatitis. (E–F) Lesions resulting from natural infections of two of the investigators. *(From Del Giudice P, Blanc-Amrane V, Bahadoran P, et al. Pyemotes ventricosus dermatitis, southeastern France.* Emerg Infect Dis. *2008;14:1759–1761.* http://www.cdc.gov/Ncidod/eid/vol14no11/.htm.*)*

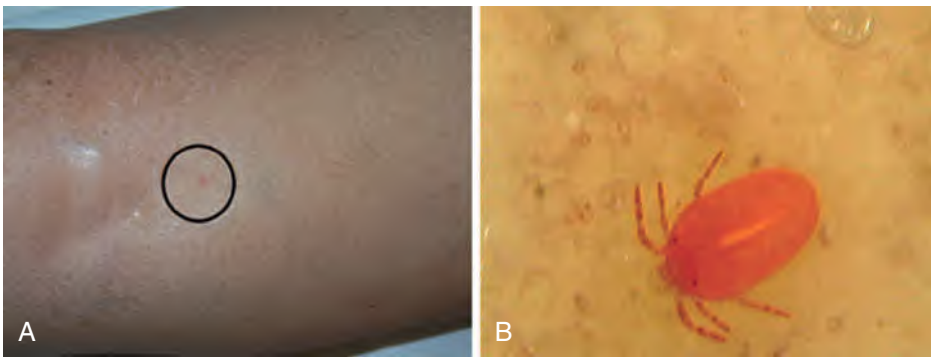


FIG. 295.7 (A) Visual observation of a nonspecific, slightly raised and red pruritic lesion *(circle).* (B) Videodermatoscopic observation of a skin-attached and feeding larval *Neotrombicula autumnalis* mite (magnification ×150). *(From Nasca MR, Lacarrubba F, Micali G. Diagnosis of trombiculosis by video-dermatoscopy.* Emerg Infect Dis. *2014;20:1059-1060. Courtesy Centers for Disease Control and Prevention, Atlanta, GA.)*

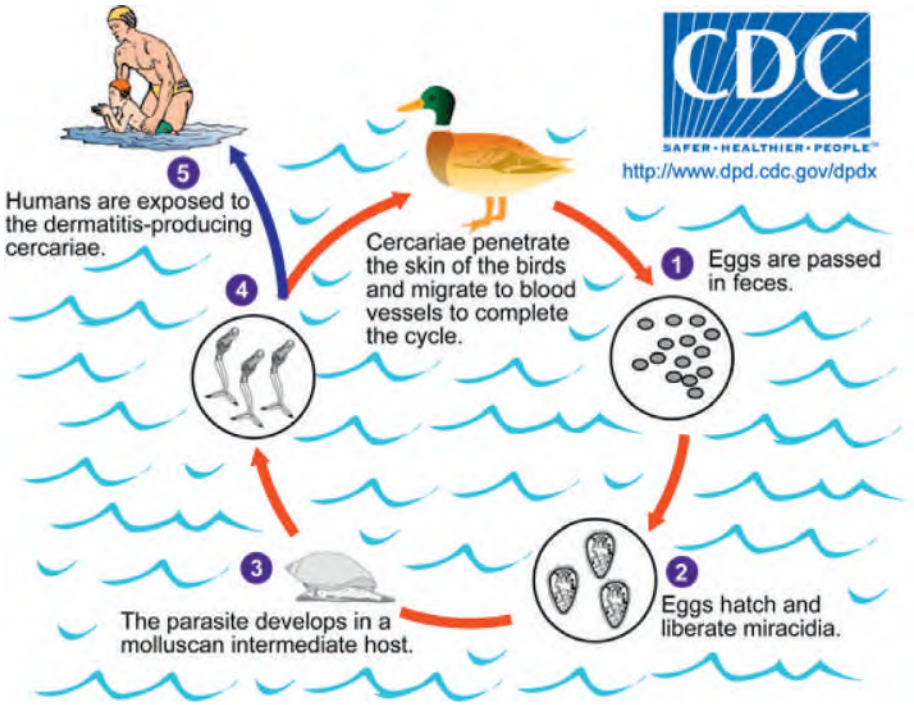


FIG. 295.8 The life cycle of several species of avian schistosomes of resident and migratory waterfowl whose eggs are passed in bird feces and hatch into ciliated miracidia that penetrate aquatic snail intermediate hosts. Infected snails later release infective cercariae into freshwater sources to penetrate the skin of definitive bird hosts or inadvertent or dead-end hosts, such as humans, causing self-limited, intensely pruritic cercarial dermatitis (commonly known as "swimmer's itch." *(From Centers for Disease Control and Prevention. DpDx Image Library.* http://www.dpd.cdc.gov/dpdx/HTML/CercarialDermatitis.html.*)*

both ultrastructurally and in its component proteins (see Fig. 295.2). Nevertheless, mite-transmitted scrub typhus and rickettsialpox have similar clinical presentations; both infections respond to treatment with oral tetracycline, oral doxycycline, or intravenous chloramphenicol, which is not recommended owing to its bone marrow toxicity (see Table 295.2 for comparison of the clinical manifestations, diagnostic methods, differential diagnosis, treatment strategies, and outcomes for scrub typhus and rickettsialpox).

Delusional Mite and Other Ectoparasitic Infestations

In 2014, Diaz and Nesbitt reported their case series and review of delusional infestations in very distraught patients who believed that they were infested with external or internal parasites and described crawling sensations of mites or worms on or under their skin.[17] Some synonyms for delusional infestations have included Ekbom syndrome, acarophobia, and Morgellons disease.[17] These patients should never be dismissed by clinicians without careful environmental exposure histories and physical examinations.[17]

Several true ectoparasitoses have been incorrectly described as delusional infestations, including cutaneous larva migrans, cercarial dermatitis, and *Pyemotes* mite infestations (see Fig. 295.4).[17] Cercarial dermatitis is a very common condition among aquatic gardeners, birdwatchers, fishermen, wildlife photographers, and wildfowl hunters.[17]

Cercarial dermatitis is characterized by pruritic rashes caused by aquatic exposures to the infective-stage cercariae of several avian schistosomes or flatworms that are released into freshwater lakes and rivers in the tens of thousands by infected aquatic snail intermediate hosts in complicated life cycles (Fig. 295.8).[17] Cercarial dermatitis, also known as duck hunter's or swimmer's itch, has occurred in massive seasonal outbreaks in swimmers in freshwater lakes and rivers whose resident or migratory waterfowl are infected with avian schistosomes (Fig. 295.9).[17]